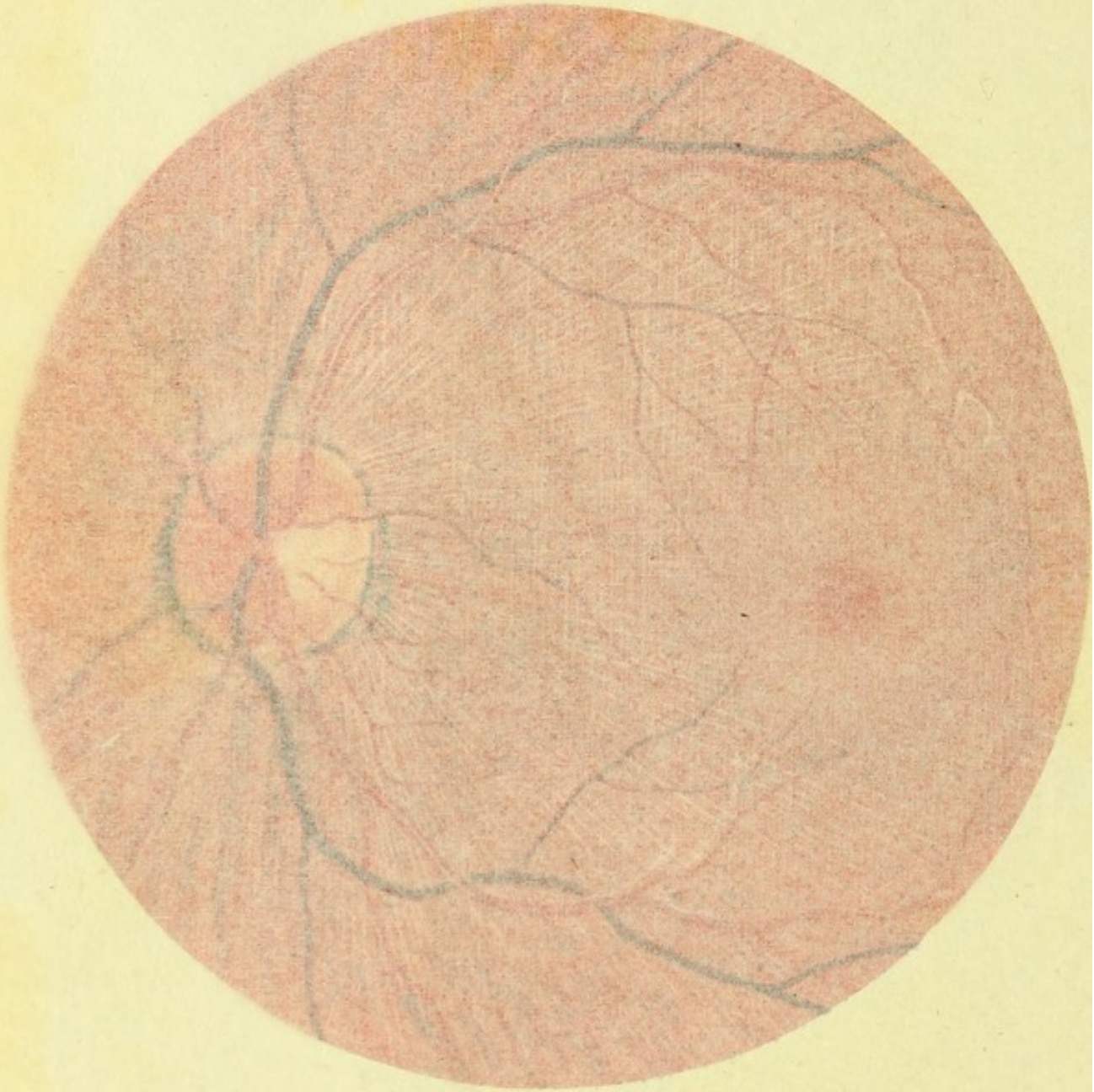
A pocket atlas of the fundus oculi / by George Lindsay Johnson ; with drawings from life by Arthur W. Head.

THE POCKET ATLAS OF THE FUNDUS OCULI

FRONTISPIECE.



FIBRILLATED APPEARANCE OF THE INTERNAL LIMITATING MEMBRANE AND SEMI-TRANSLUCENT NERVE-FIBRES.

From a girl, aged 10 years, with fair hair and blue eyes. Slight hypermetropia; otherwise the vision was normal. Both eyes presented exactly the same appearance. The writer has met with a similar condition in some of the reptiles, but not in any of the mammals.

A. W. Head, del. *Adlard & Son, Im*

A

POCKET ATLAS

AND

TEXT-BOOK

OF

THE FUNDUS OCULI

WITH NOTE AND DRAWING BOOK

TEXT BY

G. LINDSAY JOHNSON, M.A., M.D., F.R.C.S.

FELLOW OF THE ROYAL SOCIETY OF ITALY (MODENA); HON. FELLOW DER GESELLSCHAFT NATURFORSCHENDER FREUNDE, BERLIN; FELLOW OF THE AMERICAN SOCIETY OF OPHTHALMOLOGY AND LARYNGOLOGY; LATE OPHTHALMIC SURGEON WEST END HOSPITAL FOR NERVOUS DISEASES; CONSULTING OPHTHALMIC SURGEON WESTERN GENERAL DISPENSARY; ETC.

WITH DRAWINGS FROM LIFE BY

ARTHUR W. HEAD, F.Z.S.

ILLUSTRATOR OF 'THE MAMMALIAN EYE' 'FROST'S ATLAS OF THE FUNDUS OCULI,' ETC.

London

ADLARD AND SON, BARTHOLOMEW PRESS

BARTHOLOMEW CLOSE

1911

PRINTED BY ADLARD AND SON,
LONDON AND DORKING.

PREFACE.

THE object of this work is to afford the practitioner and medical student a ready means of diagnosing the internal diseases of the eye, as well as the variations in type of the healthy fundus which they are likely to meet with in practice, and which can be seen with the ophthalmoscope. The chief characteristics, pathology and course of each disease, as well as an outline of the treatment, are described.

A description of the principal varieties of the ophthalmoscope and their methods of use are appended, and full details for determining the magnification of the image and refraction of the eye by means of the direct and indirect methods of examination as well as by retinoscopy are given. The theory and estimation of astigmatism are also considered in detail, and illustrated by practical examples.

To make the work as concise and practical as possible, minute details have been omitted and theories and debatable points merely touched on; at the same time it is hoped that no essential matter

has been omitted. Nevertheless a great deal of new material has been inserted, including many of the results of the writer's own investigations, some of which appear here for the first time. As far as space would permit, the latest investigations on each subject have been included.

The retina and choroid are fully dealt with, both as regards their minute anatomy and functions, as well as their congenital abnormalities.

The coloured illustrations, fifty-five in number, have been selected from a large amount of material drawn from life by Mr. A. W. Head, F.Z.S., whose great experience is sufficient guarantee of their faithfulness to nature.

As far as possible various stages of the disease have been depicted, so that the student may have a forecast of the future changes which will probably occur in the appearance of the disease. Very rare diseases have been omitted, except in a few instances in which they have been inserted for special reasons. All the drawings were made by the direct method of examination, with a magnification of about fourteen to sixteen times. Many of them are reduced here from a half to a quarter the original size. The diagrams of the papilla in the text are reproduced by the kind permission of Mr. Gustavus Hartridge.

The book has been made very compact so that it can readily be carried in the pocket and consulted in the dark room. To still further its usefulness a sketch book with blank pages for notes and drawings

is bound up with it, and also a red and black pencil which can be used for drawing the arteries and veins respectively, and sketching in the disease. A more elaborate drawing can readily be worked up by any competent ophthalmoscopic artist from the rough sketches.

In conclusion, the writer desires to thank Mr. W. Anderson, F.R.C.S., and Dr. Thomas Fleming for kindly revising the proof-sheets, also Dr. Alexander Fleming for revising his modification of the Wassermann serum reaction; and Mr. Oscar Wood for revising the first chapter.

G. LINDSAY JOHNSON.

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THE OPHTHALMOSCOPE.

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Principle of the Ophthalmoscope.—If a pencil of rays after passing through any number of refracting media be finally reflected from a portion of a surface at right angles to the axis of the pencil, it will return along the same path. Consequently if a dark room be lighted by a single small window it will be impossible for anyone outside to see any part of that room, unless his eye is placed inside the cone of

the emergent rays. This is rendered difficult and usually impossible by the fact that the observer's head is placed in the direct path of a portion of the entering rays, and as, from what has just been stated, the rays return along the same path, the only beam which could be reflected back into the observer's eye is prevented from entering the chamber at all. If, however, a plane or concave mirror having a central aperture be held in front of the observer's eye and illuminated from any source at the side of the window, and the light be reflected into the chamber, the eye which is placed behind the mirror will see a portion of the room lighted up by virtue of the reflected cone of rays. This is the principle of the ophthalmoscope. The darkened room is the observed eye, the small window the pupil, and the mirror with a central hole, the ophthalmoscope. The pupil, owing to the fact that none of the reflected light under ordinary conditions reaches the observer's eye, appears jet black.

History and Description of the Various Kinds of Ophthalmoscopes.—The history of this instrument is curious. It was invented by Mr. Charles Babbage, the originator of the celebrated calculating machine, in the year 1848. Cushing, a medical student attached to the London Hospital, found that under certain conditions he could see the reflex from the human eye, and that the light was not entirely absorbed by the choroid, as was thought to be the case. This was brought to Babbage's notice, who thereupon constructed an instrument precisely similar in principle to that used at the present day. He placed it in the hands of a veterinary surgeon, but although it is recorded he saw the fundus with it, he failed to appreciate its value, and Babbage took no further trouble to make it known. Three years

afterwards Helmholtz discovered it, and to him the credit of the invention must be given, since he demonstrated the principle and was the means of its being taken up by Ruete, von Graefe, Zander, and Liebreich, in whose hands it soon proved itself to be of extraordinary value. The original instrument of Helmholtz consisted of three superposed and rather large flat plates of glass, which were used as a reflector and transmitter of the light, and set to the source of light at the angle of greatest polarisation,

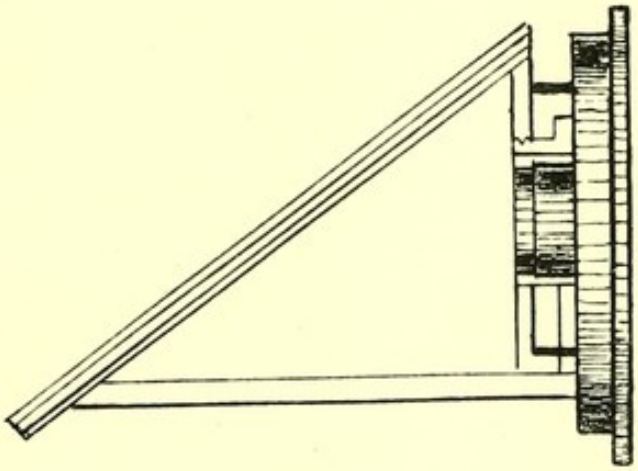

FIG. 1.—Von Helmholtz's original ophthalmoscope.

so as to reduce the dazzling effect of corneal reflection as far as possible.

Ruete in 1852 employed, instead of the glass plates, a small perforated concave mirror attached to a handle—in fact the identical instrument of Babbage. We know now that any piece of looking-glass having a small patch of amalgam scraped away from the centre will perform the functions of an ophthalmoscope, and the only thing needed to make it complete is a convex lens held between the mirror and the patient's eye to form an aërial image. This method of examination merely gives a low magnification and is of small value for determining the refraction of

the eye. To ascertain the latter the lens is discarded, and to get a greater field of view it is necessary to hold the instrument as near to the patient's eye as possible and to correct the refraction of both his own and the observer's eye by means of lenses. This is effected by mounting the lenses either round the circumference of a wheel or in small brass rings, which are rotated in a grooved space which runs

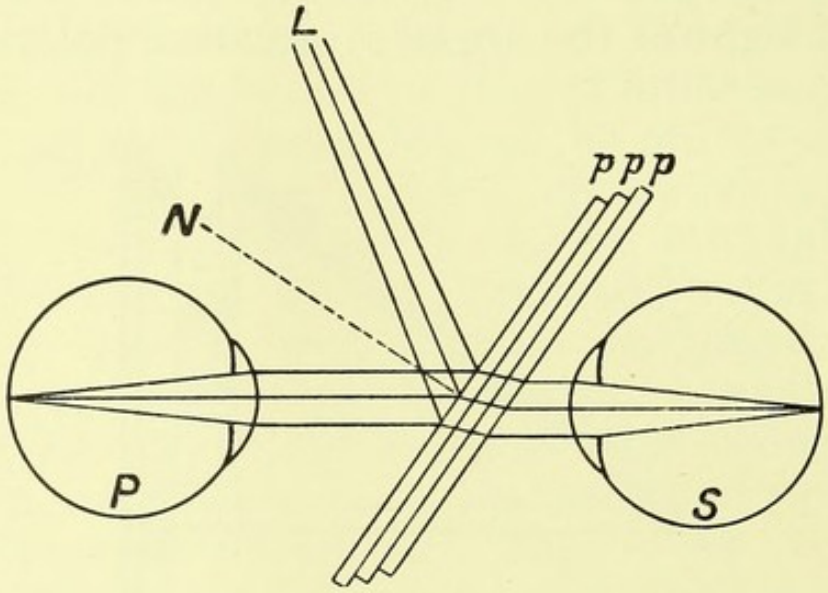

FIG. 2.—Principle of Helmholtz's ophthalmoscope. The greater part of the light is refracted through the plate, but part is reflected to the patient's eye, P, which it illuminates. The rays then return along the same path, and after refraction pursue a parallel course to the surgeon's eye (S) where they are perceived. N is the normal to the plates.

round the body of the instrument by means of a toothed gearing. Since the mirror used in the near (or direct) examination must have a shorter focal length than that used in the indirect method, two mirrors are provided in the more complete instruments. The shorter focus mirror is set at an angle of about 40° so as to face the light, which is usually placed at the side of the patient's head. It can be rotated in any direction with the fingers. A con-

venient handle completes the instrument. Nearly every ophthalmic surgeon has his pet ophthalmoscope, which is oftener than not his own invention. Of course, the highest class of observations can be performed with any form, and the best ophthalmoscope is generally the one the user is accustomed to. Mr. Couper had the lenses fitted in small rings and carried round the sides of the body of the instrument. This is a most excellent plan, as it enables almost any number of lenses to be attached to the instrument. Nearly all magazine ophthalmoscopes (excepting Couper's and the author's) have an additional wheel containing a + 10 D — 10 D, a + 0·5 D, and — 0·5 D lenses, which can be superposed on the other lenses within. This is done to reduce the number of lenses. In the writer's opinion this is a mistake, since each surface continually collects dust, which, when closely applied to the observer's eye causes each particle to spread out into a diffusion circle, which greatly interferes with the clearness of the field. Half-dioptre lenses are an unnecessary refinement, since a very small movement of the lenses nearer or further from either the observer's or the observed eye will cause a greater difference than 0·5 D. If the following thirty lenses are inserted they will suffice for every possible case: 0, + 1, + 2, + 3, + 4, + 5, + 6, + 7, + 8, + 9, + 10, + 12, + 15, + 20, + 40 D, — 1, — 2, — 3, — 4, — 5, — 6, — 7, — 8, — 9, — 10, — 12, — 14, — 16, — 18, — 20, — 30 D. The + 20 and + 40 lenses are useful as loupes to examine the cornea. The — 30 D is only required when the observer possesses a myopia of 5 to 10 D, since a myopic eye of over 25 D cannot be seen by the direct method.

The handle should balance the weight of the rest of the instrument, and screw on to the body by a

double pitch and very coarse thread; this enables the handle to be screwed home by a single turn of the hand. Two mirrors are sometimes considered a great convenience. They should be fitted to a revolving plate in imitation of the double nose-piece of a microscope, which allows of either being swung into position, and further the oblique mirror should be fitted into a sleeve to enable it to be turned in any direction. This method has become universally adopted. Morton was the first to combine all these improvements in his admirably constructed instrument (Fig. 4). As the result of numerous experiments and calculations, it has been found that the large mirror should have a focal length of about 25 cm. and a perforation of 4 mm. The small mirror should have a perforation of 2 to 2½ mm. and a focal length of 5 to 7 cm., to throw the best sized disc of light on to the fundus. Smaller apertures afford no improvement in examining the human eye. It will be seen from the above description that the shape or curvature of the mirror cannot affect the image; it can only alter the size of the illuminated field. The mechanism should be such that a slight movement of the first finger will rotate each lens in turn into position; the ratchet-wheel should be held by a spring armed with a semi-circular projection at its end, which fits into a depression in the wheel. This should work quite smoothly, yet sufficiently for the catch to be felt by the surgeon. Lastly, the large mirror is occasionally made flat on one side and concave on the other. It can be lifted up by a spring and rotated on side pivots. Whether this is an advantage is doubtful. If one wants to use a flat mirror for retinoscopy (sciascopy), it is best to have a separate one rather larger in size. A concave mirror of 120 cm. focus, which will throw a circle

a little larger than the circumference of the orbit, is sometimes an advantage for retinoscopy, although a concave ophthalmoscopic mirror should answer quite well enough. For the indirect method of observation a lens of $2\frac{3}{4}$ or $2\frac{1}{2}$ inches focus (14 D or 16 D) is the best. Opticians generally make the lens either too small or too large. The most convenient size is about 2 in. (5 cm.) in width. It is better to have it unmounted. A good plan is to have the margin

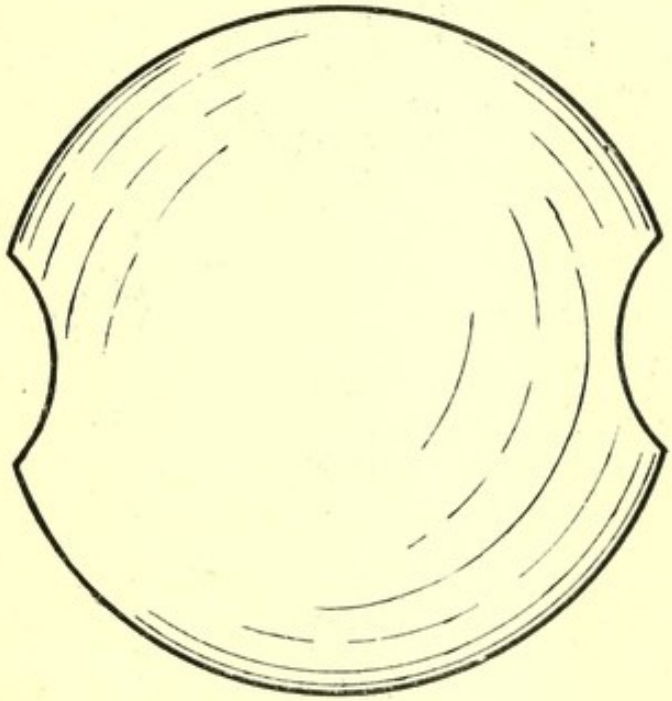

FIG. 3.—Focussing glass showing rim hollowed out for convenience of holding.

hollowed out, to fit the thumb and first finger, as in the accompanying diagram. It does not cut off the view, as one can hold it in any position. It allows of a good grip and greatly lessens the chance of its falling down and breaking. A prismatic, *i. e.* a decentred lens, is also useful, as it enables one to explore the remote parts of the fundus near the ora serrata much more readily. This is often difficult with the indirect method without this aid. It was first introduced by Dr. Abadie, of Paris.

The margins of the apertures and the areas of the bevels, must be painted a perfectly dead black to get rid of all reflexes. The mirror case should be

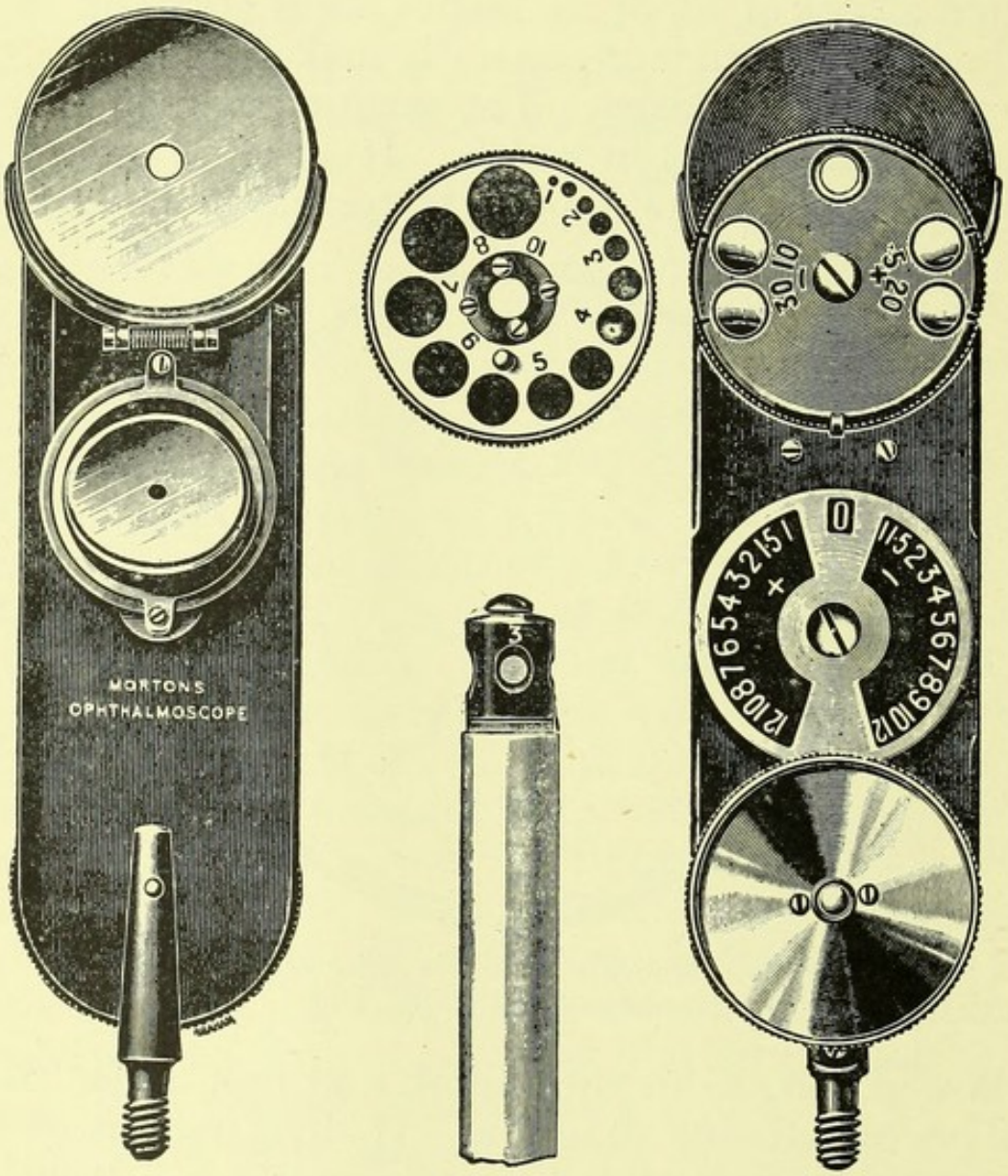

FIG. 4. – Ophthalmoscope showing the rotating mirrors. (By permission of William Gowlland, Ltd.).

bevelled on the back round the aperture, so as to slope very gradually to the glass, which should be as thin as possible. This is essential to avoid reflexes. The same should be done with the ophthalmoscope

plates, so as to reduce the tunnel to the utmost limit.

Binocular Ophthalmoscope.—This was invented by Giraud-Teulon, but although highly praised at the time, it has long been discarded as of little value. Besides, it is very heavy and difficult to use. Its stereoscopic advantages are, in the author's opinion, purely theoretical.

Self-luminous Ophthalmoscope.—Within the last ten years several forms of electric ophthalmoscopes have been put on the market. The pattern (made by Gowlland), is, perhaps, as good as any. It is illuminated by a two candle-power glow-lamp, which is placed in a little tube immediately below the mirror, which is inclined downwards at an angle of 45°. A small accumulator, which runs for a month and can be carried in the pocket, is connected with the terminals of the lamp by two insulated wires. A larger liquid cell-battery is sometimes supplied, but it is much more cumbersome. I have found the former just as good and much less trouble. The light in all the instruments is very dazzling, and I would suggest that the lamp-globe be made of ground-glass or ground over the upper part. It would be quite bright enough and not so hurtful to the eyes. Before I had the lamp made of ground glass I found that few animals could be made to stand the brightness of the glare. Moreover, it caused a great contraction of the pupils, which rendered observation increasingly difficult. The ordinary circular Argand gas burner is much to be preferred to any form of electric light, as it is softer and can be regulated to a nicety. Besides, the flame occupies a considerable area, which is a decided advantage for ophthalmoscopy, since it causes a greater diffusion of focus over the fundus. A candle, though not equal

to an Argand burner, is fairly good. On the other hand, a mantle is too bright. When seeing a patient in the country a bicycle lamp is useful, if one has not an electric ophthalmoscope at hand. It is light, gives a good steady light, and can be held in any position by an assistant. The chief advantage of the

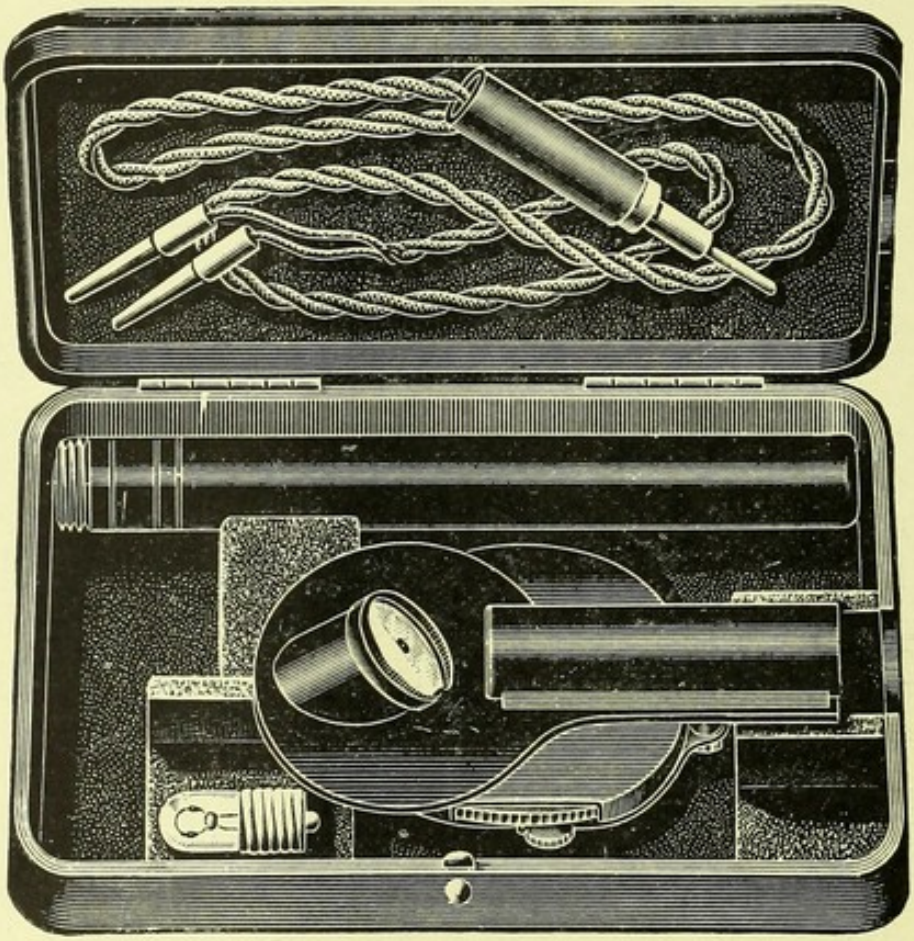

FIG. 4A.—Inskeep's electric ophthalmoscope.
(By permission of William Gowlland, Ltd.).

electric lamp ophthalmoscope is that it can be used anywhere; and is especially useful at the bedside, when the patient cannot move or sit up in bed. In this position the examination with an ordinary ophthalmoscope is often very difficult to manage, owing to the impossibility of putting the candle just where one wants it, whereas with the electric lamp

no difficulty arises. Electric lamps are now made with a resistance by which the light can be altered in the ratios of 2, 4, 8, and 16 c.p. If the room cannot be darkened one may readily see the fundus by bending a piece of brown paper or dark-coloured cloth round the patient's head, keeping the candle on the opposite side while shutting out as much light as possible. Such examinations are best made by the direct method. The writer has sometimes used direct sunlight. It does not injure the eye as one might suppose, since the cone of light does not come to a focus on the retina, but is diffused over the whole visible area. The appearance is very striking to one who is accustomed to observations with gas or electric light, the fundus having a peculiar chamois colour—a sort of violet buff—while the vessels appear of an indescribable brown-purple colour. The disc does not appear pink, but straw colour. The most remarkable appearance of all, however, is afforded by the mercury vapour light, which causes the fundus to appear of a greenish-buff colour. It enables the details to appear strikingly sharp and clear, although the colours are altogether misleading. Some of the leading surgeons believe it may prove of considerable value in diagnosis.

Examination of Animals' Eyes.—In examining rabbits' fundi the best plan is to put them in an oblong box with a sliding lid. The box should be just big enough to hold the animal, and an oval aperture 2 in. by 1½ in. should be cut out near one end so as to expose the eye and its surroundings. The animal will usually keep quiet, and does not seem to mind the examination and confinement. Frogs and toads form excellent practice for the beginner. They are very quiet, and do not seem to mind the light at all. Moreover, their pupils are

very large, having a ratio aperture of F/1½. The American bull-frog and Giant toad show the blood-corpuscles running through the veins and capillaries beautifully, and also the Sprengel suction action.

The Sprengel Suction Action.—This may be observed by everyone in these animals. The rush of the blood-stream in a large vein produces a partial vacuum at the mouth of each contributory vein, and the sudden quickening of the stream as it approaches the outlet is a very instructive, and, we believe, an altogether convincing experiment. Diapedesis may also be studied with great facility, and the effect of drugs noticed. The frogs and toads can be readily held in the hand.

Birds, snakes, and lizards can be held by the neck without any trouble, and stand the light well. Cocaine should never be used for the purpose of dilating the pupil in animals' eyes. For some unexplained reason it causes bubbles to appear under the lens capsule, and prevents a clear view of the fundus being made. In human eyes it never has this effect. Homatropin, 1 per cent. solution, is by far the most suitable mydriatic to use, both for human beings and animals. The author rarely uses any other drug for the purpose of either examining the fundus or testing the refraction, since it paralyses the accommodation sufficiently for every purpose, and its effects only last twenty-four hours, whereas the effects of atropin often last from seven to ten days.

Comparison between the Two Methods of Examination.—There are two methods of examining the fundus by means of the ophthalmoscope, viz. the direct or upright method and the indirect or inverted method. The former is used without any intervening hand-lens, hence the term "direct method";

the latter causes an image of the fundus to be formed in the air just behind the lens. As this image is inverted, it is a real image and can be directly examined with the ophthalmoscope. It therefore forms an indirect method of examination. The indirect method gives a smaller image, but comprises a larger field of view than the direct; in fact, it bears the same relation that a low-power does to a high-power objective in a microscope. The latter shows more detail and is generally to be preferred. But the student must be able to use both methods with equal facility if he wishes to do his work efficiently, for in certain cases, such as high myopia and in eyes with cloudy media, the indirect method is the only one which can be satisfactorily used.

Theory and Estimation of Refraction by the Direct Method.—(1) S *and* P *both Emmetropic.* — Eyes adjusted for infinity. The curvature of the mirror does not affect the refraction, as it merely lights up the interior of P's eye. This being emmetropic, the issuing rays from R_1 are parallel to the axis. As S is also adjusted for infinity, the parallel beam will be conveyed by the media of S to form an image of P's fundus at R_2. R_1 and R_2 are therefore conjugated points. The image seen by S is upright, virtual, and magnified. Since the rays which proceed from P are parallel, it makes no difference to the size of the image how far away S is situated, except that the further S is from P the smaller the area of view. This decreases rapidly, so that even a few inches away the angle of view (which is limited by the size of P's pupil) is so small that a blood-vessel will nearly occupy the whole field—in fact nothing can be seen but a reddish disc of light.

(2) S *Emmetropic*, P *Ametropic.*—Since P is no longer emmetropic, it must be made so in order

that the rays which enter S shall be parallel, and thereby come to a focus at R_2. This is effected by rotating the magazine and bringing the correcting lens for P into position. The condition of things is now restored to that of the former case. *Then the correction necessary for distinct vision is measured by that lens, which when placed behind the mirror at the*

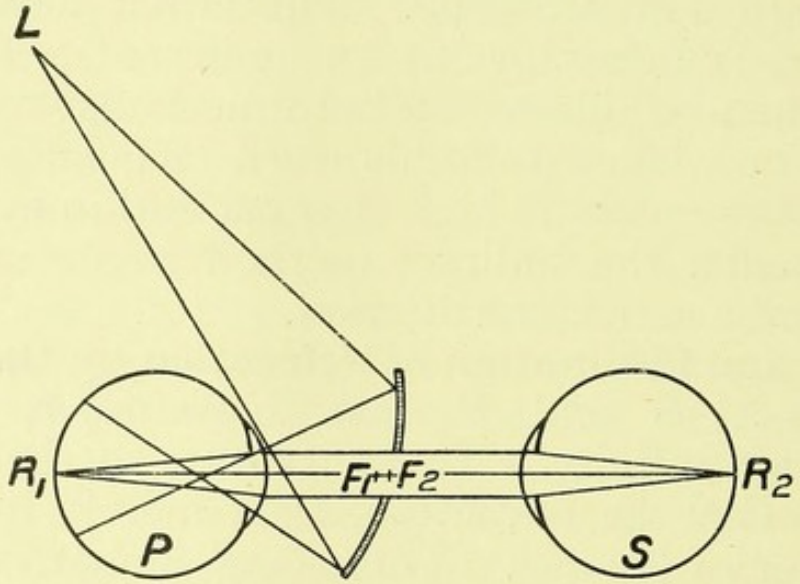

FIG. 5.—Diagram showing the course of rays in the direct method of examination.

S = surgeon's eye (or surgeon).
P = patient's eye (or patient).
L = source of light.
R_1, R_2 = focal points on P's and S's retinæ.
F_1 = Anterior focus of P.
F_2 = Anterior focus of S.

anterior focus of the patient's eye, will enable an emmetrope with relaxed accommodation to see the details of the pole of the fundus. F_1 is (approx.) 15 mm. in front of the pole of the cornea, and therefore corresponds to the position of a pair of spectacles when adjusted to the face.

Note.—The correcting lens must always be the highest convex or the lowest concave glass which

enables S to see the details of the fundus. If this rule is not followed the observer will insensibly accommodate and give rise to a false estimation of the refraction.

(3) *Both* S *and* P *are Ametropic.*—In this case S must just correct his ametropia by bringing into the axial line the glass which corrects his eye for distance. He must then add (algebraically) the corrections for P's ametropia. This restores things to the condition shown in Fig. 5, and S will see the details in P's fundus, since the correcting lens is at the common focus of F_1 and F_2. If the ophthalmoscope be turned round and the light placed behind S's head, P will be able to see the details of S's fundus.

In the above case if C is the correcting glass which will enable S to see P's fundus or *vice versâ*, then—

$$C = D_1 + D_2 \quad . \quad . \quad . \quad . \quad . \quad (1)$$

and $D_1 = C - D_2$.

D_1 being the glass which corrects P's eye, D_2 that of S's eye.

Example.—Both eyes are emmetropic. Therefore C = 0, *i. e.* no correcting glass is required.

Again: S is myopic 6 D; P is myopic 4 D.

Then from (1) $C = -4 + (-6) = -10$ D.

If, therefore, S, knowing that his myopia = 6 D, finds that he can see the fundus of P with − 10 D when the ophthalmoscope is held at the common focus of F_1 and F_2, D_1 (the correction glass necessary for P's distant vision) $= -10 - (-6) = -4$ D.

Another Example.—S is myopic 6 D, as before, but he sees the fundus with + 2 D. In this case $C = +2 \text{ D} - (-6 \text{ D}) = +8$. The patient has, therefore, a H of 8 D for distant vision.

In high cases of M * or H the student must be

* The letters M, H, E are universally used to denote myopia, hyperopia, and emmetropia respectively.

careful to see that the correcting lens is exactly at F_1, otherwise a considerable error will creep in.

Example.—Suppose S is myopic 5 D, and the correcting lens in the ophthalmoscope, which is at his own anterior focus, F_2, but 32 mm. from P's cornea (a not uncommon distance at which the instrument is used), is − 25 D. What glass will correct P for distant objects?

Here the 25 D lens is 32 mm. from P's cornea, and therefore 32 − 15 = 17 mm. from F_1. Now, − 25 D = − 40 mm. ∴ − 40 − 17 = 57 mm. = − 17·5 D. So that $D_1 = C - D_2 = -17{\cdot}5$ D -- (− 5 D) = -- 12·5 D. The patient therefore requires a − 12·5 D lens at F_1 to correct his far sight—a vastly different thing from − 20 D, which a careless observer would have estimated.

Another Example.—P has an aphakic * eye, and S is hypermetropic 3 D. He holds a mirror at his own anterior focus (F_2), but which is at 10 mm. outside F_1. What glass will P require for distance, when S sees the fundus best with a + 20 D on relaxing his own accommodation? A 20 D lens = 50 mm., and since the power of a positive lens is increased when it is withdrawn from the anterior point, we must therefore deduct 10 from the focal length. Therefore 50 − 10 = 40 mm. = 25 D, which represents the power of the lens at F_1. So that $D_1 = D - D_2 = 25 - 3 = 22$ D. Had the student required a + 20 D lens to see the fundus when the ophthalmoscope was held at F_1, the glass required would not have been 22 D, but 17 D.

We remarked on the first page that without some such contrivance as the ophthalmoscope one cannot see the interior of the eye. If, however, the eye is very hypermetropic, the issuing rays may form such

* An eye from which the lens has been removed is termed aphakic.

a divergent cone that more than one observer may see the fundus at the same time. If the refraction of the cornea is neutralised artificially, either by examining the eye under water, by pressing a flat piece of glass against the cornea, or by putting a strong concave glass in front of it, the fundus can be seen by several people at once, since each of these methods will form a highly divergent cone of light.

By holding the large concave mirror several inches away from P one is able to see some of the details of the fundus. For, since the rays diverge, an erect virtual image will be formed behind the eye of P by the projection of the rays backwards. If the eye is

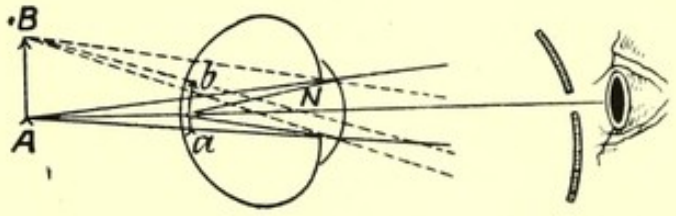

FIG. 6.—Diagram showing how an erect image of the fundus of a hypermetrope can be seen by holding the mirror at a distance.

very hypermetropic, one may even see the image by looking beyond the edge of the mirror for the same reason as we have just stated. By moving the head slightly the vessels of the fundus will be seen to move with the head.

If P be very myopic (*i. e.* 6 D and over), and one holds the mirror about fourteen inches away, or nearer if S has a convex lens behind the mirror, or is himself myopic, the observer will see a very clear image of the fundus. This, unlike the hypermetropic image, is inverted, and by moving the head from side to side the image is seen to move in a contrary direction. In this way one may distinguish between M and H. As the image is inverted, it is

a real image, *i. e.* it can be caught on a screen, and one may magnify it with a Ramsden eye-piece exactly as is done in the field telescope.

Sources of Error with the Direct Method.—The surgeon will often find that his estimation of refraction by the direct method will not agree with the results obtained by trial glasses. This may be due to—

(1) His not holding the correcting lens of his ophthalmoscope at F_1 and F_2, *i. e.* at the coincident anterior focal points of both eyes.

(2) He measures the refraction from some other part of the fundus than the macula area, which may not be the same distance from P's equivalent point.

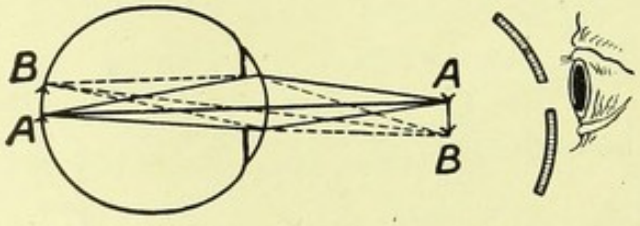

FIG. 7.—Diagram showing how an inverted image of the fundus can be seen in a myopic eye by holding the mirror at a distance.

Sometimes a difference of two or even three dioptres may be noticed between the plane of the disc and that of the macula.

(3) P may not have relaxed his accommodation, or may have developed latent hypermetropia, or may have a partial ciliary spasm.

(4) S may have used too strong a concave or too weak a convex lens than the one necessary to correct the ametropia, or he may be using his accommodation.

(5) The vessel on which he focusses may run deeper or more superficially in the retinal tissue than usual.

(6) He may have directed his line of vision through a part of the cornea outside the visual axis. This is the most common fault of all, and the source of nine

tenths of the errors both in direct ophthalmoscopy and in retinoscopy, but curiously enough, I have never seen it pointed out in any book.

(7) Lastly, P may be stupid and lead S astray over the test types, in which case the latter must counter-check by retinoscopy.

This list is enough to show that it is not such an easy matter to determine the refraction by the direct method as one might suppose at first sight.

Magnification of the Image.—(1) Direct method. Emmetropic eye. Let P be the observed eye, y'

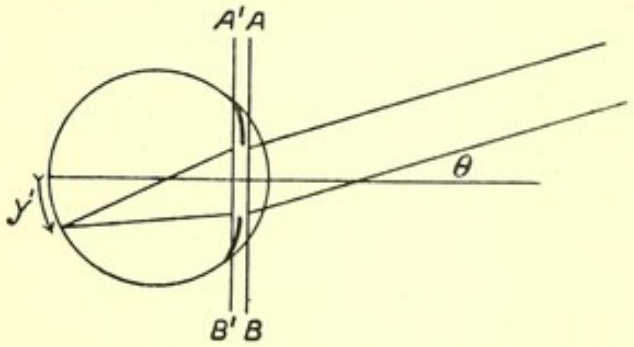

FIG. 8.—Diagram to show method of estimating the magnification.

A B, A′ B′ = Entrance and exit pupils.
θ = Angle subtended by the image y'.
θ' = Angle of image subtended by the eye (not shown in the figure).

a portion of the fundus, A B and A′ B′ the entrance and exit pupils respectively, which lie on each side of the iris. Rays start from the end of the object remote from the axis, and diverging, fill the En P and Ex P*; on emerging they continue their course as a cylinder of parallel rays, making an angle θ with the axis. Ignore for the moment the refractive media of the eye, and consider the object y' seen in the fundus bodily conveyed to a spot outside the eye and regarded from the near point of vision, *i. e.* at

* The contractions Ex P and En P are used here instead of the German ones, A P and E P (Austritt and Eintritt pupille).

250 mm. (or 10 in). This will then be seen under the angle θ', whose tangent is—

$$\theta = \frac{y'}{250} \quad . \quad . \quad . \quad . \quad . \quad . \quad (2)$$

As the angles θ and θ' are small the angles may be taken for their tangents, and the magnification M will therefore correspond to the proportion which θ' bears to θ, or—

$$M = \frac{\theta}{\theta'} = \frac{\theta \times 250}{y'} \quad . \quad . \quad . \quad . \quad (3)$$

But we know that the focal length (f) of a lens is equal to the height (h) of the image, divided by the tangent of the angle which it subtends, measured from the second principal point, or—

$$f = \frac{h}{\theta} = \frac{y'}{\theta'} \quad . \quad . \quad . \quad . \quad . \quad (4)$$

Hence, from (3) $M = \frac{250}{f}$ or the magnification = distance of near point from the eye divided by its anterior focal distance. Taking the anterior focal distance of the eye as 15 mm., we find $M = \frac{250}{15} = 16\frac{2}{3}$, which is the magnification which an emmetropic fundus gives when seen by an observer. Now the distance at which the surgeon observes the fundus does not enter into the equation, therefore it makes no difference to the magnification how far off the patient the observer holds his ophthalmoscope. Since it is necessary for both a myope and a hypermetrope to correct their visual error by a suitable lens, the magnification will be the same for every observer whatever his refraction may be, provided that when he uses a lens behind his ophthalmoscope he holds it at F_1.*

* F_1 is the position of the anterior focus of the eye and is exactly 13·5 mm. in front of the cornea, but we have assumed it to be 15 mm. for the sake of easy reckoning.

Field of View.—The visual field is more extensive when using the direct method, owing to the closeness with which the observer can get to the pupil of the patient's eye. By having the small mirror set at 45° or 50° it is quite possible to approach the mirror within F_1.

The extent of field can be graphically shown by drawing two lines from the centre of the En P H^1 (the

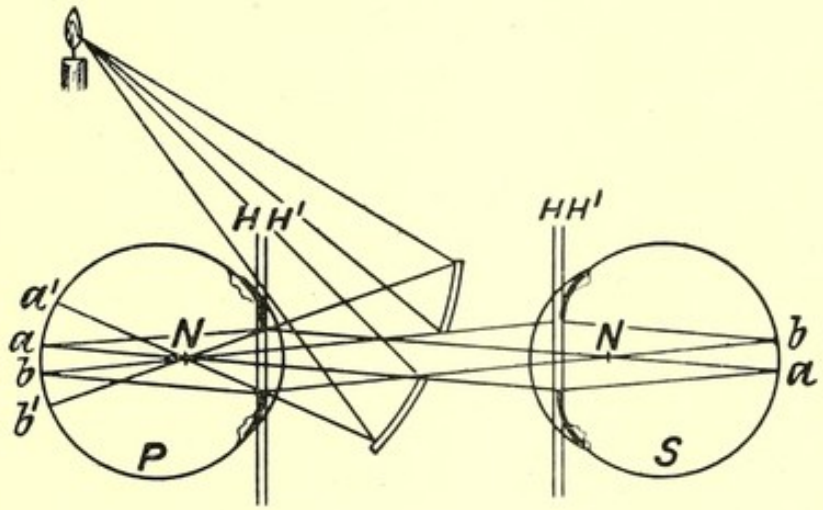

FIG. 9.—Illustrating the field of view, as well as the path of the refracted rays in direct ophthalmoscopy.

$a\,b$ = Image formed on fundus and seen by S.
$a'\,b'$ = Extent of illuminated field.
N = Nodal points.
HH' = En P and Ex P.
$b\,a$ = Image formed on observer's retina.

centre of the real pupil is near enough) of the observer's eye to the margins of the patient's eye, and then following these rays through the refractive media to the fundus. This will form two points, a and b, on the diameter of the visible circle of illumination. This is not the total circle illuminated, for this occupies the space between a' and b'. If the patient's eye is emmetropic and not accommodated all the rays from any point of the fundus will emerge parallel to one another and be re-focussed on the observer's eye.

By getting the patient to rotate the eye, or by the surgeon altering his point of view, the illuminated circle can be shifted over a very large area; in fact if the patient's pupil be fully dilated, especially if the patient is a young subject and the eye hypermetropic, one may trace the vessels nearly to the ora serrata. In a myopic eye this would be almost impossible. In the indirect method, Abadie of Paris uses a thick prismatic lens, by which, according to him, one is enabled to see the ora serrata, although it is difficult to understand how any prism can deflect the rays sufficiently to enable one to see it. Theoretically, in order to get the largest and best illuminated field the centre of the bundle of rays should pass through the centre of the En P of both S and P. In practice one pays no attention to these refinements.

Method of Examination by the Direct (Upright Image) Method.—Examination of R.E. Put your chair a little in front and on the right side of the patient. Place the light near the back of the patient's head and a little to his right. Bring the small mirror into position and rotate it until the surface is normal to the direction of the light. You now correct your vision for the far point by rotating the lenses. Hold the mirror close to your right eye and lean forward until the mirror is close to the patient's eye ($\frac{3}{4}$ inch). Then move the light away from the patient until you just get the full light on the mirror and manœuvre it so as to get the reflexes of the cornea out of the line of sight. The patient is directed to look slightly inwards, so as to bring the disc (papilla) into view. This is a very striking object, and forms a pinkish, vertically oval, patch, often bordered with a thin white margin, which may be pigmented on one side. This is called the scleral border, and is due to the sclerotic shining through.

Here the retina and choroid are absent. Sometimes the whole disc is pink, but often only the marginal part, the middle two thirds being quite white and depressed (physiological cup) (Pl. 10, fig. 20). In most myopes the disc is surrounded on the outer or lower and outer side by a patch or crescent of whitish colour, due to atrophy of the choroid and retina (myopic crescent) (Pl. 23, fig. 46). It is nearly always present in myopes of high degree, and is often partly covered with choroidal vessels. The retinal vessels should be traced in all directions as far as possible, while the patient assists the surgeon by rotating his eye *in the same* direction to which the observer wishes to follow the vessels. For example, if you wish to follow an artery in the patient's right eye towards the temporal side of the fundus, tell him to rotate his eye outwards. In the same way, if the patient is looking through the mirror aperture, he must rotate his eye slightly inwards to bring the disc into view, or, keeping his eye still, you must run your eye along the fundus inwards. The whole fundus can thus be traced nearly as far as the ora serrata. The best way to see the macula and avoid the reflex is to direct the patient to rotate the eye *slightly outwards*. One should then just be able to catch sight of the outer half of the disc. Observe its lower border and run your eye for two or two and a quarter disc breadths *outwards* on a level with it. You will then see the macula, or at any rate the place where the macula is, as it is not always visible, and never in a tessellated fundus (*i. e.* one with stippled black pigment areas). You will recognise the spot by the entire absence of blood-vessels, greater reflex, and often by the more intense red colour and finer "grain" of the tissue. The pit is often seen as a golden or reddish dot, occasionally having a brilliant comet-like reflex radiating from the pit at one side.

In hypermetropes below middle age, and nearly all children and dark-haired people, you will see the macula ring which surrounds the macula area. It appears as a bright round or horizontally oval circle, a little larger than the disc (2 mm.). In the centre of this circle is the foveal pit. The macula ring defines the limit of the area of critical definition of vision.

The red colour of the fundus is due partly to the choroidal vessels, but chiefly to the natural colour of the choroidal pigment, as we have shown elsewhere. Defects of the cornea, viz. striæ, blood-vessels, superficial and deep opacities, can best be seen by means of a corneal microscope, but they can be fairly well observed by throwing the light obliquely on it with the large lens. The details can then be examined with a loupe, or by looking through the + 20 or + 40 D lens of the ophthalmoscope. The cornea may be first stained with fluorescine solution, which brings up abrasions and ulcers into view by the contrast of colour. Defects in the crystalline can be seen by the hand lens in the same way, or by the ophthalmoscope direct method by rotating up a convex lens. Foreign bodies can only be seen by bringing them into focus by the ophthalmoscope, using the direct method, and their position estimated partly by the way in which they stand out in front of the rest of the background, but more exactly by noting the strength of lens required to bring them into focus and comparing it with the lens necessary to see the fundus. It has been proved that 1 mm. in depth (*i. e.* the difference between two vertical planes 1 mm. apart) near the retina = 3 dioptres.

Example.—Supposing the highest convex lens required to see a retinal vessel in a line with the axis is 4 D, and an opaque spot can be seen with + 9 D. The

spot will probably be situated $1\frac{2}{3}$ mm. in front of the retina. A much more exact way is to get the patient to find the position of the opacity himself by means of a pinhole disc adapted to Professor Barrett's entoptiscope.

Theory of Indirect Method.—So called because the fundus is not examined "directly" as we have seen, but in addition a hand lens is held before one's eye to form an inverted aërial image of the fundus in front of the eye which is examined.

P is adjusted for infinity and emmetropic. The rays therefore emerge in parallel bundles, and, on meeting the lens L held near P, they are brought to

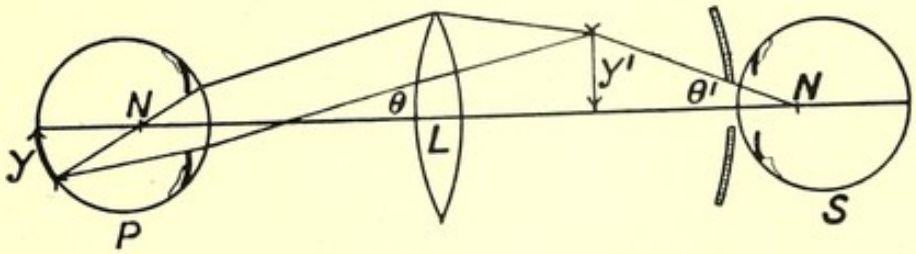

FIG. 10.—General scheme of the examination of an emmetropic eye by the indirect method. *y*. Object formed by a portion of the illuminated fundus. *y'*. Image.

a focus in front of S. The portion of illuminated fundus *y* thus forms an aërial inverted and magnified image *y'*. In order to see *y*, the surgeon, holding the large ophthalmoscope mirror close to his eye, approaches or recedes the hand lens until he gets the image *y'* at his near point. Usually he has to put a convex lens behind the mirror to bring his near point up to *y'*.

The Position of the Focussing Glass and Mirror: Telescopic Position of the Focussing Glass.—If the focussing glass be held so that its posterior focus F coincides with the anterior focus (ϕ) of the eye, it is said to be in the telescopic position, for the glass and eye together will then form a telescopic system, *i. e.*

any ray in the eye which is parallel to the principal axis will emerge from the focussing glass unchanged in direction. Thus, supposing we hold a focussing glass of 6·5 cm. focus (+ 16 D) at 8 cm. from the eye,

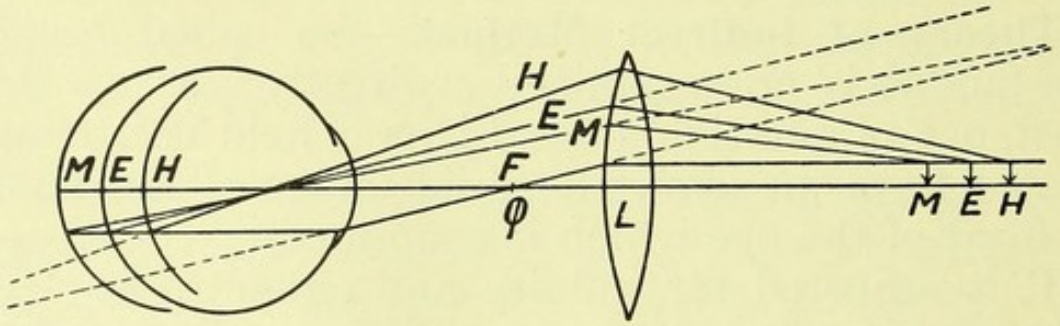

FIG. 11.—Diagram showing that the aërial images produced by the lens (L) are the same size for all three forms of eyes when the lens is in the telescopic position.

which is the sum of the focal distances of the eye and lens (1·5 cm. + 6·5 cm.), the lens will be in the telescopic position. In this case the image of any eye, whether H, E, or M, will appear the same size. This is made clear in Fig. 11. The focussing glass is

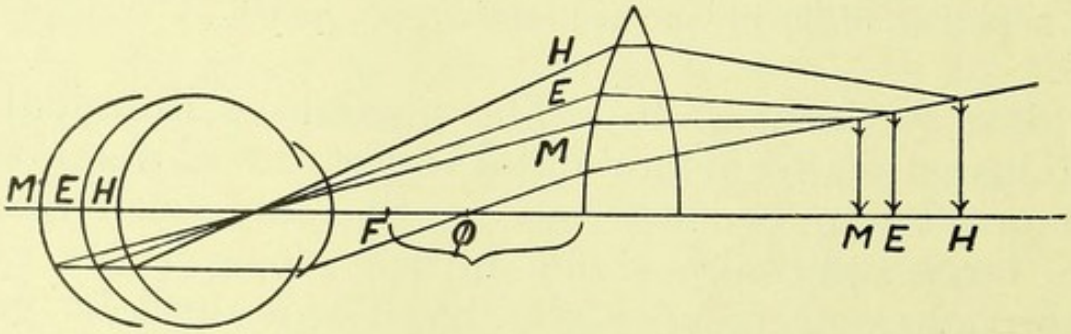

FIG. 12.—Diagram showing that the hyperopic image is greater, and the myopic one less than the emmetropic image, when the posterior focus of the focussing glass lies within the anterior focus of the eye.

generally held in practice just within the above position, when the myopic image will be seen to be *less*, and the hyperopic *greater* than the emmetropic one (as shown in Fig. 12).

Estimate of Refraction by the Focussing Glass.—

If the glass be withdrawn from the telescopic position, the myopic image will grow larger and the hyperopic smaller than the emmetropic one. Since the rate of increase or decrease varies with the amount of refractive error, it will indicate very roughly the amount of ametropia. The emmetropic image will remain the same size wherever the lens is held, because the emergent rays always form parallel beams if the accommodation is relaxed (Fig. 13). We can always increase the magnification by using a weaker focussing lens, but at the same time it will throw the image further back, which gives a smaller field and is often a disadvantage.

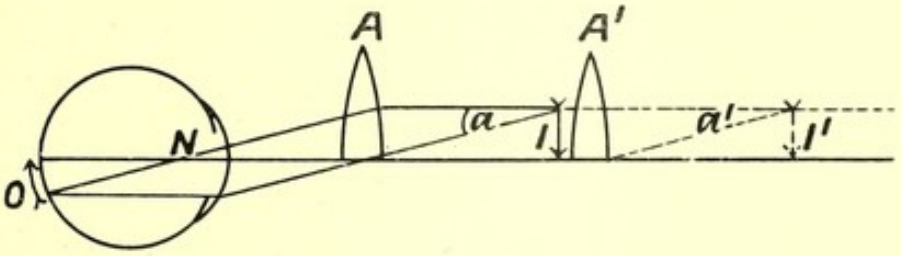

FIG. 13.—Diagram showing why the position of the focussing lens does not affect the size of the image formed by an emmetropic eye.

Image seen by the Mirror used alone at a Distance from the Eye.—Since the emergent rays of a myopic eye converge and form an inverted aërial image in front of the eye (Fig. 14), it is evident that this image of the fundus can be seen with the ophthalmoscope without any focussing glass, provided the patient has over 5 or 6 D of myopia so as to bring the image within a few inches from the patient's eye. This will enable the surgeon to view the image at his near point of vision.

In the same way one may see the fundus of a highly hyperopic eye with the mirror alone, since the emergent rays are very divergent and form an image behind the eye. This image is virtual and

erect, and not inverted and real like the myopic one. It is formed in the same way as the image of an object seen through a hand magnifier, the divergent rays being projected back to form an image where they meet in focus behind the fundus, or the object viewed at the retina.

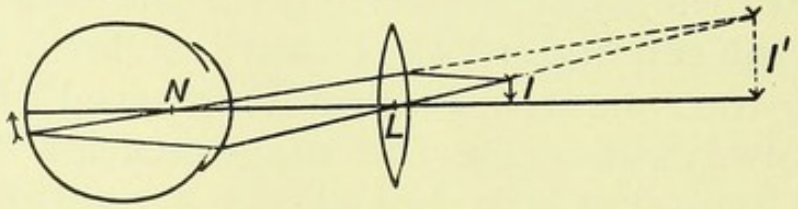

FIG. 14.—Diagram showing how the image (I′) formed by a myopic eye can be seen with the ophthalmoscope alone without the intervention of the lens L.

I = Image produced by the lens L.
I′ = Image formed without using the lens.

Summary.—If on withdrawing the focussing glass the image grows larger, the patient is myopic. If it increases rapidly it is a case of high myopia.

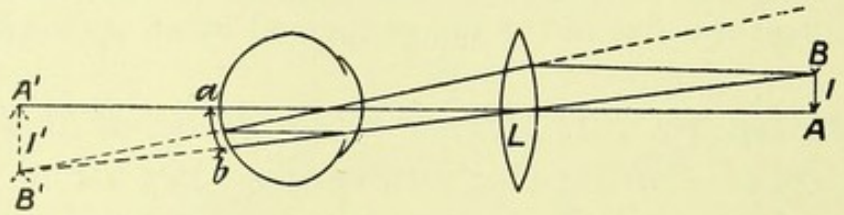

FIG. 15.—Diagram showing how the image may be seen by the ophthalmoscope alone in a hyperopic eye.

If the image remains the same size it is probably a case of emmetropia. If it grows smaller it is a case of hyperopia.

If the image is seen by the ophthalmoscope alone at a distance from the eye, it is a case of either myopia or high hyperopia.

If the image is real and inverted—myopia. If virtual and erect—hyperopia.

If, on using the mirror alone at a foot or more from the eye, one sees a distinct inverted image of the fundus which moves the reverse way to the ophthalmoscope (or one's head), it is a case of myopia (or else E or low H with strong accommodation), and the degree of myopia can be roughly guessed by the distance of the image from the eye.

If one sees an upright image which moves with the ophthalmoscope it is a case of a high degree of hyperopia. As the emmetropic eye projects parallel rays, it forms no image except during accommodation.

In the case of high myopia this method is of considerable value, since the magnification by the indirect method is often insufficient and the direct method difficult to employ. It gives a high degree of magnification, but owing to the distance from the eye the field is objectionably small, and one can only see a small portion of the disc at a time. We may further enlarge this image by holding the focussing lens in front of it, so as to get an enlarged virtual image of the real aërial one, and one can then observe it with the ophthalmoscope in the usual way. The only difference between this method and the ordinary indirect method is that the focussing glass is held on the observer's side of the image instead of between the patient's eye and the image.

Method of Estimating the Refraction by the Indirect Method.—The determination of the refraction is rather more complicated than with the direct method, but by a little artifice it can be greatly simplified.

Let E M H represent three different eyes having the three forms of refraction. L = intervening hand lens. If the patient relaxes his accommodation his far point will be at infinity if his eye is emmetropic, near his eye if he is very myopic, and theoretically,

behind his eye if he is hypermetropic. Of course, this point can only be found by geometrical construction, as the rays which pass out of the eye are

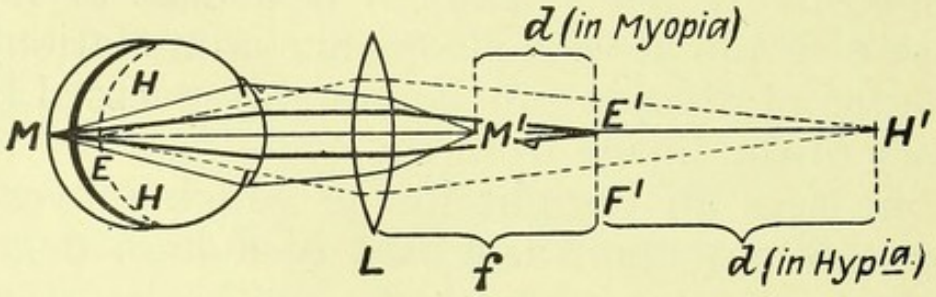

FIG. 16.—Diagram illustrating the principle of Mauthner's (Schmidt-Rimpler's) device for determining the refraction by the indirect method.

divergent and require to be traced backwards in order to meet at a focus. Now let the student place a + 10 D lens in front of the patient's eye. The

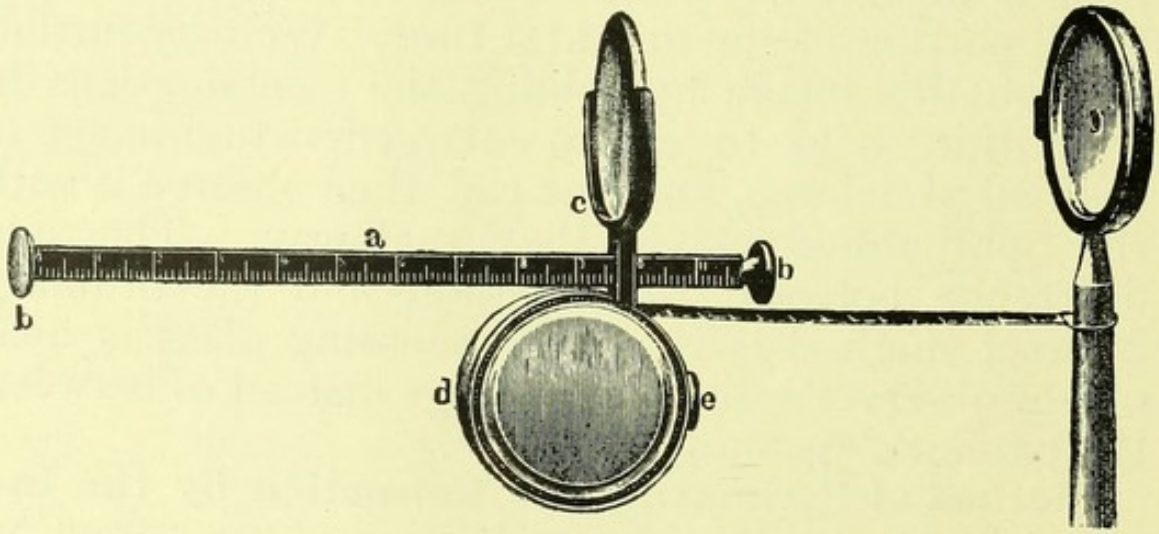

FIG. 17.—Schmidt-Rimpler's apparatus for determining refraction. Ophthalmoscope, tape measure, and graduated rule (a) along which the + 10 D lens (c) is made to slide.

observer, if not already myopic, makes himself so by putting a + 5 D = 20 cm. lens behind his ophthalmoscope. He holds the + 10 D lens exactly at 10 cm. from the patient's cornea. This lens is best fixed in a stand while the patient has his eye kept steady in

a head-frame and chin-rest. A tape or measuring-rod marked in centimetres is attached to the lens-holder. This tape can be made to slide through a groove and drawn towards the observer. The surgeon holds the ophthalmoscope with the + 5 D lens to his eye and moves his head to and fro in a line with P until he sees the image most clearly.

This image A will obviously be at E^1 in emmetropia, between E^1 and the lens L in myopia, and beyond E^1 in hypermetropia. Consequently in the last two conditions there will be an interval d between the point at which the observer sees the image best and E^1 (which is the same point as F^1), the anterior focus of L. This interval d can be measured and the patient's refraction found from the usual conjugate formula : $\frac{1}{f} = \frac{1}{a} + \frac{1}{b}$, where f = the focal length of L, a = distance of the aërial image from the lens, and b = distance of the patient's far point (A^1) from the lens.

Hence in emmetropia $A = f$ . . (5)
(this being the focal length of the lens) = 10 cm., and therefore in this case A coincides with a ;

in myopia $A = a = f - d$ . . . (6)
hypermetropia $A = a = f + d$ . . (7)

Take the case of a myope first :

By substitution in the formula $\frac{1}{f} = \frac{1}{a} + \frac{1}{b}$ or $\frac{1}{f} - \frac{1}{a} = \frac{1}{b}$ we get $\frac{1}{f} - \frac{1}{f-d} = \frac{1}{b}$,

or $\frac{(f-d)-f}{f(f-d)} = \frac{1}{b}$,

$\therefore b(f-d-f) = f^2 - fd$,

or $-bd = f^2 - fd$

and $-b = \frac{f^2}{d} - f$ . . . . (8).

But (and here the artifice comes in) the lens is at the distance 10 cm. from the eye $= f$. Therefore the far point A^1 of the myopic eye from the cornea

$$= b + f = \frac{f^2}{d} - f + f = \frac{f^2}{d} = \frac{100}{d}.$$

Thus, if d = 1 cm., his far point is at 1 metre,
d = 2 ,, ,, ,, 0·5 ,,
d = 3 ,, ,, ,, 0·3 ,,
d = 4 ,, ,, ,, 0·25 ,,

so that each centimeter of difference between the position of the image and F^1 *is equal to* 1 D *of refraction necessary for correction.*

Example.—The observer (whom we will assume is emmetropic) looking through his ophthalmoscope behind the + 5 D lens sees the image sharp at 24 cm. At 26 cm. the image loses sharpness, so that 25 cm. is the furthest distance at which he can yet get a clear image. But he is looking through a + 5 D lens = 20 cm., therefore the image is at 25 − 20 or 5 cm. from the lens. Hence d is equal to 5 cm.

Therefore from our rule given above he has 5 D of myopia.

In Hypermetropia the same rule holds good, but instead of A being $= f - d$, A $= f + d$, so that we change the signs and write—

$$A^1 = \frac{-f^2}{d} + f - f = \frac{-f^2}{d} \quad . \quad . \quad (9).$$

Example.—The observer sees the image best at 34 cm. Here 34 − 20 = 14 cm., or 4 cm beyond F, $\therefore d = 4$ and $-\frac{100}{4} = -25$ cm., *i. e.* his far point is 25 cm. behind his eye and he needs a + 4 D for correction.

In Emmetropia A and F coincide, $\therefore d = 0$, consequently $A^1 = \infty$, and he needs no glass at all.

Example.—The observer sees the image best at 30 cm. Therefore 30 − 20 = 10, or the image is at the principal focus of the lens. But when the image is at the principal focus the rays from the object must be parallel. The patient is, therefore, emmetropic.

The reason why this method is much less used than formerly is that so small a distance as 1 cm. makes a difference of 1 D, and to measure to within half a centimetre is almost impossible. It affords, however, excellent practice for the student, and for that reason we have explained it in full.

Schmidt-Rimpler's Method.—This consists in projecting a contrast image (Fig. 18) on to the retina by the concave mirror. This is a white design drawn on a black ground and strongly illuminated by the ophthalmoscope lamp, to which it is attached by a movable arm. It can be seen projected in space as a real inverted image on the further side of the 10 D lens. This method is precisely the same as the last, only more accurate, since the artificial object can be more exactly focussed than a portion of the retina.

Difficulties connected with the Indirect Method.—(1) *Surface reflections.*—These consist of reflections of the source of light from P's cornea and the two surfaces of the intermediate lens. These can be displaced towards one side of the cone of light by moving the illuminant to one side or the other.

(2) If the patient looks at the centre of the mirror the light will be thrown on his macula, which will cause the pupil to become contracted and render the fundus more difficult to see, and at the same time the macula will give rise to a dazzling reflex. It is therefore necessary to start with examining the papilla, which, being insensitive to light, will obviate these objections. The student having observed the

disc, can now throw the macula into view either by shifting the intermediate lens, and so make use of its prismatic action, or he can get the patient to rotate his eye outwards through a small angle, which will produce the same result.

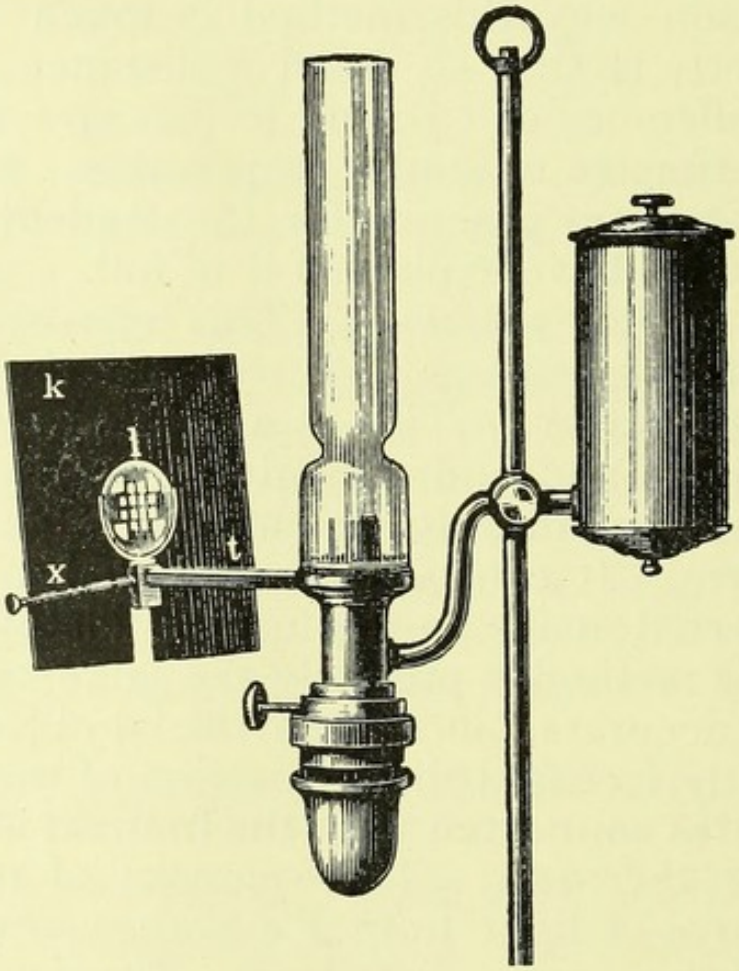

FIG. 18.—Schmidt-Rimpler's contrast image and lamp. Design (1) in black and white (illuminated by the lamp), the image of which is projected on to the fundus of the patient by the ophthalmoscopic mirror.

(3) There is a further difficulty in locating the position of the aërial image. This is done by using one's accommodation, and can only be arrived at by constant practice.

(4) The field of view is smaller than with the direct, but owing to the small magnification the

amount seen without shifting the ophthalmoscope is greater.

(5) The estimate of refraction, although quite easy in theory, is in practice somewhat uncertain and attended with considerable difficulty to the beginner, since it solely depends upon keeping the lens exactly at 10 cm. from P's eye, and also upon the correct measurement of the distance of the eye from the condensing lens.

Magnification by the Indirect Method.—Here the patient's eye and the lens L may be considered as a

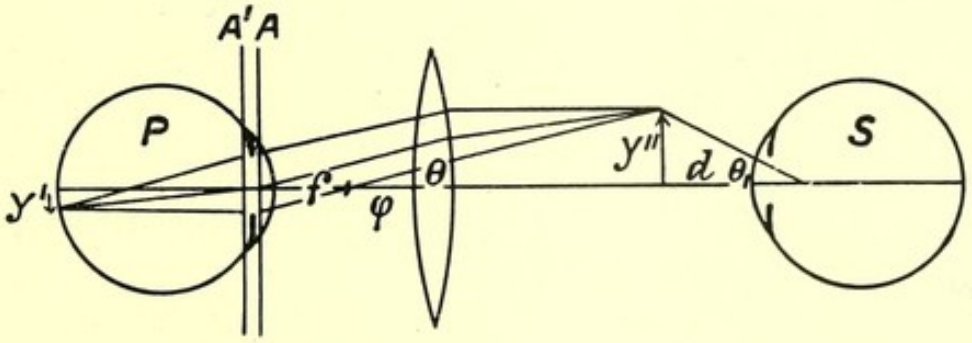

FIG. 19.—Magnification produced by the intervening lens in the indirect method of observation when the lens is in the telescopic position.

y' = Object seen in fundus.
y'' = Magnified aërial image.
f = Anterior focal length of P's eye.
h = Height of image.
d = Distance between y'' and surgeon's pupil.
θ = Angle which issuing rays make with the axis.
$\theta_,$ = angle subtended by the magnified image at the surgeon's eye.
ϕ = Focal length of the lens L.

single sytem having y' and y'' as conjugate object and image.

From (4) $f = \frac{y'}{\theta}$

$$\therefore y' = f \times \theta \quad . \quad . \quad . \quad . \quad (10)$$

$$\text{and } y'' = \phi \times \theta \quad . \quad . \quad . \quad . \quad (11),$$

that is to say, the image y'' is as many times as large as the object y' as ϕ is greater than f.

Multiplying up and removing θ we get—

$$y'' = \frac{\phi}{f} y' . \quad . \quad . \quad . \quad . \quad (12).$$

This is only another way of expressing the same thing, and gives the actual size of the image.

The magnified image y'' will be seen by S under an angle θ_1 or $\tan \theta_1 = \frac{y''}{d}$.

When θ_1 is small, which it usually is, the tangent is the same as its angle, so that $\theta_1 = \frac{y''}{d}$.

If we now consider y' to be removed outside the eye and regarded from S's near point under an angle θ_2, then $\theta_2 = \frac{y'}{\text{D}}$ (D being the conventional near point distance = 10 in. or 250 mm.),
and we get the magnification—

$$\text{M} = \frac{y''}{d} \times \frac{\text{D}}{y'} = \frac{\phi}{f} \times \frac{\text{D}}{d} \quad . \quad . \quad . \quad (13).$$

This is not the actual size of the image, but the size of this image as it appears to an eye at the conventional distance of 10 inches or 250 mm. In other words, the magnification is equal to the angle which the aërial image subtends at the observer's eye, divided by the angle which the retinal object subtends at the same spot.

If S sees the image from the conventional distance 250 mm.,

then (from 13) $\text{M} = \frac{\phi}{f} \times \frac{\text{D}}{d} = \frac{\phi}{f} \times \frac{250}{250} = \frac{\phi}{f}$. (14),

or the magnification is equal to the focal length of the lens divided by the anterior focal distance of the eye. Thus if ϕ (the focal length of L) = 10 cm. or 100 mm.,

then M $= \frac{100}{15} = 6{\cdot}66$ times.

If S is hypermetropic d will have a greater value than 250 mm., say 300 mm.,

then M $= \frac{100}{15} \times \frac{250}{300} = 5{\cdot}5$ times.

If S is myopic d will be less, say 125 mm.,

then M $= \frac{100}{15} \times \frac{250}{125} = 13\frac{1}{3}$ times.

If P is myopic and L be slowly withdrawn from P, a glance at Figs. 14 and 19 will show that y'' will increase and d diminish.

Therefore the image will enlarge rapidly, until L arrives at the far point of P, when the image will be the same size as though the lens were not there. Further withdrawal will cause the image to enlarge still more, and if the far point is sufficiently near, a real erect image of y'' will be formed on the cornea, after which it will apparently vanish. Still further withdrawal will bring this final real image within the observer's range so that he will again obtain a view of the fundus, but inverted with respect to the original view. This can only happen when the myopia is very high (over 6 D or 8 D), as the observer's eye would be too near to P to get an image at all.

If P be emmetropic, moving the lens L away will make no difference to the magnification.

If P be hypermetropic, moving the lens away will diminish the magnification and reduce the field of view. Hence, if a hypermetropic, a myopic, and an emmetropic eye be examined by the same person with the lens L at the same spot, the image will diminish in the case of the hypermetropic and increase in the case of the myopic eye as the lens is withdrawn, while it remains the same size in that of the emmetrope.

Examination of the Patient by the Indirect Method.—After having examined the eye as far as possible by means of oblique light, the student should commence the examination of the fundus by the indirect method.

The room is darkened sufficiently, or a curtain may be drawn to screen off the outside light. Sit close to and in front of the patient. If the right eye is to be examined the light is placed on the right side of the patient's head or just above it. Rest your third and little finger on the patient's right brow, holding the lens between the first finger and thumb from one to two inches in front of the patient's cornea. First rotate the lens behind the ophthalmoscope, which will correct your vision for about 14 inches reading distance. Then hold the ophthalmoscope close to your right eye in your right hand, steadying the instrument by pressing the rim of the mirror against the edge of your orbit. Then direct the patient to look at the top of the right-hand little finger, which you extend, or else the tip of your right ear. This causes the patient to rotate his eye slightly inwards, and brings the disc into the line of vision when the observer's eye is about 16 inches from that of the patient.

Advance or recede the hand lens until the disc is sharply defined, and if necessary do the same with your eye and ophthalmoscope. If you cannot see anything sharply defined, examine the cornea with oblique light and search for an opacity. If the cornea is clear, examine the back of the cornea carefully for precipitates, with the ophthalmoscope and oblique light. Then examine the crystalline lens the same way. If the obstruction is not there search for it in the vitreous. Dilate the pupil with cocaine if necessary to see the details clearly. Trace the

vessels from the disc is every direction as far as possible by moving your head, or the lens laterally, and up or down, and thus sweep the whole fundus. Recollect that you are not seeing the fundus itself, but an inverted aërial image of it, and therefore, the image being upside down, everything observed appears to move in the opposite direction. Having inspected the disc, follow the image of the fundus *inwards* along a horizontal line, starting from the top of the disc for 2 to $2\frac{1}{4}$ disc breadths. This will be the position of the macula. If you cannot see it you must turn the light as low as will just enable you to see the details of the fundus and continue your observation. You will see two light reflexes on the cornea. These you must endeavour to get out of the line of view by either moving or tilting the lens slightly by moving your head. The reflex from the macula itself can be got rid of by getting the patient to rotate his eye well *outwards*. If you are troubled with a reflex at some part of the circumference of the aperture of the mirror or ophthalmoscope you must clean both surfaces of the ophthalmoscope lens. If that fails you can often remedy it temporarily by turning the train of lenses out of the way, *i. e.* to zero, and striking a *wax* match. This you hold with the flame close below the aperture, first on one side of the instrument, then on the other, so as to get a smoky flame. This will cover the bevels of the mirror aperture and those of the ophthalmoscope plate holes with soot. Then clean the mirror, and the reflex will probably have vanished.*

* A permanent dead black may be made and applied as follows: Take 1 grm. of black paint in powder (bone black will do), mix it with turpentine to consistency of treacle, and then add 2·5 grm. of ordinary terebene; mix well, and apply with a brush to bright parts well warmed (R. E. Heppell). Or: Take a pinch of ivory-black in powder, mix it with a little nitro-cellulose (gun-cotton) dissolved in

High = magnification ophthalmoscopes. — Every physiologist or ophthalmic surgeon has at times felt the want of a high magnification ophthalmoscope. By an elementary knowledge of optics and a little ingenuity it is quite possible to very considerably increase the power of the instrument up to sixty or even one hundred diameters. In fact, the writer has on several occasions applied the compound microscope with a water-immersion lens to study the fundus of the mole's eye, which is the smallest mammalian eye existing. In every case it is essential that both the observed eye and the instrument should be as rigid as possible, and to obtain this desideratum some form of fixed ophthalmoscope, such as Nachet's, Thorner's, or Dimmer's must be used.

In addition to a rigidly fixed stand, it is necessary to have a head-rest to secure immobility on the part of the patient. Further, the fundus has to be illuminated in such a way that the reflected rays shall reach the observer's eyes without any extraneous light whatever. Otherwise the reflexes will prevent one seeing anything properly.

The following methods can be employed for this purpose: First, by interposing in the tube of the compound ophthalmoscope (which is, in fact, a compound microscope) a thick cover-glass placed at an angle of 45°. A circular hole is cut in the tube and an oil lamp, in front of which is a collimator or bull's-eye condenser, to project parallel rays through the aperture on to the cover-glass. This form is not to be recommended, as a great deal of light reaches

acetone or ordinary collodion to make a thin paste, and apply with a camel's hair brush to the parts previously well warmed. Wood or bone charcoal rubbed up with methylated spirit is effective, but not so permanent.

the eye which is not derived from the object itself, and which entirely spoils the image. Secondly, a perforated concave mirror may be fixed in the tube at an oblique angle and illuminated as in the first case. This is used exactly as an ophthalmoscope mirror, and the aërial image magnified by a Ramsden ocular. Third, one may secure illumination by means of one or more glass prisms. This is quite the most satisfactory of all methods, as one can very effectually ensure that only the useful light reaches the eye.

In all cases when reflected light reaches the eye the light must be separated either by a suitably placed reflector or by a prism or prisms into two parts, viz. useful light from the object, and injurious light from all other sources. The latter must be screened from the eye by some method, such as a diaphragm, while the other half, which is derived from the fundus, reaches the eye undisturbed.

Further, one may magnify the image either by means of a compound microscope or a telescope. In the former case one must combine the object glass with the eye to be examined, so as to form a compound system having its posterior focus at the front surface of the system, *i. e.* on the retina or choroid. The magnified image must be formed somewhere in the tube of the instrument, and this image can then be further magnified by means of an eye-piece. The tube should be capable of considerable extension (as in the ordinary compound microscope), so as to compensate for slight movements of the object glass, which may either be a dry or a water-immersion of long-working distance.

But there is another way in which magnification can be maintained, viz. by means of a telescope.

We know that in an emmetropic eye at rest the

emergent rays are adjusted for infinity and therefore parallel. Since, therefore, the rays reflected from the fundus emerge in a parallel beam, a telescope, either in the form of a field-glass, an astronomical, or a Galilean telescope, may quite conceivably be used with advantage by employing one of the lateral means of illumination mentioned above. For if a convex and a concave lens be separated by an interval equal to the difference of the two focal lengths, or if two convex lenses be separated by an interval equal to the sum of the two focal lengths, the system becomes a telescopic one, and the rays enter and leave the instrument as parallel beams. If, therefore, the objective of a telescope be placed in front of the pupil of an eye, and the tube carrying the eyepiece be adjusted for a star, or, in other words, for infinity, when the fundus is illuminated it will be in focus. If the conditions are favourable, its details, magnified by the eye-piece, will be seen in focus, because parallel rays come to a focus on the retina of an emmetropic eye when the accommodation is relaxed. The additions necessary are : First, to secure rigidity for the telescope and the patient's face ; second, to provide for illumination of the fundus ; third, to arrange the light, by means of mirrors or prisms, so that all light not reflected from the fundus is cut off before it reaches the eye. This telescopic method has one great advantage over the microscope in that, as it is adjusted for parallel rays, a little shifting backwards or forwards of the patient's eye will not affect the sharpness of the image at all. On the other hand, the illumination must be very intense for photographic purposes, since the diameter of the object-glass is confined to the size of the dilated pupil. In order to increase the actinic power, both the objective and eye-piece should be constructed of quartz, which

permits the highly actinic, ultra-violet rays to pass which ordinary glass does not, at least, not to any great extent.

Photographing the Fundus Oculi. — Successful photographs of the fundus oculi are extremely difficult to obtain owing to the small size of the image, the red colour of the background, the length of exposure necessary, and the disturbing effect of reflexes and useless light. Dr. Thorner was, I believe, the first to obtain successful negatives, but they were too small to be of much value. Subsequently Professor Dimmer, with the help of the staff of Zeiss's firm, constructed a large and optically perfect instrument, with which he has made a series of highly interesting and instructive negatives.

The apparatus is as follows:

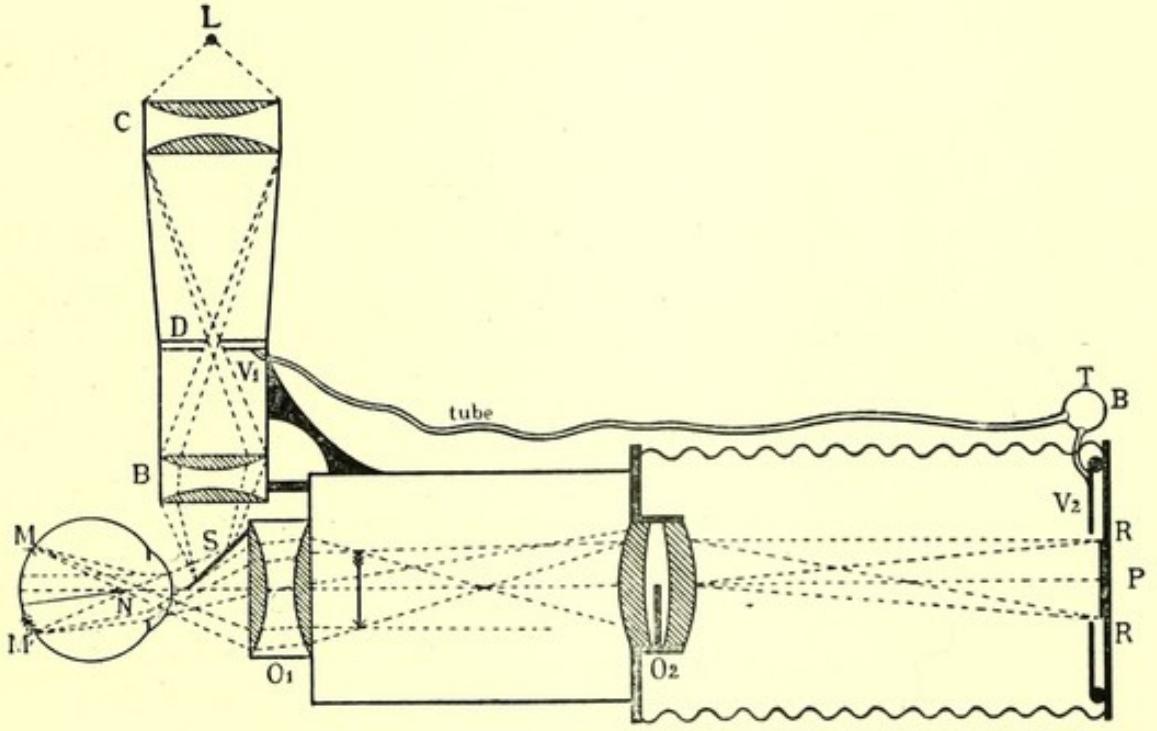

FIG. 20.—Diagram illustrating Prof. Dimmer's apparatus for photographing the fundus oculi.

L is a powerful arc light; C the condenser; D a diaphragm, which cuts off all excepting the central

rays from the condenser, which are here brought to a focus. From this point the rays spread out to fill the collecting system B, and pass directly on to the plane mirror S, from whence they are reflected through the upper part of the pupil of the eye and, crossing at the nodal point N, illuminate a large area of the fundus. This forms the object to be photographed, which in man, of course, forms a brilliant, orange-red, concave, spherical mirror MM. The system of lenses is so arranged that the rays which enter through the upper half of the pupil emerge only through the lower half of it, and are collected into a convergent beam by the condenser O, on to the objective O_2. This objective works at an aperture of F/4·5 or thereabouts, so as to pass as large a beam of light as possible, and is perfectly achromatised for both red and green rays, as these are the chief colours we have to deal with. This object-glass focusses the rays sharply at R, where the sensitive pan-chromatic plate is inserted. A pan-chromatic plate is sensitive to all the colours of the visible spectrum, and is a *sine quâ non*, since ordinary gelatine plates are insensible to the red end of the spectrum, which the greater part of the fundus reflects. The entire space between O and R is, of course, surrounded by a box or bellows which forms the camera. The shutter V_1 lies just behind the diaphragm D, while the second shutter V_2, which is in the form of a roller blind, lies immediately in front of the plate P. By means of a small mirror (not represented in the figure) the image can be observed up to the instant of exposure. A squeeze of the ball T causes the image to be exposed, and at the same instant both shutters, controlled by a single wire, are opened and closed by an electric current.

The front part of the apparatus between O_1 and O_2 is never altered. The focussing necessary owing

to errors of refraction in the observed eye is got by racking the focussing screen R in or out. The position of the observed eye is assumed by getting the patient to fix the image of a flame in a mirror placed before him with his other eye.

The original negatives show a magnification of three diameters, which can readily be enlarged two or three times more, so that one can obtain a picture

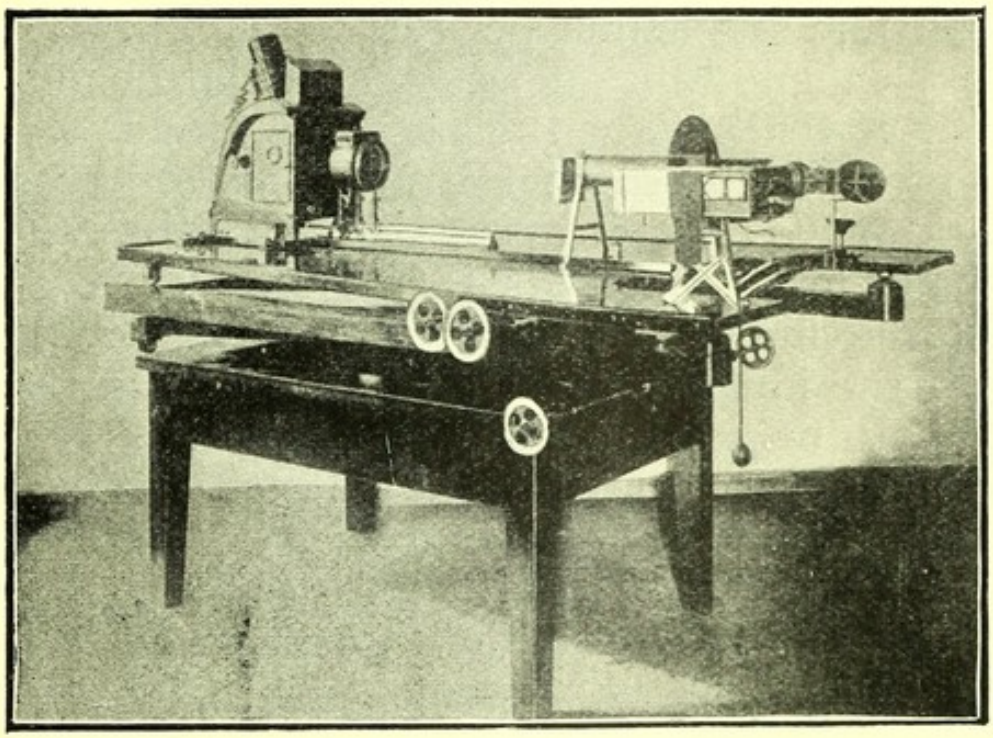

FIG. 21.—General view of Prof. Dimmer's apparatus for photographing the fundus oculi. From a photograph.

eight or nine times that of the original. In Professor Dimmer's hands the photographs have been very successful, in fact they stand unequalled.

For ordinary clinical observation the smaller and very compact instrument of Dr. Thorner is very useful. It is constructed on the same general principles as Dimmer's, but although greatly inferior for the purpose of photography, affords very good images to look at. They are extremely clear and

bright and nearly devoid of reflexes, without the drawback of all ophthalmoscopes.

Retinoscopy (Sciascopy, Koroscopy, Shadow-test).—This valuable method of estimating refraction was first practised by Cuignet, of Lille, in 1873, and in 1875 by Charnley, and by Parent, of Paris, who first accurately explained the phenomena. In England, Hartridge has brought it to a state of great perfection. Retinoscopy is largely practised in England and the United States.

The principle of retinoscopy may be defined as follows: A reflex from the patient's fundus illuminated by a plane or concave mirror is observed by the surgeon. On slightly rotating the mirror to and fro, this reflex will appear to move either with or against the movement of the mirror, according to whether the image formed by the mirror is real or virtual with respect to the observed eye, or whether the punctum remotum of the latter is situated within or beyond the observer's cornea. The optical correction necessary to exactly arrest or just reverse the movement produces in the observed eye a myopia of 1 D, because for the movement of the shadow to be stopped the punctum remotum of the observed eye must coincide with the cornea of the observer. The distance between S and P being taken as 1 metre, the measure of the refractive error is that lens which arrests all shadow movement with the addition of — 1 D to compensate for the artificial myopia produced.

Case A : Mirror Concave.—On examining Fig. 15 it will be seen that there are four images, I^{I}, I^{II}, I^{III}, I^{IV}. The object O is the source of light placed behind on one side of the patient's head. Since O is about 120 centimetres from the concave mirror, which usually has a focal length of 15 cm. to 25 cm., the image I^{I} will

be real, inverted, and situated between M and P. Its exact position can be readily found from the usual formula, $\frac{I}{A} = \frac{I}{F} + \frac{I}{U}$, where F = focal length of the mirror and V and U the distance of I and O respec-

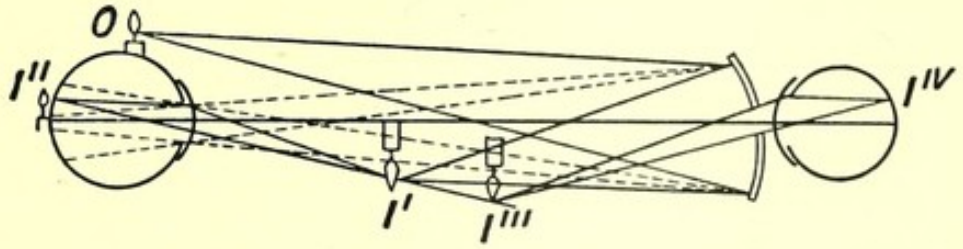

FIG. 22.—Diagram showing the formation of images in a myopic eye and concave mirror.

O = Object (candle).
I^{I} = Aërial real image of O projected by mirror (inverted).
I^{II} = Real image (erect) of I^{I} on P's fundus.
I^{III} = Real image (inverted) of I^{II}.
I^{IV} = Real image (erect) of I^{III} on S's fundus.
M = Concave mirror.

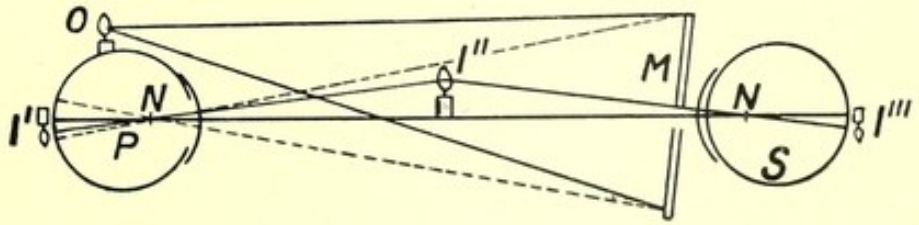

FIG. 23.—Diagram showing the formation of images in a myopic eye and plain mirror.

O = Object.
I^{I} = Real image (inverted) on P's fundus.
I^{II} = Real image (erect) of I^{I}.
I^{III} = Real image (inverted) of I^{II}.
M = Plane mirror.

tively. For let the candle be 140 cm. from the mirror which has a focal length of 20 cm., then its real image I^{I} will be situated at 23·3 cm. from the mirror. It really forms an aërial object which projects a second image on P's fundus (I^{II}). This will be inverted as regards I^{I}, but erect with respect to the

source of light O. If P's eye is myopic the illuminated fundus will form a third image I^{III} somewhere in front of P, inverted both actually and as regards I^{II}. If P is myopic 1 D, the image I^{III} will be 1 metre in front of P, if he has M = 2 D it will be at 50 cm., if M = 5 D at 20 cm., in front of P, and so on. Hence it follows that if S and P are separated by 120 cm. (the usual distance) and P has a myopia not less than 1 D, the image will be formed in the air in front of the observer's eye. This will form a final image I^{IV} on S's fundus, which, being real, is again inverted as regards I^{III} but *erect* as regards the source of light. Then as the mirror is moved to the right, I^{I} moves also to the right. Therefore to the observer the fundus reflex apparently moves with the mirror. If P has a myopia exceeding 1 D, the only difference will be that the image of P's fundus will be proportionately nearer P's eye, and consequently whatever the refraction is, the image seen by S (and consequently the shadow) will move with the mirror. If, however, P's myopia is less than 1 D, the image will be formed behind the observer's eye, with the result that the rays will not cross at all but will form an inverted image of I^{II} as with the plane mirror, and consequently the image (or shadow) seen by S will move in a *contrary direction* to the mirror. If P be emmetropic the rays will emerge as a parallel bundle and will form a virtual image at ∞, which will produce the same result to the observer's eye as in the last condition. If P is hyperopic the same appearance will be seen by S, only in this case a virtual image will be formed behind P's fundus, and therefore the image seen by S will still move against the mirror.

Case B : Mirror Plane.—Now consider the second diagram, Fig. 16. The mirror is flat, consequently a virtual image will be formed as far behind the

mirror as the source is in front. Since this first image is erect it follows that I^{1} is inverted, the rays only coming to a focus on P's fundus where they form a real *inverted* image and not a real *erect* one, as in the first case. If the eye is emmetropic this image will fall on the fundus, if myopic, in *front* of, and if hyperopic, *behind* the fundus, but in all three conditions the only difference will be one of relative sharpness. The image will always be the same in character, *i. e. inverted* and not erect. The image will in all cases be projected exactly as in the former case, *i. e.* if P is myopic it will form a real upright image somewhere in front of P. If myopic 1 D or more it will be formed in front of S's eye. If less than 1 D (or, strictly speaking, if less than 0·83 D, since we took 120 cm. as the distance of separation between S and P), it will be formed behind S. If P be emmetropic no image at all will be formed (except at infinity), and if hyperopic, a virtual image will be formed behind P's fundus.

Thus as M is rotated to the right the virtual image behind it will move to the left, and therefore I^{II} will move to the right and I^{III} to the left. The student, therefore, sees the reflex I^{III} move against the rotation of the mirror. In fact, it will always move in the contrary direction to what it would do if the curved mirror were substituted. In a word, in E, H, and myopia under 1 D it will move *with* the mirror, in all degrees of M over 1 D it will move *against* it.

This is the explanation of the difference in action between the two mirrors.

Method of Procedure. — The room is partially darkened as when using the ophthalmoscope. The light is placed either directly above or on one side of the patient's head, taking care that the face is completely in shadow. A small bright light is the

best, and the smaller the better. The student sits down about 4 feet (120 cm.), or rather less from the patient's eye, and places a pair of trial frames on the patient's face. It is immaterial whether the observer's refraction is corrected or not, but it is well to do so if he is myopic, otherwise the reflex will not be distinct. Either mirror may be used at pleasure. The plane mirror is preferred by some, because it gives a better and more even illumination of the fundus, and the disc of light is of the same size for all distances of the observer. The great thing is to throw a bright disc about 2½ inches wide on to the patient's orbit. This just allows room on the eye for the rotation of the mirror. If the fundus reflex is not seen (as is often the case when the eye is first examined and the pupil is small—2 to 3 mm.), get the patient to rotate the eye slightly inwards so as to bring the disc into the field. This will cause a bright whitish reflex, which is very readily perceived. On rotating the mirror laterally one can judge by the rate of movement of the shadow whether the refraction is near emmetropia or not. The quicker the movement and the better defined the shadow the nearer is the approach to emmetropia. If the ametropia is very high you will probably not see any reflex at all, in which case hold the mirror close up to the eye and observe the appearance of the fundus, and roughly bring the details into view with the correcting lenses behind the mirror. If you cannot do this, withdraw the mirror slowly, and observe if the image enlarges rapidly or remains the same size. This will show M or E as the case may be, and you will be able at once to place a lens in the frame which will enable you to get the reflex. If the pupil is large, or the patient is young, you will have no difficulty in getting the reflex from the very first.

If the eye is emmetropic, or M under 1 D, the image-shadow will move against it with a concave mirror, and with the mirror if a flat one, the reason of this being that the observer's eye is one metre, or thereabouts from P's eye. Now the far point of a low myope of 1 D is at 1 metre; consequently the image will be formed at or behind the mirror, so that the inverted image of the fundus and not its erect image is seen by the observer. Hence the shadow behaves as in the case of E and H, *i. e.* against the motion of a concave mirror, and with that of a flat one. In all myopes exceeding 1 D the opposite takes place, because the image of the fundus is formed in front of the observer.

Let us suppose the student employs a concave mirror. As soon as he has ascertained the direction of the shadow he should proceed to place in the trial frame + 1 D, + 2 D, + 3 D, etc. (beginning with + 1 D), if the shadow is against, or − 1 − 2 − 3 D lenses if the shadow is with the movement of the mirror. As soon as a lens causes the shadow to vanish on rotation, add a + or − 0·25 D lens to the lens of the same sign. If this causes the shadow to reverse you know the previous lens is the right one. If you have any doubt add first a + ·50 D and then a − ·50 D. If the shadow reverses with each, the mean lens is the right one.

This description holds equally true with a flat mirror, always bearing in mind that the movements are of the opposite character. You have now to add − 1 D to the first lens which gives no shadow or a feeble reverse.

The beginner always has a little difficulty in testing low grades of ametropia owing to the turning-point being near zero. Thus, if without any correcting lens you get no shadow, add a + 0·5 D. If the

shadow goes with the mirror, try a — 0·5 D. If the shadow goes against, you will probably find a — 0·25 D gives an uncertain shadow or no shadow at all, and a + 0·25 the same. The patient is therefore feebly myopic, probably requiring a lens between — ·50 D and — ·75 D, since + ·25 and — 1 D = — 0·75 D. If the turning-point is at + 1 D, + 1 D and — 1 D = 0, the patient is emmetropic. If the turning-point is reached with a — 1 D, he probably requires somewhere about — 2 D to correct his sight.

The rule is, therefore: Sit at 120 cm. from the patient's eye. Note the last lens of the series which gives the same kind of shadow and the first lens of the same series which turns the shadow. The mean lens between the two, added to — 1 D, will be the correcting lens required.

Thus, suppose + 3 D is the last lens, beginning from + 1 D, which gives a shadow against the mirror, and a + 4 D is the first of this series to give a shadow with the mirror, then + 3·5 will be the mean, and + 3·5 — 1 D = + 2·5 will be the lens required.

Again, suppose — 4 is the last lens of the series before the shadow ceases, and a — 4·5 reverses. The mean, then, is — 4·25. You will not find a — 4·25 D in the trial case, so you put up a — 0·25 in front of the — 4. If that gives no shadow, add — 1 D. Then — 4·25 + (— 1 D) = — 5·25 will be the correction. But as there is no such lens in the case, it is much better to take off 0·25 D and give — 5 D than add a quarter and give — 5·5.

Many refractionists are of opinion that if there be any indications of spasm or accommodation, or of astigmatism, it is advisable to instil a 1 per cent. solution of atropine into the eye for two or three consecutive days before examination. This rule refers to all children and most persons under eighteen

years of age. As the effects of atropine do not pass off for at least ten days after the last instillation, many surgeons use a 1 per cent. solution of homatropine instead, the effects of which only last about twenty-four to thirty hours.

If mere dilation of the pupil is required, a few drops of a 2 per cent. solution of cocaine hydrochlorate will answer perfectly. Should there be any apprehensions of glaucoma supervening, which, however, is highly improbable in patients under thirty years of age, a single drop of sulphate of eserine solution ($\frac{1}{4}$ grain to the ounce of water) instilled inside the eyelids will quite overcome the cocaine, and if repeated in an hour's time will do the same in the case of the homatropine solution.

Paracentral Shadow.—This phenomenon in an exaggerated form is highly characteristic of conical

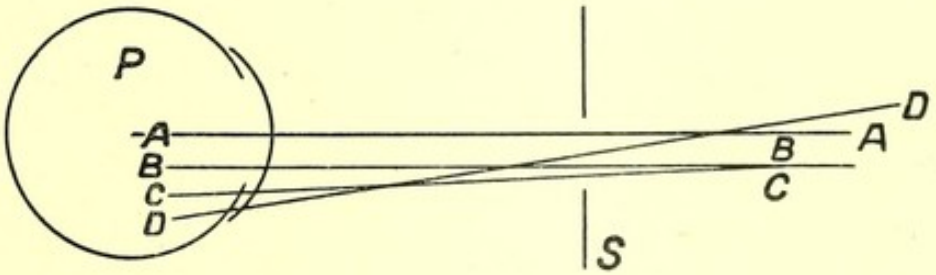

FIG. 24.—Illustrating the theory of the paracentral shadow. (After Tscherning.)

cornea, and exists in a slight degree in all cases in which the cornea is irregular, or where the refraction varies in different meridians. It is also characteristic of a high degree of spherical aberration. Let P be an emmetropic eye, A, B, C, D, paraxial rays,* having a high degree of spherical aberration, so that the oblique pencils form a myopic focus, or in other words, cross the axis at, or in front of, S. Hence the

* Rays which only make a small angle with the principal axis, and which therefore lie near to it, are termed "paraxial rays."

observer will see the bundles of rays along the lines A and B, and, perhaps, D, but the rays round C will be cut off by S's pupil, and the illuminated fundus corresponding to that portion will appear dark. Hence the observer will see a bright central reflex surrounded by a dark border, and possibly another bright circle outside again. If S's pupil be displaced a little downwards, the bundle A would be cut off and B C D would be seen. This produces the phenomenon of the paracentral shadow.

Conical cornea may be diagnosed by giving the mirror a slight rotary motion in a vertical plane, keeping the mirror perpendicular to the axis. In this case the paracentral shadow revolves with the mirror in a highly characteristic fashion, while tilting the mirror, as for retinoscopy, does not cause the shadow to cross the field as is usually the case.

Astigmatism.—One of the most important applications of retinoscopy is the determination of astigmatism. It is quite the exception to meet with an eye which is free from this refractive error. An astigmatism amounting to 0·25 D, either in the vertical or horizontal meridian, is almost universal, and unless it exceeds that amount it is rarely necessary to correct it. Consider an axially placed cone of light which falls on a *small* central area of the cornea from a distant source. If the surface of the cornea is perfectly spherical the rays which pass along any meridian will, after refraction, form a second cone whose apex is a point which will lie in front of, behind, or on the retina, according as the eye is M, H, or emmetropic. If the refraction of the peripheral rays of the cone is not the same as the paraxial rays the point will form a disc of confusion, but it will be circular and due to spherical aberration and not to astigmatism. If, however, the cornea or either of the surfaces of

the crystalline lens are not portions of a sphere, but have a greater radius of curvature in one meridian than in another at right angles to it, the focus will no longer be a point or disc, but will form an oval or even linear focus. This is termed "regular astigmatism." If different segments of the crystalline lens or cornea (through malformation or injury) have different refracting powers, the image point will spread out into a stellate or irregularly shaped patch. This condition is termed "irregular astigmatism." One of the chief functions of retinoscopy is to diagnose the latter, as well as to determine the amount of difference between the two chief meridians in the former case, which will permit of a glass of such form being placed before the eye that the refraction of the two meridians will be equalised. Irregular astigmatism can sometimes be benefitted by glasses, pinhole or stenopaic diaphragms, but it can rarely be satisfactorily corrected.

Regular astigmatism is divided into simple, compound and mixed, according to whether one meridian only or both are ametropic in the same sense (*i. e.* both hyperopic or both myopic), or whether one meridian is hyperopic and the other myopic (mixed astigmatism). A little consideration will make it clear that all cases of regular astigmatism can be corrected by means of a cylindrical lens, or by the combination of a spherical lens with a cylinder of the same or opposite sign. One may correct each meridian by a given cylinder in any case of regular astigmatism and combine them with the chief meridians at right angles, but in practice this is never done, because a sphero-cylinder can always be found which will replace any combination of cylinders, and such a lens is much cheaper and easier to grind.

Principle of Regular Astigmatism—The path formed

by a cone of rays projected from a point situated on the axis and allowed to fall on a cornea which has its two principal meridians of different curvature, and, consequently, of different refractive power, may be shown as follows :

Select a convex spherical lens of, say, + 5 D, and combine it with a convex cylinder of + 3 D axis horizontal. This combination will be the same as a toric lens* of 8 D or 12·5 cm. focus in the vertical meridian and 5 D or 20 cm. focus in the horizontal, and the two foci can be received on a screen held at these distances. Place a candle on a table, preferably in an open box having a small hole opposite the flame. Fix the combined lenses in a clip attached to an adjustable stand with its principal axis in a line with, and half a metre in front of, the light. Hold the screen vertically in front of the lens and slowly withdraw the screen, and observe how the circle projected by the combination narrows first to a horizontal oval and at 16·6 cm. from the lens contracts a horizontal line. This forms the *first focal line*. It is horizontal because the rays in the stronger (vertical) meridian are all focussed to a point, while the horizontal rays are only partly contracted, since they come to a focus further off. Continue to withdraw the screen and observe that the vertical rays now having crossed diverge, while those in a horizontal plane continue narrowing so that we get first a horizontal oval and then a small circle. This is the spot at which the diverging and converging beams are of equal diameter (circle of least confusion). Further withdrawal brings about a vertical oval, which narrows down to a line at the point where the hori-

* A toric surface is one having two cylindrical powers worked on its convex surface at right angles to each other. It gets its name from the vaulted ceilings of cloisters. (Latin, *torus*, a cushion.)

zontal lines meet at a focus, while the vertical ones have still further diverged. This forms the *second focal line*, which is 33·3 cm. from the lens. This line is in the vertical meridian (meridian of greatest refraction), the first line being in the horizontal meridian of least refraction. The space between the two focal lines is the interfocal distance (interval of Sturm).

The following diagram will show the course of the rays:

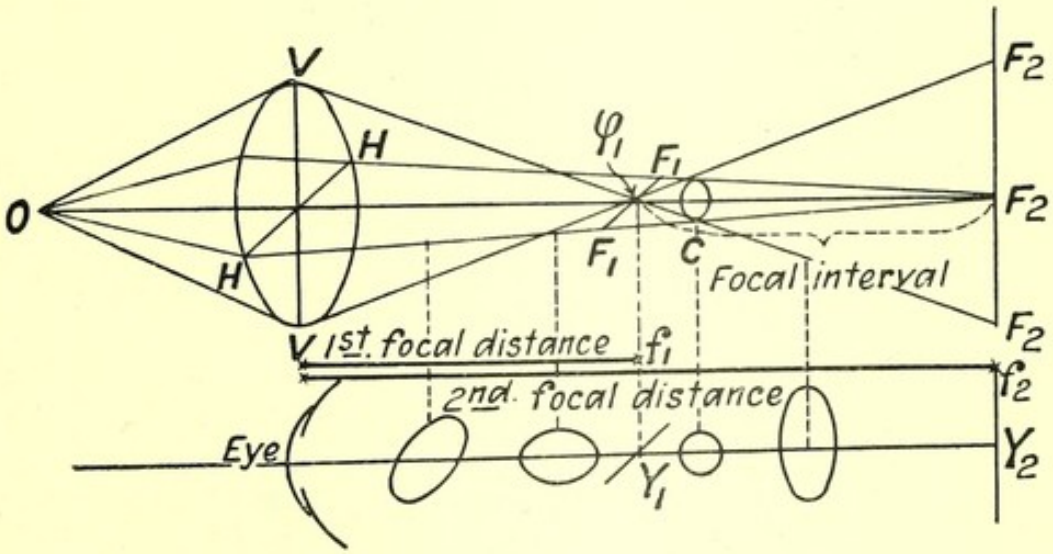

FIG. 25.—Showing course of rays in vertical and horizontal meridians of an astigmatic cornea (tori-cornea).

O = Object forming images at F_1 and F_2.
F_1 = First focal line containing the focus of vertical meridian.
f_1 = First focal distance.
f_2 = Second.
F_2 = Second focal line containing the focus of horizontal meridian.
HH = Horizontal, and VV vertical meridians.
C = Circle of least confusion.
F_1, F_2 = Interval of Sturm (focal interval).

The length of these lines is directly proportional to their distances from the lens. Thus let f_1 = focal distance of the first line, and y_1 its length; f_2 the focal distance of the second line, and y_2 its length.

L = diameter of the toric lens, = diameter of exit pupil of the eye. Suppose L = 7,

$$\text{Then } y_1 = \frac{f_2 - f_1 L}{f_2} = 3{\cdot}5 \text{ cm.}$$

$$\text{And } y_2 = \frac{(f_2 - f_1)\,L}{f_1} = 7 \text{ cm.}$$

The circle of least confusion will lie between these two lines at a point whose distance is proportional to their focal distances. Thus let C = position of circle of least confusion, and let f_1 and f_2 be the focal lengths.

$$\text{Then } \frac{f_1}{f_2} = \frac{y_1}{y_2} = \frac{16{\cdot}6}{33{\cdot}3} = \tfrac{1}{2},$$

so that as the interval is 16·6 cm. C will be $\frac{16{\cdot}6}{3} =$ 5·53 cm. from F_1 and $\frac{16{\cdot}6 \times 2}{3} = 11{\cdot}07$ cm. from F_2.

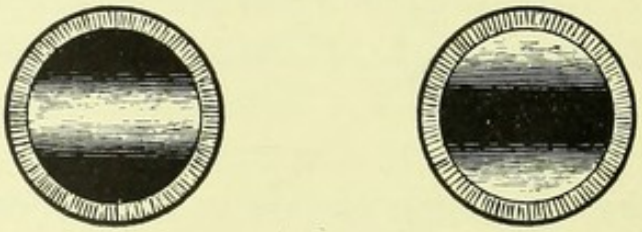

FIG. 28.—Low degree of astigmatism showing central light band and dark band.

Although the cause of astigmatism is totally distinct from that produced by oblique pencils, which pass through a series of axially centred and truly spherically curved lenses, yet the section of the cone of rays corresponds exactly to what takes place in an astigmatic eye, or through a toric lens, which latter is the nearest approach to an astigmatic cornea which the arts have produced.

Since every object can be considered as made up of an infinite number of points, it follows that what

holds good for an astigmatic image of a single point also holds true for the image of a complete object. Now the appearance of every point of the object depends upon the position of the retina in the astigmatic pencil and therefore, if we take the example in Fig. 25 and imagine the retina to be placed at various distances from the cornea, we shall be able to demonstrate every condition of astigmatic refraction and the nature of the images obtained therein.

(1) If the retina is situated anywhere between the cornea and F_1 we have the condition of compound hypermetropic astigmatism, the image of a point being represented as a horizontal ellipse of confusion.

(2) If the retina is exactly at F_1 we have the condition of simple hypermetropic astigmatism, a point appearing as a short horizontal line.

(3) If the retina is situated anywhere between F_1 and F_2 we have the condition of mixed astigmatism, which may roughly be divided into three classes: (*a*) At any point between F_1 and C_1 the image is a horizontal ellipse; (*b*) at C it is a circle of least confusion; and (*c*) at any point between C and F_2 it is a vertical ellipse.

(4) The retina exactly at F_2 gives the condition of simple myopic astigmatism, the image being a vertical line.

(5) If the retina is beyond F_2 the condition of compound myopic astigmatism obtains, the image being a vertical ellipse of confusion.

Now the most convenient object for testing astigmatism is either an astigmatic clock or a radiating fan or circle of lines whose relative distinctness or blackness affords a means of determining the powers in the principal meridians of the cornea. Each point of the several lines is represented in the astigmatic eye as either a line, an ellipse, or a circle,

the direction of the line or that of the two axes of the ellipse *always corresponding to either, or both, of the principal meridians.* Thus it is only necessary for us to consider the one case, since all others are similar, the only difference being in the directions of the principal meridians, which may be in any direction around the half circle. As the cornea under consideration (Fig. 25) has its powers horizontal and vertical we will consider the horizontal and vertical lines only of the astigmatic chart.

In Case 1 (compound hypermetropic astigmatism) the images of the lines will be made up of horizontal ellipses, which, as they overlap lengthways in the horizontal, and sideways in the vertical, cause the horizontal line to be much less blurred than the vertical, since the overlapping in the horizontal direction is not noticeable, but it is in the vertical, so the horizontal line of the fan will be seen clearest and blackest.

In the same way, in Case 2 (simple hypermetropic astigmatism) the horizontal line will be seen *quite sharp*, but the vertical blurred. In Case 3 (mixed astigmatism, division *a*) the lines will be seen as in Case 1, because the ellipses of confusion are still horizontal. In Case 3, division *b*, both vertical and horizontal lines are seen equally blurred, the confusion discs being circles. In Case 3, division *c*, the vertical line is seen the least blurred since the long axes of the ellipses of confusion are now vertical. In Case 4 (simple myopic astigmatism) the vertical line will be seen *quite sharp* and the horizontal the most blurred. Finally, in Case 5 (compound myopic astigmatism) both lines are blurred, but least in the vertical. We need not consider the appearance of lines oblique to the principal meridians, because these always have an intermediate degree of blur between that of the lines parallel to the principal meridians.

Now, the measure of the astigmatism is simply the dioptic difference between the two principal meridians, *i. e.* the difference in the dioptic values of F_1 and F_2; the correcting lens being that cylindrical which will, when convex, increase the weaker meridian up to the stronger (hypermetropic astigmatism), or when concave will reduce the stronger to equal the power of the weaker (myopic astigmatism). As a matter of fact, the astigmatism can be corrected equally well with either a convex or a concave cylindrical, since the inequality of the meridians is quite independent of all variations in length of the optic axis which determine the precise nature of the refractive error.

It will easily be seen from what we have said regarding the relative clearness of the lines on the astigmatic fan, *that in all cases the axis of the correcting cylindrical must be placed parallel in direction to the bar of maximum blur.*

Method of Procedure.—The shadow test for astigmatism is carried out exactly as for ametropia. Thus, supposing the observer measures the horizontal meridian of the right eye, and finds the shadow last appears with a + 3·75 D, vanishes with a + 4 D, while it appears in the opposite direction with + 4·5 D. He draws a horizontal line on a memorandum card thus:

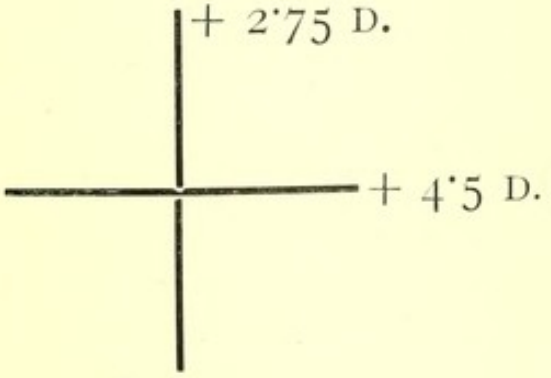

FIG. 27.

and writes opposite it + 4·5 D. He then tries the

vertical meridian, and finds the shadow turns with + 2·75 D. He, therefore, writes—

$$\text{R E } \frac{+ 2{\cdot}75 \text{ D sph.}}{+ 1{\cdot}75 \text{ D cyl.}} \text{ axis vert.}$$

Subtracting 1 D (since he is 120 cm. away), the formula becomes $\text{R E } \frac{+ 1{\cdot}75 \text{ D sph.}}{+ 1{\cdot}75 \text{ D cyl.}}$ axis vert. He then tests the patient with the types at 5 or 6 metres, and sees if he can get $\frac{6}{6}$ with this glass. If he can, he removes the cylinder, and tests on the astigmatic chart to get the axis quite exactly. He may have to shift it 5° or 10° either way from the vertical, altering the lenses until all the lines on the chart are equal. If uncertain, increase the power of the

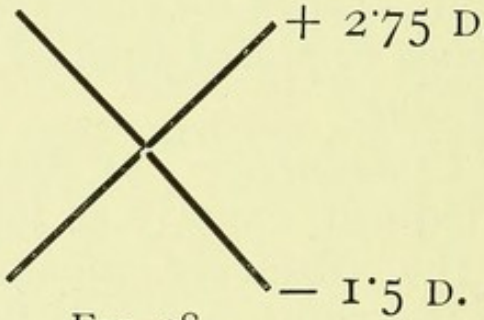

FIG. 28.

spherical so as to make the patient slightly myopic, and repeat. Having got the axis and amount of astigmatism corrected, he sees whether the patient will accept a stronger + lens (or in myopia a weaker lens) than the + 1·75 D sph., still keeping the cylinder in front.

Sometimes the observer will find that the vertical line is seen the clearest. In this case he has over-corrected the spherical lens. On substituting a weaker lens the horizontal line will probably be the best.

If the shadow goes obliquely, he must draw the line on his memorandum card in that direction (Fig.

28), altering the direction of movement of the mirror, until no further change occurs in the direction.

Supposing the student finds + 2·75 in the meridian 135°, and — 1·5 in the opposite meridian 45°, the formula will become R E $\frac{+ 2{\cdot}75 \text{ D sph.}}{- 4{\cdot}25 \text{ D cyl.}}$ axis 135°. The — 4·25 D cyl. is arrived at by adding — 2·75 D cyl. to 1·5 D cyl., leaving the + 2·75 D sph. as before. This, after adding — 1 D, becomes—

$$\frac{+ 1{\cdot}75 \text{ D sph.}}{- 4{\cdot}25 \text{ D cyl.}} \text{ axis } 135°.$$

But this formula may be transposed with advantage if the patient desires bifocals, since the reading glasses will probably be planos, when it becomes—

$$\frac{- 2{\cdot}50 \text{ D sph.}}{+ 4{\cdot}25 \text{ D cyl.}} \text{ axis } 45°.$$

This is effected by adding + 4·25 sph. to the cylinder and — 4·25 to the spherical. This will make no difference to the refraction whatever, but it neutralises the — 4·25 cyl. and leaves + 4·25 in the opposite meridian, while the — 4·25, less + 1·75, leaves — 2·50 D sph. Sometimes when the mirror moves horizontally the shadow moves obliquely so as to form an angle with the mirror. This is due to the elliptical form of the diffusion disc. If one draws an ellipse in ink on a card, and moves a second card having a pinhole aperture to and fro in front of the eye in a lateral direction, the ellipse will appear to move in the direction of the arrow.

The Scissors Movement of Jackson.—If in any meridian one part of the pupil is slightly more myopic than another part, so that on getting reversal with one part the other part falls short, a band-like shadow will start from each side of the pupil at right angles to the meridian and meet in the centre like a pair

of scissors. It thus forms a black band across the pupil, leaving the parts on either side illuminated. It always indicates imperfect centring of the lens or cornea, and probably a high angle alpha. The eye must be corrected for the central black band by getting reversal in the usual way.

Astigmatism according to Rule.—By far the majority (about 80 per cent.) of hyperopic cases exhibit astigmatism in which the meridian of greatest refraction is vertical, and which are consequently relieved by a convex cylinder having a vertical axis. This is known as *astigmatism according to rule.* If a

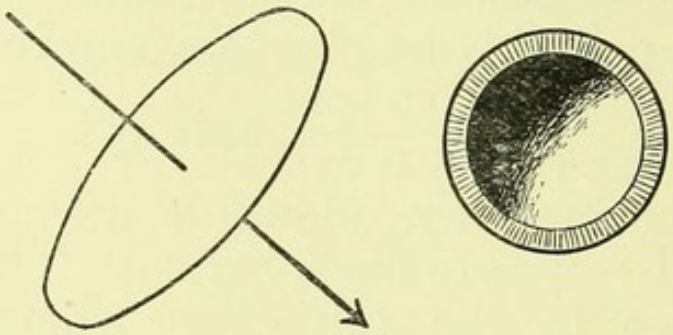

FIG. 29.—Shadow in oblique astigmatism.

case needs correction by a + cyl. having its axis in the opposite horizontal meridian, it is said to be *against the rule.*

In myopic cases the opposite meridian is found to be the rule, *i. e.* the greatest refraction usually lies in the *horizontal meridian,* and such eyes are corrected by a concave cylinder having the axis horizontal. The writer finds oblique astigmatism more frequently present in myopic patients than in hyperopes, and the most usual angle for the axis after the horizontal one is 30° from the vertical. After operations for cataract and iridectomy the artificial astigmatism is generated by the gap caused by the corneal wound. It is usually very considerable (several dioptres) just

after the first week, but it slowly disappears as the cicatrix contracts, until only 1 D or 1·5 D of astigmatism remains permanently.

Pflüger's Operation.—Pflüger, of Bern, made use of this fact to cure astigmatism by section of the cornea, the incision being made at right angles to the meridian of greatest refraction, *i.e.* in astigmatism with the rule through the upper fifth of the corneal margin. In this country the operation has been performed with considerable success, but frequently it has been found that, although the astigmatism was greatly diminished at first, the eyes returned to their former condition. In a few cases the astigmatism was even increased.

According to Helmholtz, vision is not affected in any noticeable degree until the astigmatism exceeds 1 D, although experience teaches us that a much lower power will cause headaches and eye-strain if the eye is much used for reading.

Difficulties met with.—The testing of astigmatism by the shadow test is exceedingly simple and easy in theory, but often very difficult in practice, especially if the reflexes are poor and the pupil very small, as it is often impossible to make sure when the shadow turns. As a rule, beginners over-estimate the refraction in both meridians often to the extent of 1·5 or 2 D. Again, the same difficulties arise as we mentioned on p. 18, when estimating refraction by direct vision with the ophthalmoscope. For instance, if the eye be widely dilated, the periphery of the cornea will inevitably give a different refractive value to the pole of the cornea, and if the pupil be contracted it is most difficult to notice when the shadows cease or reverse. Again, the patient will often accommodate involuntarily, especially if a child (hence the value of atropine). Moreover, as the

lens is often slightly tilted, the least trifle makes a great difference in the amount of astigmatism recorded. There are often variations in the density of the lens over parts of the surface, and lastly, the disc—the brightest object in the fundus—often shows a difference of a whole dioptre of refraction from the macula, which, after all, is the only part of the fundus you need pay attention to.

If the student imagines he is always going to get his retinoscopy values to agree with the ophthalmometer tests, or with the subjective tests with the types, he will at first be greatly disappointed. With practice he will get better results, as he will be able to see the shadow through a smaller pupil, which means that he confines his reflex to the centre of the cornea, where the aberrations are at a minimum. In any case he would do well to make it a rule to confirm the shadow test by the test types, which, after all, is the decisive test, unless the patient is unable to read his letters. In this case it is a good rule to test with all three methods—the ophthalmoscope test, the shadow test, and the ophthalmometer image. If these three methods agree to within one dioptre, which they ought to do, one gives the patient the lowest value of the three, and for the astigmatism the lowest cylinder shown by the three tests.

CHAPTER II.

ANATOMY OF THE CHOROID, CILIARY BODY, AND RETINA.

The Choroid.—Uvea.—Layers of the Choroid—Tapetum Cellulosum and Tapetum fibrosum.—Summary.—Ora Serrata.—Ciliary Body.—Ciliary Muscle.—Ligamentum Pectinatum.—Spaces of Fontana.—Canal of Schlemm.—Retina.—Nature of Rods and Cones.—Macula Cones.—Macula and Fovea.—Three Systems of Fibres or Neurons.—Summary of the Several Layers.—Fibres of Müller.

THE posterior two thirds of the eyeball is lined by three coats, the *outermost* (sclerotic) consisting of dense fibrous tissue, which supports and maintains the shape of the eye; the *middle coat* (choroid) forms the vascular and secreting layer. It has two functions: first, it supplies nourishment to the outer layers of the retina and indirectly to the vitreous and lens, and secondly, it reflects the images of the external objects on to the nerve terminals (rods and cones) of the retina. The *internal coat* (retina) consists of highly specialised nervous elements, which during the transmission of a visual impulse are in direct continuity with the fibres of the optic nerve.

The Choroid.—This occupies the whole posterior two thirds of the globe, extending as far forward as the ora serrata. This latter forms a well-defined uneven border, produced by the abrupt termination

of the retina and choroid, so that the border describes a circle in a verticle plane round the eye-

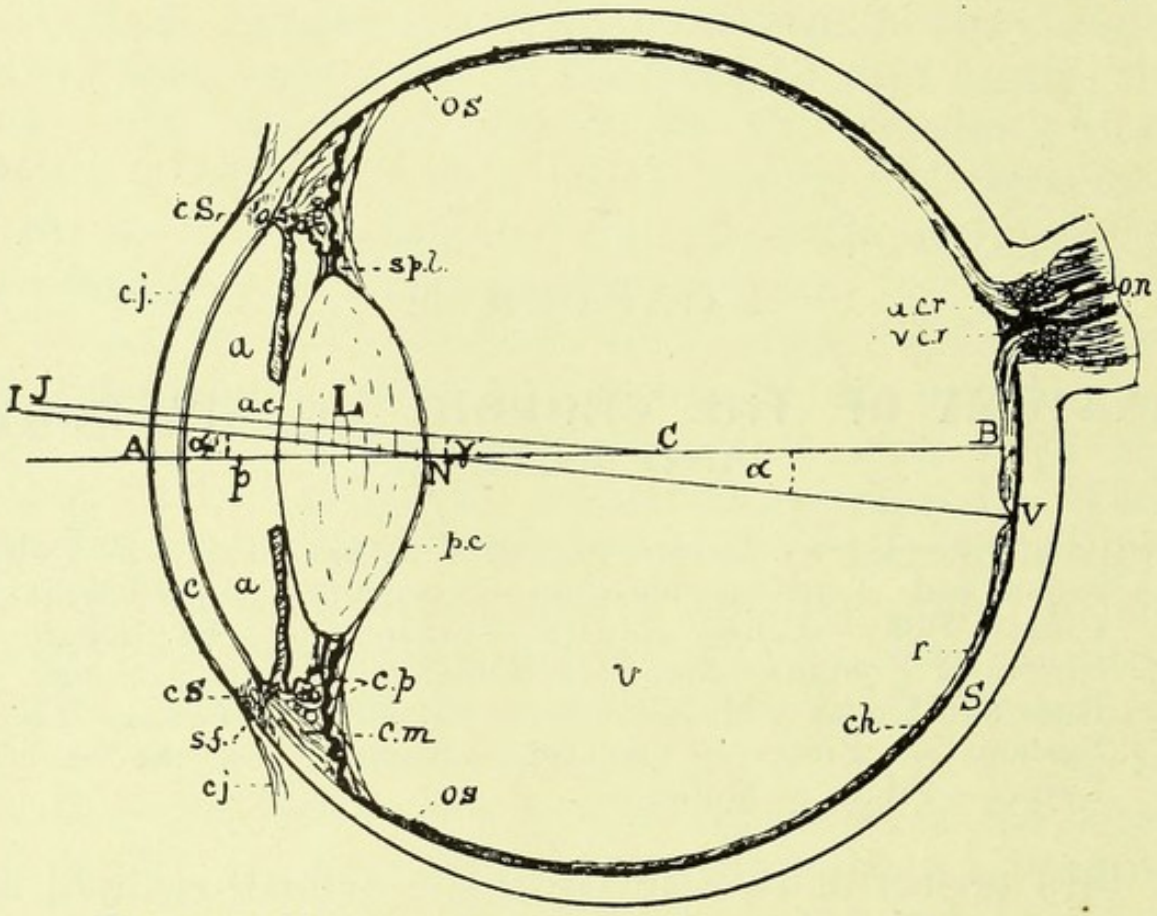

FIG. 30.—Horizontal section of the eye. × 2½ times.

A. Anterior pole.
B. Posterior pole.
C. Centre of rotation.
p. Principal point.
N. Nodal point.
V Fovea centralis.
A. B. Optic axis.
I. V. Visual axis.
J. C. Line of fixation.
a. Aqueous humour.
v. Vitreous.
L. Lens.
S. Sclerotic.
ch. Choroid.
r. Retina.
c.m. Ciliary muscle.
c.p. Ciliary processes.
i. Iris.
sp l. Suspensory ligament.
s.f. Spaces of Fontana and pectinate ligament.
c.s. Canal of Schlemm.
a.c. Anterior capsule of lens.
p.c. Posterior capsule.
c.j. Conjunctiva.
o.n. Optic nerve.
o.s. Ora serrata.
v.c.r. Central vein of the retina.
a.c.r. Central artery of the retina.
α. Angle alpha.
γ. Angle gamma.

ball. Its position is such that the plane of the circle just touches the posterior pole of the lens, or, measured outside the globe, forms a circle

situated about 5 mm. or 6 mm. behind the sclero-corneal junction, and 15·5 mm. in front of the macula. The interval between the ora serrata and the margin of the cornea is occupied by the ciliary muscle, ciliary processes, and base of the iris. Collectively, it is termed the ciliary body, and is the spot where most of the deep-seated inflammatory changes take origin. It is sometimes called the *danger zone* from the fact that wounds in this region are specially liable to give rise to sympathetic ophthalmia, iritis, and choroiditis.

Both the choroid and retina are firmly attached and bound down by connective-tissue fibres in two places — first, at the ora serrata, and, secondly, around the disc of the optic nerve. Between these two places the choroid is loosely attached to the tissue lining the sclerotic, being merely pinned down in various places by the ciliary nerves and vessels, which enter and leave the sclerotic. The choroid itself is destitute of sensory nerves, and therefore pain is not a symptom of choroiditis.

Uvea.—The choroid, ciliary body, ciliary processes, iris, pectinate ligament, and Descemet's membrane are all histologically and pathologically continuous; together they constitute the uvea. If an eye be cut in half, and the lens, vitreous, and retina be removed, a dark pigmented layer will be noticed, which extends without any interruption of continuity from the optic nerve to the pupillary border of the iris, forming a lining to the choroid, ciliary processes, and iris. The appearance of this layer *in situ* closely resembles that of the inside of the skin of a black grape. Hence the name uvea, or uveal tract (uva—a grape), has been assigned to these parts. A few anatomists confine the term to the pigmented cell layer, but the wider meaning is the

one generally received. This hexagonal pigment layer, which covers the choroid as far as the ora serrata, was thought by the older anatomists to be part of that layer, but it is now known to be a development of the outer lamella of the optic cup, and, therefore, belongs strictly to the retina.

LAYERS OF THE CHOROID.—Taking the layers from within outwards we find :

(1) *The Membrane of Bruch* (lamina vitrea).—A highly-transparent homogenous layer, on which rests the hexagonal pigment layer or outermost layer of the retina.

(2) *A Layer of Capillaries* (chorio capillaris).—This consists of a very dense network of large bore capillaries anastomising very freely in all directions. Immediately beneath this layer are two other layers, which are without importance in ourselves, but owing to their extraordinary colouring throughout the Carnivora and Ungulata in the mammals are worthy of note.

(3) *Sub-capillary Epithelial Layer.*—First comes a continuous membrane (sub-capillary epithelial layer), which is transformed into the *tapetum cellulosum* (or tapetum lucidum, as it is generally called) of the Carnivora. This layer gives rise to the brilliant colours observed with the ophthalmoscope and occasionally with the naked eye. Thus the brilliant gold purple and green sheen seen in cats' eyes is entirely due to the colours reflected from this layer. If the eye is enucleated and opened the colours will be seen to fade rapidly in daylight, or if all the pigment lying on the membrane of Bruch is brushed away with a camel's hair pencil the colour likewise vanishes, the choroid then resembling a piece of dirty washleather.

(4) *Elastic Fibre Layer.*—Next in order are several

fine layers of wavy elastic fibres quite free from pigment cells. This is the layer of the medium-sized vessels and small arteries, and in most of the higher Ungulates (Artiodactyla), as well as the horse and giraffe, it is brilliantly coloured, often blue or bluish green, constituting the *tapetum fibrosum*. Unlike the Carnivores, brushing away the retinal pigment has no effect on the colour, which remains, and will keep its colour indefinitely in a weak solution of formalin.

(5) *Layer of Fusca Cells* (Sattler's layer).—These are large pigmented branched cells each having an oval nucleus and capable of amœboid movements when acted on by light. They constitute the fusca cells.

(6) *Layer of Larger Vessels* (Haller's layer), which are chiefly veins which anastomose very freely and form a dense network. This layer is very thick, occupying about three fourths of the entire thickness of the choroid. The spaces between the vessels are filled with branched pigmented fusca cells which give a dark background to the orange-coloured vessels. These latter form the characteristic choroidal vessels which are seen with the ophthalmoscope in nearly all eyes.

(7) *Lamina Supra-choroidea* consists of a layer of brown pigmented cells destitute of blood-vessels, and is separated from a similar pigmented layer attached to the sclerotic. Between the two is a lymphatic space which can be traced underneath the choroid to the lymphatic space between the sheaths of the optic nerve, and is probably the channel along which the infective materials are carried in sympathetic ophthalmia.

To Summarise.—The uvea is primarily the nourishing and secreting organ of the eye. Now all secreting layers and glands consist essentially of

three parts. First, a network of blood-vessels usually followed by one of capillaries. Secondly, a basement membrane; and finally, a layer of active secreting cells. In the uvea we find an abundant supply of blood-vessels arranged in three rows, which, commencing from the outside of the choroid, are: First, Haller's layer, which consists of numerous large anastomising vessels; secondly, a plexus of smaller blood-vessels (Sattler's layer), which is intimately connected with the tapetum fibrosum in the Ungulates and with the tapetum cellulosum in the Carnivores; thirdly, we have the layer of capillaries which are in close contact with the outer surface of the basement membrane (lamina vitrea); and finally in contact with its inner surface a layer of densely pigmented secreting cells, which constitute the hexagonal pigment layer of the retina throughout the whole extent of the choroid. In the same way the pigment layer of the ciliary processes lines the internal surface of the ciliary body and its continuation along the posterior surface of the iris. The chorio-capillaris secretes the fluids necessary for supplying the laboratory of the hexagonal pigment layer, which latter appears to have a double function, viz., to nourish the external layers of the retina (bacillary and outer granular layers) and also to secrete the visual purple which envelopes the free ends of the rods. It is probable that the whole of the pigmentary layer in front of the ora secretes a fluid which nourishes the lens and vitreous, but the greater part of the work is carried on by the pigment layer of the ciliary processes, which are plicated to afford a larger secreting surface. The pigment layer of the iris probably serves the same purpose to a lesser degree. The aqueous is said to be secreted by the ciliary processes, and especially by Treacher

Collins's glands, which the latter discovered embedded in the pigment layer just in front of the ora. Some authors maintain that the chorio-capillaris layer secretes the highly complex visual purple, but this is obviously impossible.

The colour of the iris, or colour of the eyes as it is popularly expressed, depends to some extent, but not entirely, upon the amount of pigment in this layer, and the difference in shade between the fundus of a negro's eye and that of a European is also due to some extent to the amount of pigment in the hexagonal layer, but still more, as will be seen later, to the extent of pigmentation of the fusca cells of the choroid.

Ora Serrata and Ciliary Body.—You cannot quite see the ora with the ophthalmoscope, but by pressing the sclerotic over that region with the finger so as to bulge it slightly inwards, and looking as obliquely as possible with the ophthalmoscope, the fundus can be seen very nearly to the ora, but the pupil must be widely dilated first. A young subject is best. For the indirect method a lenticular prism with the base held towards the side to be examined is necessary. The tips of the ciliary processes cannot distinctly be seen by oblique light illumination (without the ophthalmoscope) in normal eyes, although the edge of the crystalline lens is often visible, but in irideremia they can readily be seen, the ciliary process under observation forming an irregular dense black protuberance very nearly touching the circumference of the lens. It is most instructive in such a case to observe how it thickens, and at the same time bulges inwards, pushing almost right against the edge of the lens during accommodation.

We have said that both the choroid and retina terminate abruptly at the ora, but the fibrous stroma

of the choroid passes on without interruption, so that in the place of the hexagonal pigment layer a single layer of densely pigmented columnar cells each having a large oval nucleus is substituted. This layer is at first flat, and then continues over the ciliary body (here called the pars ciliaris retinæ) in a series of folds—about seventy in all. These *ciliary processes* are densely pigmented between the folds, but are paler at their apices. At this plicated part of the ciliary body the cells become cubical, each having in the centre a spherical nucleus, and are covered with a delicate hyaline membrane. To this latter the straight supporting fibres of the zonule of Zinn are attached, and these, being inserted into the lens capsule, thus keep the lens in position. Just in front of the ora, the layer of cells with its supporting stroma bends inwards away from the sclerotic, thus leaving a triangular space, which is filled up with the fibres of the ciliary muscle. These form two groups: (1) The *radiating fibres* which arise around the canal of Schlemm and the root of the iris, and are inserted partly into the above-mentioned fibrous stroma which supports the pigmented cells and ciliary processes, and partly into the stroma of the choroid itself. When, therefore, the ciliary muscle contracts, it draws the choroid and retina forwards towards the muscle. (2) Several bundles of muscular fibres (circular fibres of Müller), which form a series of irregularly placed solid rings outside the bases of the ciliary processes. These lie in a plane nearly at right angles to the radiating fibres, and tend to increase the effect of the latter by pushing the ciliary processes onwards towards the lens, and drawing the iris away from the former inwards, thus contracting the pupil. In myopes, who habitually keep their accommodation more or less relaxed, the

circular bundles are few in number, or even entirely absent.

The ciliary body contains not only motor fibres from the third nerve which supply the ciliary muscle, but also a large number of sensory branches from the fifth nerve. Hence inflammation of that part is usually accompanied by a great deal of pain. It also contains a large number of veins and lymphatics which assist in draining the eye. The veins communicate with Schlemm's canal and with the ciliary veins.

The Retina.—In vertical sections through the retina ten, and between the macula and the disc, and for an equal distance on the opposite side of the macula, eleven layers can readily be distinguished. They are, counting from without inwards, as follows:—

(1) *Hexagonal Pigment Layer.*—This consists of a single row of hexagonal cells, which in vertical sections appear like a course of bricks, each containing a large oval nucleus; each is about 0·015 mm. in diameter. The cells are generally so loaded with small, round, pigmented granules that (excepting the outer third, which is free) the separation between them cannot be clearly defined. Attached to the inner border is a delicate hexagonal network of extremely fine fibres, like a thin large-meshed gauze, which the author termed the "crystal pigment plexus," since it is loaded with oat-shaped pigment crystals which lie in clusters and adhere to the network. They are quite distinct from the granules of the hexagonal layer, which are smaller and spherical* (see Fig. 31 A). In very bright light the inner part of the hexagonal layer, which faces the bacillary layer, exhibits pseudopodic movements, the proto-

* For details respecting the differences between the two see "Observations on the Macula Lutea," 'Arch. of Ophth.,' p. 301.

plasmic filaments of which carry the network, and with them the crystals between the rods. At the same time the nuclei of the cones appear to retract somewhat towards the membrana limitans. As the light diminishes the pseudopodic filaments retract, until in total darkness they become closely packed against the surface of the cells.* The writer was the first to demonstrate this in 1894 in the human eye, although Engelmann and Von Genderen-Stort noticed the same phenomena in frogs a few years

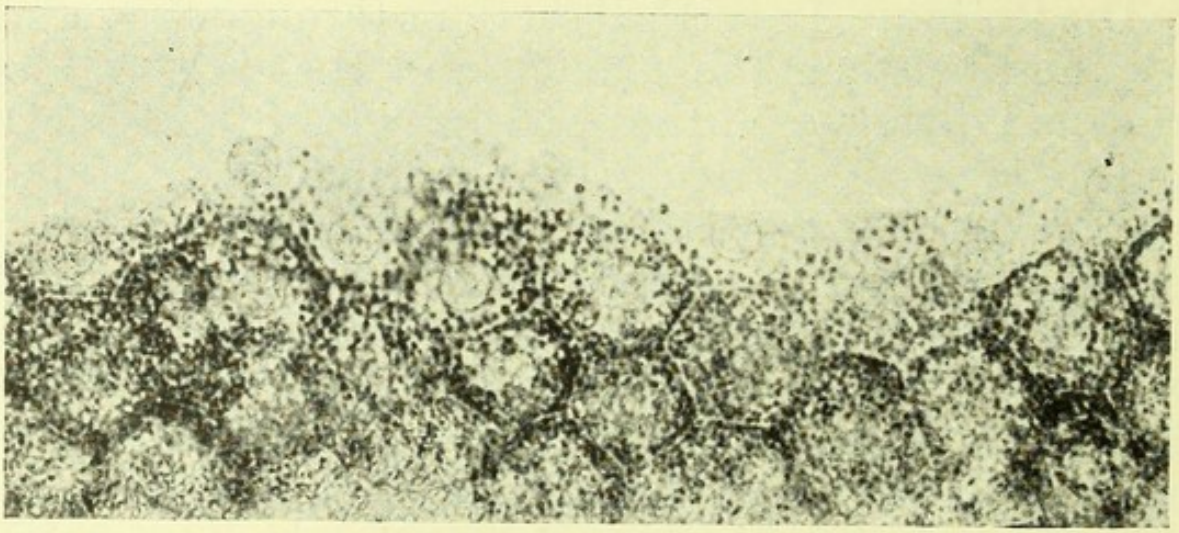

FIG. 31.—Horizontal section through the hexagonal layer showing the arrangement of the cells and granules.

before. Possibly the object of these movements is to prevent halation and flare, which would obscure the definition of objects in a strong light, by interposing the screen of pigment between the rods and cone ends. In sections made through monkeys' eyes enucleated and fixed in the arc light, the crystals may be seen extending as far as the external limiting membrane. Detachment of the retina is nearly always between the bacillary layer and the hexagonal pigment. These two layers are merely in apposition, without any organic union, and the

* See the writer's paper in the 'Arch. of Ophth.,' 1894

retinal membrane may be readily lifted off with a pair of iris forceps, except at two spots, one being around the optic nerve at the disc, the other at the

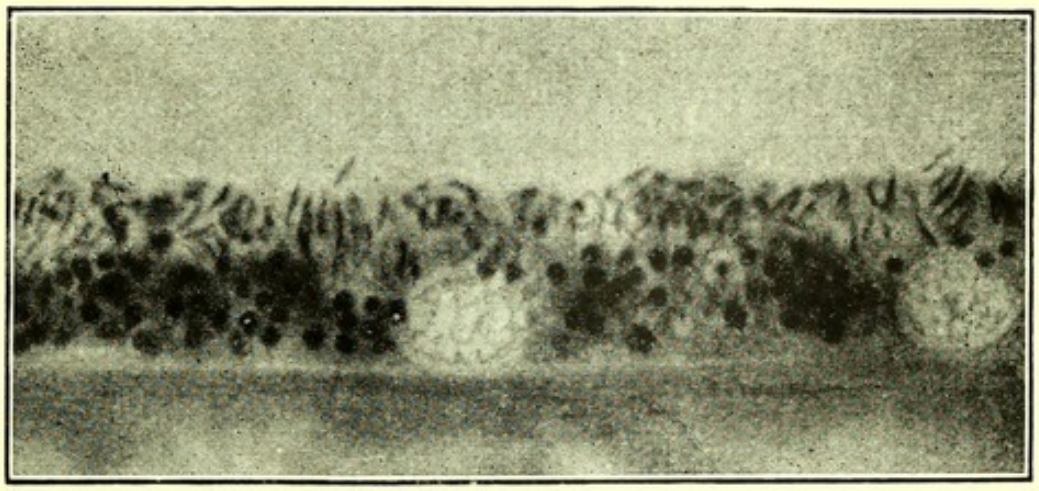

FIG. 31A.—Hexagonal pigment layer showing two of the nuclei, and also the two kinds of pigmented bodies, granules, and acicular, crystal-like particles. Vertical section.

ora serrata. At these two places the retina is very firmly bound down.

(2) *Bacillary Layer.*—This layer is entirely composed of the terminals of the optic nerve. These consist of three distinct organs: (A) *The rods:* These are by far the most numerous and surround the cones in single, double, or triple rows, according to the distance from the macula. A rod consists of a cylindrical shaft which may be split up into fibres by reagents. The outer half is a fine, highly refracting cylinder, the distal end of which is buried in the hexagonal crystals, and adheres so firmly to the hexagonal layer that the rods break in half, leaving a portion attached to the pigment layer when the rest of the retina is forcibly detached. The inner half of the rod presents a slight spindleform swelling and is granular. It may be traced through the next layer (limitans) and then expands into a nuclear body (nuclear layer), and again contracting to a thin fibre

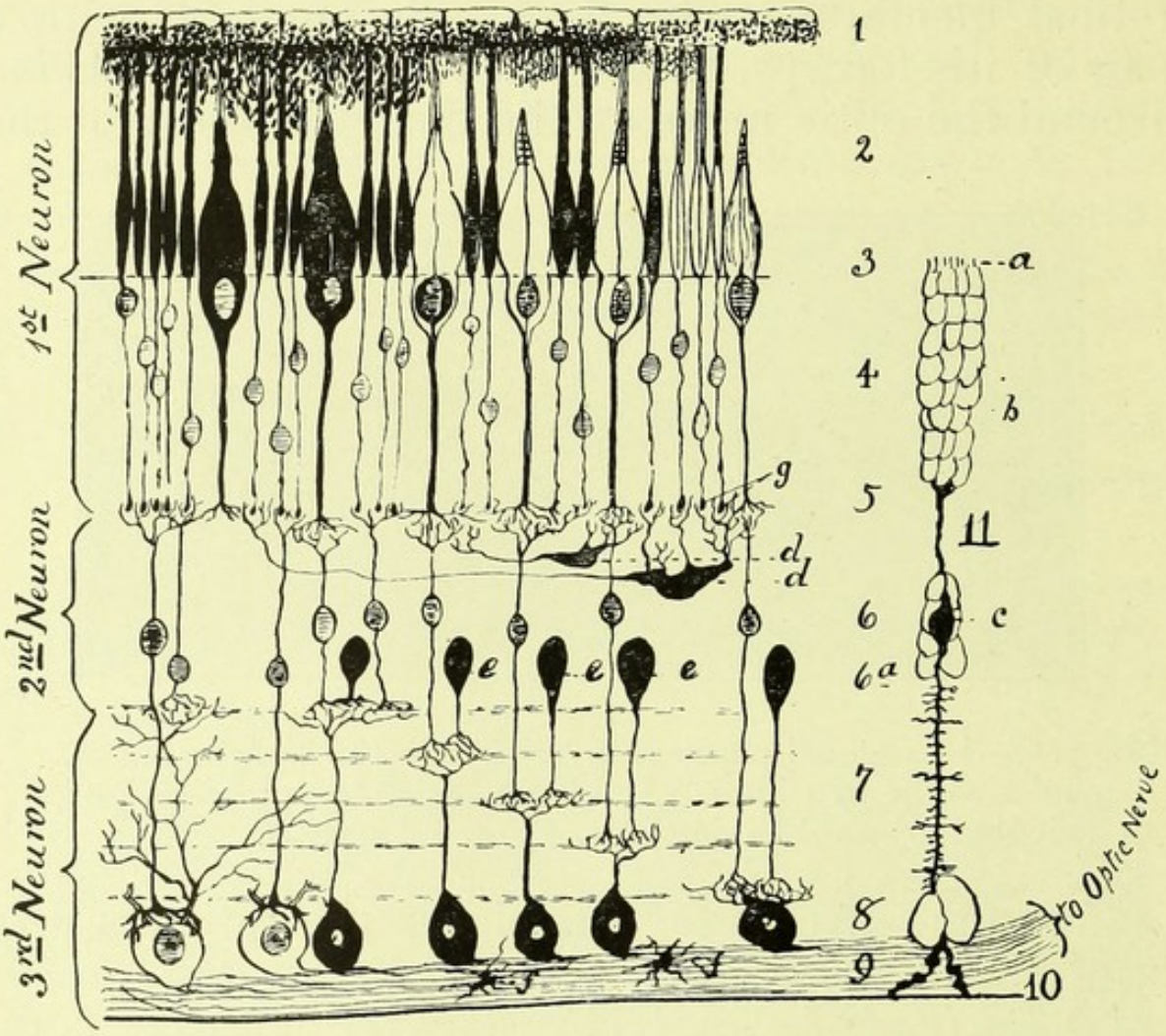

FIG. 32.—Semi-diagrammatic vertical section through the retina showing the layers and systems. After Golgi, Cajal, and Greeff, slightly modified.

1. Hexagonal pigment layer showing granules and elastic network laden with oat-shaped pigment bodies.
2. Bacillary layer, or layer of rods and cones.
3. External limitating membrane.
4. External nuclear layer.
5. External plexiform layer (layer of horizontal cells, Cajal).
6. Inner nuclear layer (bipolar layer, Cajal).

6*a*. Paramacula layer (over the central part of the fundus only).

7. Internal plexiform layer (layer of amakrine cells, Cajal).
8. Ganglion cell layer.
9. Nerve-fibre layer containing spider-cells, *f f*.
10. External limitating membrane.
11. One of the fibres of Müller. *a*, Fibre basket; *b*, lateral buds in the external nuclear layer; *c*, nucleus; *d d*, linking up horizontal cells; *e e e*, diffuse amakrine cells; *f f*, spider cells; *g*, terminal bulbs of the rod-fibres.

terminates in a minute bulb, like the head of a pin, in the outer reticular layer, either free or in the centre of the rootlets (arborisations) of the bipolar system of fibres. Whether the rod-fibres are connected with the ganglion cells at all, or whether (if they are connected with the brain) they conduct light impulses, has not been decided by anatomists. (B) *The cones proper:* These are very short near the periphery of the retina, but get gradually longer until they reach the macula, where they extend to the hexagonal layer. The writer is convinced from numerous methylene-blue specimens prepared *in vitam* that the peripheral ends of the cones exhibit pseudopodic movements, and can, and often do, stretch out to the hexagonal layer (see Fig. 33, which shows this clearly).* Looped cones have been described by Norris, but they appeared to be due to coagulation changes during the preparation of the specimen. Twin cones are frequently seen. The ultimate nerve-fibril appears to be the real organ of vision, the rest of the rod or cone being merely insulating material corresponding to the white substance of Schwann. As can readily be shown, this fibril is kept in position by supporting fibres, which in cross-section resemble the spokes of a wheel (see Fig. 34). By injecting the eye during life with a saturated solution of methylene-blue I was able to show these fibrils as red threads running through a blue cone or rod, which can thus be very clearly demonstrated. The diameter of a cone lies

* That anatomists have stated this to the contrary is quite natural, since the solid body of the cone is shorter than the rods, but the central axis cylinder surrounded by its delicate sheath is prolonged up to the hexagonal layer. Now this prolongation is so delicate that it is invariably destroyed during the fixing and hardening of the preparation, unless the eye is fixed *in vitam, i. e.* while the cells are still physiologically alive.

between 0·006 and 0·007 mm. in its widest part. The cone passes through the membrana limitans and encloses a large nucleus and narrows to a stalk, which ends in fine rootlets either in the external molecular

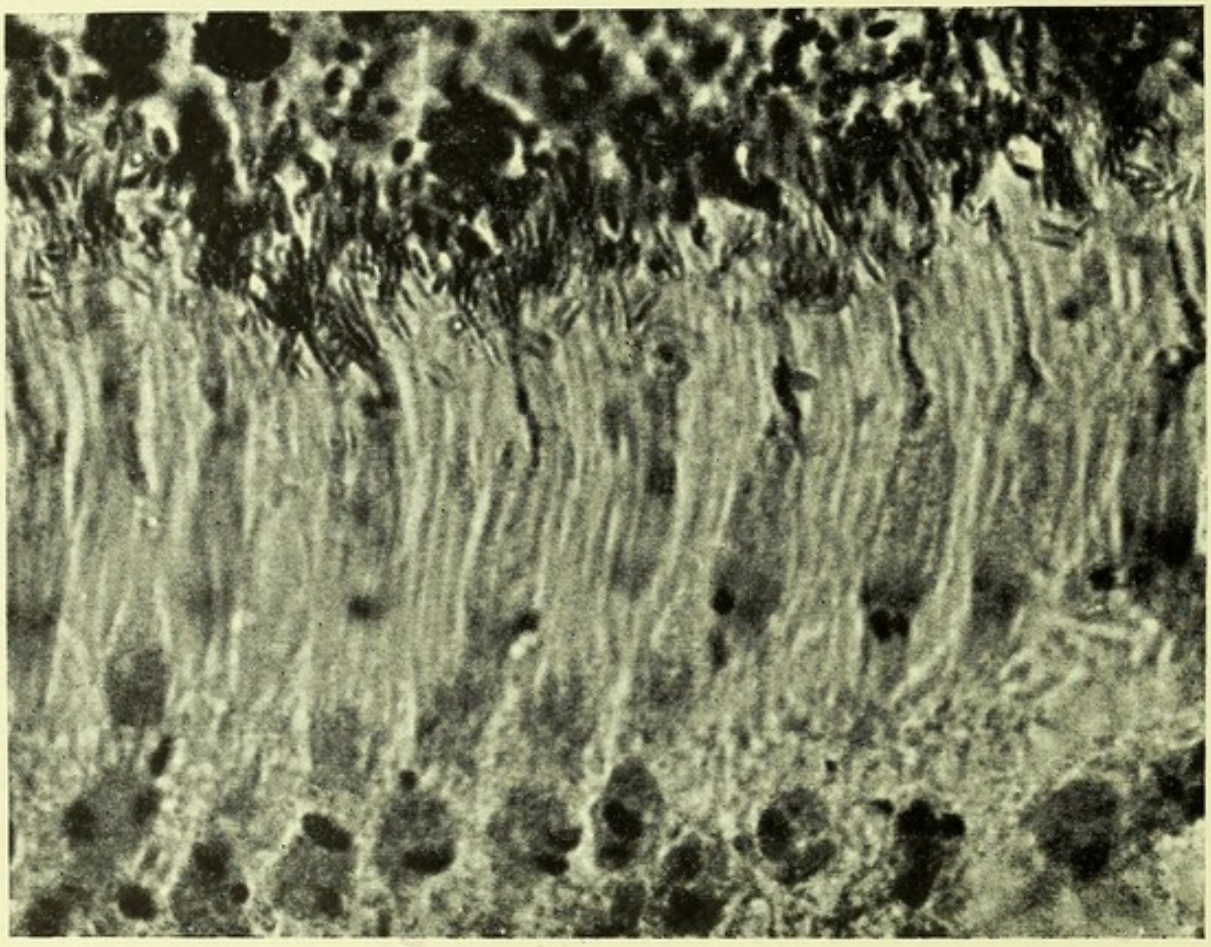

FIG. 33.—The hexagonal pigment and bacillary layers of the retina fixed and stained *in vitam* with methylene blue. The prolongations of the cones are seen to extend into the hexagonal layer. The central axis cylinders surrounded by a delicate sheath are well shown surrounded by the pigment crystals. From a photograph.

layer or the internal molecular layer—a few stalks apparently terminating in rootlets in the ganglion cells themselves.

(c) *The Macula Cones.*—These are not cones at all in the true sense of the word, although they perform the same function. They are cylindrical bodies of the

same calibre throughout each half, extending as far as the hexagonal layer, and like the rods and cones elsewhere, are divided midway into two parts by a transverse line, at which spot they are liable to break off short. The outer (terminal) half has exactly half the diameter of the inner portion. In specimens

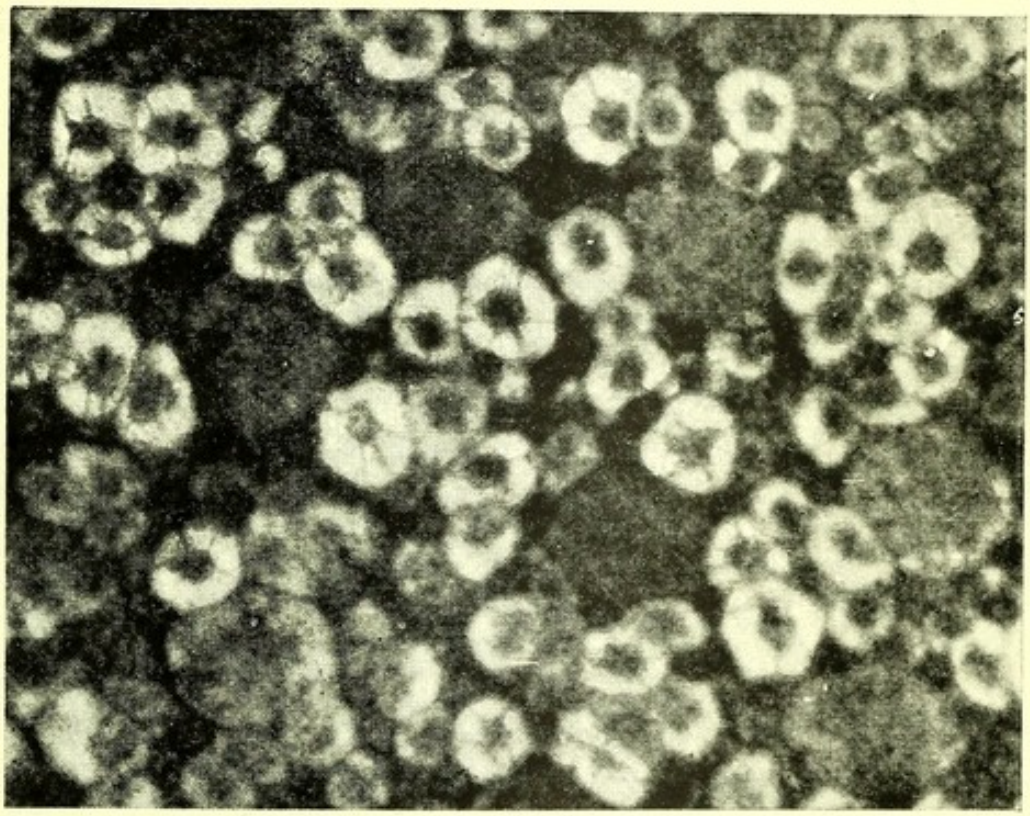

FIG. 34.—Transverse section through the bacillary layer showing how the axis cylinders of the rods are supported in the centre of their sheaths by radiating fibres. The same thing occurs in the cones, but this section fails to show it.

hardened by fixation fluids, the outer halves of the cones show a large number of transverse lines, causing the appearance of piled-up discs. In order that the layers should not obstruct the vision, all the layers excepting the cones with their nuclei and supporting membranes (m. limitans ext. and int.) and a single row of modified bipolar cells are absent at the fovea, so that the fibre prolongations of the cones are greatly lengthened, sloping away from

the centre of the pit in all directions, and by this means allow the image forming rays to enter the foveal pit with scarcely any obstruction. The macula cones, according to the most reliable measurements, are about 0·0025 mm. or 0·003 mm. in diameter. In the centre of each cone is an axis nerve-fibril.

The Macula is a specialised portion of the retina forming a well-marked pit situated in the axis of

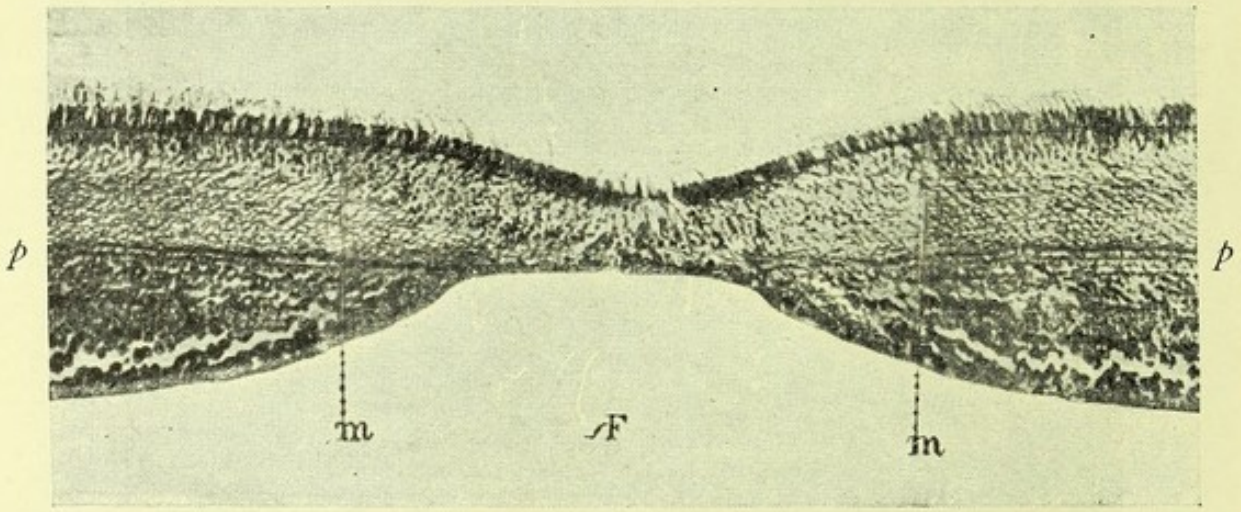

FIG. 35.—Vertical section through the macula of an adult eye. From a photograph. F. Position of fovea centralis; *m m* indicate the limits of the macula area; *p*, paramacula layer, only found in this part of the retina.

vision. In some persons the pit is conical, tapering almost to a point; in others it has a hemispherical, or occasionally a flat base.* The rods become rapidly fewer in number towards the edge of the macula, and although they may be traced to the edge of the pit they apparently cease altogether inside it. Out-

* The hexagonal layer is perfectly level throughout the macula region, and all the cone ends are in contact with it. The anti-macula or external dip does not exist during life, and it is solely due to a retinal detachment formed during the preparation of the specimen. There is no depression during life in the external limitans membrane as stated and figured by Golding Bird and Schäfer. It is entirely due to the retraction of the elastic fibres (see Fig. 35, which shows it well).

side the pit the retina is slightly raised on the front

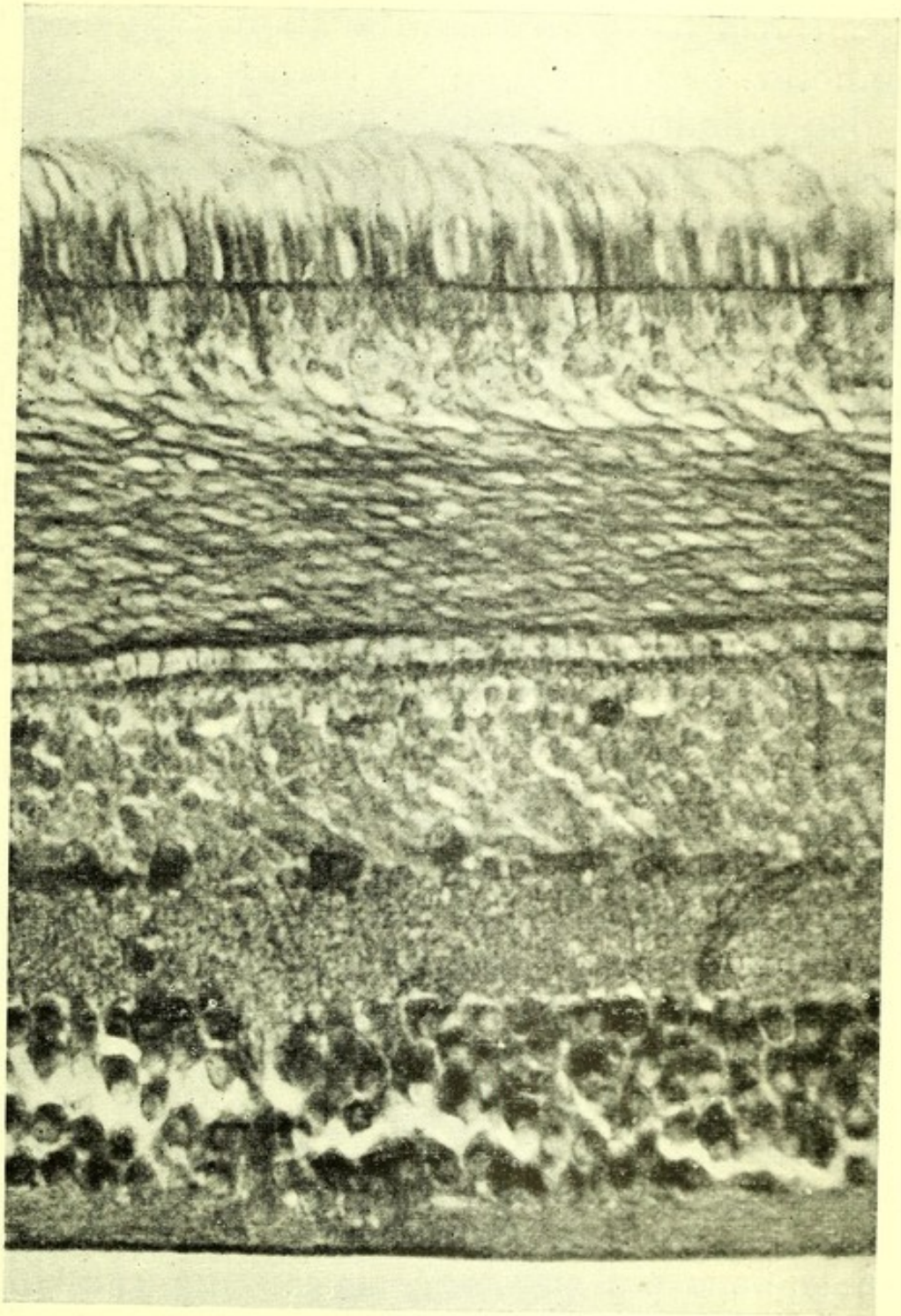

FIG. 35A.—An enlarged portion of the above microphotograph showing details of the layers, and the single row of cells forming the paramacula layer in the centre of the photograph.

(vitreous) side, forming a circular or horizontally oval reflex when seen with the ophthalmoscope.

This is known as the macula ring, and it defines the limit of exact vision. The space inside this ring is termed the macula area, and is about 1·75 or 2 mm. in diameter, or slightly larger than that of the disc. It is also called the sensitive area, because all sharp vision lies within it. It embraces an angle at the nodal point of from $4\frac{1}{2}$ to 5°. Moreover, there are no visible blood-vessels inside this ring, and at the fovea they are entirely absent. Throughout this area the cones are greatly modified, being little thicker than rods. They are packed together as closely as possible, forming at the centre of the pit (fovea centralis) a hexagonal mosaic in horizontal sections or where viewed from above. Here all the layers of the retina excepting the cone layer and a single row of nuclear cells abruptly cease. On the slopes of the pit the nuclear cells increase in number until just outside it they form rows eight or ten deep. Whether there is ever any visual purple at the macula is a disputed point. According to Edridge-Green the visual purple flows in during the act of vision. Kûhne thought that only a visual yellow existed there; Gullstrand suggested that the yellow colour was a post-mortem stain, but according to his later researches he thinks it is probably yellow during life, but remarks that the colour is difficult to be sure about, as it depends on so many factors, the chief being the nature of the illuminant used. Remak, Dimmer, Greeff, and others mention a bright yellow transparent substance as existing immediately external to the cones of the fovea.

(3) *External Limitating Membrane* is a thin cribriform membrane through which the rods and cones pass and are held in position, thereby securing their insulation.

(4) *External Nuclear Layer* is formed of several

rows of large cells, one of which encapsules a rod or a cone fibre. Each of these cells possibly forms the trophic centre for the fibre (neuron), or, at least, performs the same function that the nucleus does for any active tissue fibre. The writer found from the examination of a large number of microscopic sections that the cells of the external nuclear layer are quite different in the retina near the inner (nasal) side of the disc from those on the macula side. In addition to the ordinary form of cells, the inner part contains a large number of smaller granules, which stain differently and are much more homogeneous in structure.

(5) *Paramacula Layer.*—This is a narrow, well-defined, and very conspicuous single layer of round cells, extending from the fovea to the optic nerve on the one side, and to an equal distance on the other side of the fovea. It was discovered by the writer in man and most monkeys. In some preparations it is not very evident, but in others it is extremely conspicuous. Its use is unknown, but it is evidently connected with acute vision, since it is most developed and thickest at and near the macula. (See Fig. 35, p. 82, and Fig. 35A, p. 83.)

(6) *External Reticular (Plexiform) Layer.*—This layer was formerly thought to be a reticulum of connective-tissue fibres, but is now, thanks to Cajal's and Golgi's methods, known to be due entirely to minute neuron arborisations. In this layer each of the rods terminates in a tiny knob embedded in the middle of the arborisations (rootlets) of the second or bipolar system of neurons. Often ten or twenty bulbous rod terminals lie in a single arborisation of a bipolar fibre. The cone terminal ends in a dense mass of rootlets, which are met by a similar arborisation from a bipolar fibre. Whether the

arborisations of the two systems are permanently in contact, or whether their ends only meet when the person is awake or during the act of vision, or whether they never touch at all, is so far undecided. Speaking generally, no capillaries are found external to this layer, the layers outside it being nourished by osmosis from the chorio-capillaris and hexagonal layer.

This layer forms the bond of union between the first and second system of nerve-fibres or neurons.

(7) *Internal Nuclear Layer* consists of two or three rows of bipolar cells, the ascending fibres of which terminate in arborescent expansions in the external reticular layer, and there surround the rod terminals or meet the arborescent expansion of the cone fibres. The descending fibres spread out in the same way to meet the fibres of the ganglion layer. These bipolar cells probably act as trophic centres or controls for the second system of neurons. In addition to these cells there is a scanty layer of so-called *horizontal cells*, which are irregular shaped cells sending off a long horizontal fibre, which connects up with a distant rod or cone fibre rootlet.

(8) *Internal Reticular* (*Plexiform*) *Layer* is identical in structure with the external reticular layer, only much broader, and is entirely due to the arborisations of the ganglion neurons with the amakrine cells and cone bipolar cells. It shuts off the large blood-vessels from the external layers, and supports the arborisations of the second and third system of neurons. It therefore forms the bond of union between these two systems.

Greeff, who has so largely contributed to convert our knowledge of the retina from hopeless chaos into something like order, divides this layer into five strata, according to the position of the

arborisation of the amakrine cells. These pear-shaped cells form a layer immediately external to the internal reticular layer, and give off from their pointed ends a fibre. These are in five proportionate lengths, and terminate in rootlets, which spread out close to a bipolar cone rootlet. The two together join up with a large arborisation from a ganglion cell (see Fig. 32). It therefore appears to act as a relay to intensify the current passing along a cone fibre. But this is only a surmise. These amakrines have as a rule very short axis cylinder fibres, hence their name (ἀ priv; μακρός long; ἶνος, a fibre). A few of the amakrines, instead of sending processes to connect up with the ganglion cells, send a long process horizontally out, which connects up a distant amakrine, in the same way as the horizontal cells in the internal nuclear layer do.

(9) *Ganglionic Layer.*—A layer of large ganglionic cells provided with long stout processes, which unite by extensive arborisations with corresponding rootlets from the descending fibres of the bipolar fibres. They give off fibres which pass through the optic nerve to the brain. These fibres are either directly or indirectly connected with the cones, but whether any of the rods which reach the ganglion cells can transmit nerve currents to the brain is not known. By far the greater number of the rods end in the outer reticular layer and never reach the ganglion cells at all.

The ganglion cells probably act as trophic centres or controls for the long fibres which extend to the brain.

(10) *Nerve-fibre Layer.*—Their peripheral ends take origin in the ganglion cells. A special group of nerve-fibres are in direct or indirect connection with the macula cones. They enter the tempora

half of the disc, as a solid bundle, passing from the macula in sweeping curves. The central ends of the nerve-fibres communicate with the cells of the brain by arborisations. This we can infer must be the case, since every process from a nerve-cell or ganglion always terminates as an arborisation and never directly in another cell.

(11) *Internal Limitating Membrane.*—A thin membrane which lies in contact with the vitreous, or, according to some, with the hyaloid membrane, but it is an open question if the latter really exists.

Summary.

Layers.	*Nourished by—*	*System of Nerve-fibres.*
Hexagonal layer Bacillary layer Ext. limitans layer Ext. nuclear layer Ext. reticular and horizontal cell layer	Osmosis	1st neuron, or bacillary system.
Int. nuclear layer Amakrine and int. reticular layer	Capillaries	2nd neuron, or intermediate system.
Ganglion layer Nerve-fibre layer. Memb. limitans int.	Blood-vessels	3rd neuron, or central system.

Supporting Fibres of Müller. — These consist of neuroglia tissue whose extended processes fuse with the two limitating membranes (see Fig. 32, No. 11). The fibres send off tiny offshoots all along their course. In the two nuclear and ganglionic layers the process is surrounded by peculiar agglomerations of small spherical bodies. Each process contains a large oval nucleus. Borysiekiewicz says they are continuous with the bases of the cones. Frost says if this is true they are probably nerve organs of great

importance,* while Bernard† denies their existence altogether, and ascribes their appearance to streams of pigment matter which flow into the vitreous! Evidently we have much to learn about their nature and function.

* 'The Fundus Oculi,' by W. Adams Frost.
† 'Quarterly Journal of Microscopical Science.'

CHAPTER III.

THE OPHTHALMOSCOPIC APPEARANCE OF THE NORMAL FUNDUS.

Ophthalmoscopic Appearance of the Normal Fundus.—Macula Lutea.—Colour of the Macula.—Reason why the Macula is Yellow.—Difficulties connected with Seeing the Fundus.—Macula Reflex.— Foveal Reflex.—Stippled Fundus.—Distinction Between Choroidal and Retinal Vessels.—Choroide Tigrée.—Retinal Reflex.—Crick Dots.—Semi-translucent Nerve-fibres.—Semi-translucent Müller's Fibres.

THE colour of the fundus of the average fair-haired European seen with a candle, gas, or incandescent light, is either a pure vermilion, a bright orange vermilion (Pl. 1, fig. 1), or an orange-pink colour. This is chiefly due to the fusca cells and general stroma of the choroid, to some extent to the visual purple, and to a lesser degree to the blood-vessels, both choroidal and retinal. The colour is also modified by the pigment contained in the granules and crystals, and by the hexagonal pigment layer. The combined layers absorb all the violet and blue, about half the green, and the extreme red of the visible spectrum, with the result that the reflected colour is an orange vermilion. In dark-haired Europeans the fundus is an Indian red colour (Pl. 2, fig. 3), in the negro a chocolate brown (Pl. 2, fig. 4), and in the gorilla a very dark chocolate.

These modifications are largely due to the intensity of the hexagonal pigment layer. There are strong reasons for believing that it is not the direct light but the *reflected light* from the concave spherical mirror of the choroid which is focussed on the nerve terminals, and gives rise to image-forming vibrations. If it were not so, why should the sensitive ends of the rods and cones be turned away from the source of light and not towards it, as is actually the case in the invertebrate eye, where it is in a much more favourable position to receive the direct rays? Furthermore, why should all mammals, with hardly an exception, have such brilliantly coloured fundi, in which zones of emerald green, burnished gold, vermilion, scarlet, orange, yellow, brown, and even purple and blue (in small quantities) occur repeatedly? Of what use are these colours, or the brilliantly coloured tapeta lucida seen throughout entire families in the mammals, if the light were to be focussed direct on to the retina, seeing that the coloured layers are behind the nerve terminals? These colours would be of no advantage to the animal as regards its acuity of vision or colour perception, nor would the colours be a protection, inasmuch as they can only be seen by another animal in certain positions and under the most favourable circumstances.

It may be objected that the fundus of albinos, both in man and other animals, presents considerable areas of pure brilliant white, and has relatively few fusca and hexagonal pigment cells (Pl. 1, fig. 2). But white reflects all colours equally, and is therefore able to some extent to fulfil the required conditions. Still, all albinos see imperfectly, and they all suffer more or less from photophobia and nystagmus. Were a dead black fundus ever to be found the objection might hold good, but a black fundus is an

unknown quantity in nature. In all eyes, except perhaps the darkest races, the sclerotic reflects some amount of light, and the fusca cells and visual purple a great deal.*

The eye has often been compared to a photographic camera, but the analogy between the Lippmann method of photographing colours by interference and the eye is still more striking. Lippmann obtained negatives in natural colours by placing a reflecting mirror or mercury trough behind and in direct contact with the sensitive film of the plate, thus reflecting the light which had passed through the translucent film on to the particles of silver haloid, the colour effect being obtained by interference. In the eye the light passes through the transparent retina, which may be compared to the sensitive photographic film, and is reflected back by the choroid stroma (or tapetum lucidum) on to the sensitive nerve terminals of the rods and cones, which are analogous to the particles of silver bromide or chloride. It might well be asked, Do we not owe our colour sense to interference also?

As a matter of fact, however, we have no positive evidence of any kind in favour of this theory, but at the same time no purely physical theory has been brought forward which will meet all objections. The insurmountable difficulty which stands in the way of all theories of colour, based on chemical or physical changes either in the visual purple or the nerve terminals, lies in the rapid alterations of colour which after-images undergo when the eyes are closed, and secondly in the remarkable fact that in certain diseases, notably cerebral tumours, tabes and hysteria, the colours, especially towards the periphery, become inverted—that is to say, the complementary colours

* See the writer's paper in the 'Trans. Roy. Soc.,' 1901.

are seen. For example, red appears green, blue yellow, and violet orange, etc. There are many facts in favour of the theory that the sensation of every colour lies in the brain, and is in no way connected with the retinal elements. In fact, all the phenomena of colour-blindness point to a cerebral and not to a retinal origin. In a recently recorded case, in which the patient was affected with ascending multiple sclerosis of the cord on one side, all sense of colour gradually vanished from the eye of that side, although the visual acuity in both eyes remained normal, or nearly so.* Again, the field for *blue* becomes very contracted, and the colours are sometimes reversed in brain tumours, while in tabes and multiple sclerosis it is the field for *red* which suffers soonest and is most constricted. In hysteria not only are the colours reversed but one or the other of them may temporarily vanish, while the field for all colours is contracted one hour and may return to normal the next. These facts are all in harmony with a psychic disturbance but not with a retinal one.

The Macula Lutea and Fovea.—This, next to the papilla, is the most important part of the fundus, and should invariably be examined when the acuity is found to be subnormal.

The fovea should be sought for by the direct method on the outer side of the disc in a horizontal line with its lower border at a point from 2 to $2\frac{1}{4}$ disc breadths from the outer margin of the disc. With the indirect method of course the fovea will be situated on the apparent inner side of the disc in a line with its upper border. If there is any difficulty in seeing it owing to the reflex or smallness of the

* For a coloured illustration of this case see 'Colour Photography,' published by Ward & Son, 1910.

pupil, cause the patient to look outwards. This will bring the disc to the inner part of the field, and the macula can then readily be seen without any disturbing reflex.

Colour of the Macula.—In most emmetropic adults there is very little difference between the colour of the macula and the rest of the fundus, but inside the macula ring it is generally of a somewhat more intense or brighter red than the rest of the fundus. This colour deepens towards the fovea centralis, where it is usually masked either by the bright foveal reflex, or by a dark shadow due to the sides of the pit being unusually deep and narrow. Care must be taken to ignore the dark circular shadow due to the central opening of the mirror, which beginners often mistake for a pigment patch. It can be at once recognised by its shifting as the mirror is moved. For colour of the macula see Pls. 1 and 2.

Minute yellowish dots, probably due to fatty or cholesterin deposits, are very frequently observed in the macula area around the foveal pit. In many cases it is uncertain whether they ought to be considered pathological or not. They are certainly met with in perfectly healthy eyes with normal acuity. They are very few in number, and must never be mistaken for the pathological deposits in central choroiditis or tuberculosis, which are very numerous and seriously interfere with vision.

The yellow colour of the macula region seen in enucleated eyes which have been cut open, is for the most part a post-mortem change, and is not seen in a freshly opened eye until the retina is lifted off from the hexagonal layer.

Reason why the Macula is Yellow.—As the coloured area is very small, the yellow colour cannot

be seen by the ophthalmoscope, but that it is yellow can be readily shown by Clerk Maxwell's experiment. If you look at the sky through a solution of chrome alum a purple spot will be seen in the line of vision in the green solution. This is due to the yellow pigment which absorbs the green rays. I believe the reason for the pigment is as follows: When a photograph is taken either on an iso-chromatic plate or on a Lumière autochrome plate the photographer places a yellow filter-screen in the path of the rays to keep back by absorption the intensely active blue-violet and ultra-violet rays, and so allow the proper values to be assigned to each colour. Precisely the same thing happens in the eye. A yellow screen lies in front of the bacillary layer, which consists of the layers of red corpuscles in the capillary vessels. But, as is pointed out elsewhere, there are no capillaries in front of the cone layer at the fovea centralis. A layer of yellow colouring matter is therefore placed by Nature immediately behind the tips of the cones, which not only does duty for the yellow screen elsewhere, but the colour is more intense and better selected, and therefore the eye is less sensitive to blue, but better adapted to other colours at that spot. Since the rays from the object seen pass through the retina to the choroid and are reflected back again on to the rods and cone terminals the light only passes once through the capillary filter, but twice through the yellow pigment. The effect of the latter is therefore doubled.

What the colour of the macula appears to be to the observer depends greatly on the character of the light by which is is examined. In cases of plugging of the central retinal artery, the fovea stands out as a cherry-red spot on a cream-coloured ground (Fig. 45, and Pl. 21, fig. 41) both in the enucleated eye

and in the living eye as seen with the ophthalmoscope. Again, in atrophy of the nerve the macula and the fundus generally show a vivid red in striking contrast to the white disc (Pl. 11, figs. 21 and 22).

Difficulty in Seeing the Fundus.—Sometimes the details of the fundus cannot be seen at all when the cornea and lens are clear. Of course this may be due to a very small pupil. But if the pupil is fairly dilated, and no details can be seen either by the direct or indirect methods, focus up by rotating one convex glass after another in the ophthalmoscope so as to bring the anterior and posterior capsules of the lens into focus. This can also be done by the condensing lens and oblique light. If these are clear the trouble will lie in the vitreous, unless the retina is detached. By very careful focussing with a suitable convex glass in the ophthalmoscope, either a fine membrane like wet tissue paper may be seen stretching across the vitreous, or anchored at one end and floating, or a gauze-like film may be observed on or immediately in front of the retina. These are the sequelæ of old hæmorrhages, or exudates from the ciliary region as the result of cyclitis. They sometimes disperse completely.

In examining the fundus of an aphakic eye by the direct method, an emmetropic observer will require to rotate up a lens between + 7 D and + 11 D, according to the distance at which he holds his ophthalmoscope from the patient's eye. Often the fundus is seen best by the indirect method.

The Macula Reflex.—The macula is bounded by a circular, or in some cases a horizontally oval ring known as the macula ring. This may be seen in most people under thirty-five years of age (and occasionally over that age), especially if the source of illumination be gradually lowered (Pl. 2, fig. 4). Now the

reflection from the fundus decreases more rapidly than from the margin of the macula, so that a time is arrived at when more light is reflected from the latter than from the general fundus, and at that moment the ring appears. The ring is due to the reflection of light from the cup-shaped rim of the macula, which after adult life gradually flattens down to the general level of the fundus, and then the reflex ring disappears.

The ring is seen in hypermetropic children to perfection, especially in dark-haired children, and in all coloured races (see Pl. 2, fig. 4). In myopia owing to the general stretching of the coats of the eye, the ring is rarely seen.

There are three distinct types of rings:

(1) A bright scintillating reflex resembling shot silk, similar in appearance to that noticed skirting the blood-vessels and over the greater part of the fundus in darkly pigmented eyes. This is extremely common in dark-haired people (Pl. 2, fig. 4).

(2) A radiating ring of a greyish-white lustre round the macula. The radii are all directed towards the fovea, and although losing their outline at the edge of the macula, they may be traced for a variable distance (usually about half the papillary diameter) away from the macula. They are due to the translucent nerve-fibres which radiate from the foveal pit.

(3) The third form is so conspicuous that it may be seen even with the brightest illumination as a well-marked whitish or golden ring of metallic lustre, usually oval in shape when seen with the inverted image, and round, or nearly so, when seen in the direct method. In the marmoset's eye the ring consists of red, yellow and green, or yellow and green colours.

The importance of seeing the ring is due to the fact that it forms the border of the area of distinct vision, and any lesion inside this ring must be followed by a corresponding falling off of acuity over that area and often beyond it.

The Foveal Reflex.—At the centre of the macula reflex ring a very small ring is frequently seen at the edge of the dip of the fovea centralis, which is due to a reflex from the edge of the fovea. In consequence it assumes a number of shapes. It is generally very bright, like gold or else luminous white, being sometimes in the form of a minute circle, or with a gap like a letter "C." The central reflex varies greatly, from a mere point of light (Pl. 2, fig. 4) to a long flare like a comet, with the head at the fovea, and the tail at the circumference of the macula. The shape of these flare-spots varies with the nature of the foveal dip. We notice the same variation of reflexes on the glistening tympanic membrane around and below the handle of the malleus.

The foveal reflex, like the macula ring, becomes less visible with age, and ultimately disappears, but it may frequently be seen as a tiny bright speck in the centre of the darker red background after all traces of the macula ring have vanished.

Stippled Fundus.—Occasionally the retinal pigment is visible, giving the appearance of a fine, black stippling (Pl. 2, fig. 3). Such eyes cannot be considered as normal, since in the majority of cases the vision is defective, and cannot be brought up to standard acuity by means of glasses. Wherever the hexagonal pigment is thin and scattered the choroidal vessels come into view. This is nearly always the case towards the periphery, even in dark pigmented eyes. Choroidal vessels may be distinguished from the retinal ones as follows:

Choroidal Vessels.	*Retinal Vessels.*
Colour, orange or orange-pink.	Colour, veins crimson lake. Arteries scarlet.
Veins and arteries indistinguishable.	
Vessels are curvilinear and anastomose freely, forming networks.	Run a more or less straight course—never anastomose.
Are broad and disappear in places. Cannot be traced for any distance as a rule.	Can be traced throughout their entire course.
Vessels are flat and have no light streak.	Vessels cylindrical and have a light streak.
Vessels never pulsate.	Vessels at the disc often pulsate.

Choroide tigrée.—In some eyes, otherwise quite normal, the choroid is deeply pigmented, while the hexagonal pigment is sufficiently thin to allow the choroidal vessels to shine through so that they appear in relief on a dark background. This is known as a tesselated fundus or *choroide tigrée*, and is apt to be mistaken for choroiditis. It exists as the normal condition in the Gibbon's eye and in that of many of the lower monkeys. It is quite common towards the periphery of the fundus. All sorts of local variations of this type of fundus are to be met with, according to the degree of colour or pigmentation of one or other of the layers over different areas. Small localised circular or oval areas of black pigmentation are occasionally met with which have a sharp line of demarcation. Frost happily compares them to pigment moles in the skin. They have no pathological significance.

Retinal Reflex.—This is a kind of silvery shimmer which flashes along the retinal vessels (Pl. 2, fig. 4). The fundus sometimes glistens like "shot" silk or "watered" silk all around the macula ring, stopping there, and thus demarcates the ring very clearly. In some eyes a series of reflexes in the form of concen-

tric rings round the disc which spread towards the periphery may be noticed. They are not unlike the ripples in a pond when a stone is thrown in. These reflexes are most abundant in dark, highly pigmented, and hypermetropic eyes, especially in children and black races. They are consistent with the highest acuity of vision. In older people, myopes, and light-haired subjects, the reflexes are much rarer.

Gunn's Crick Dots.—These dots are highly reflecting yellowish-white globules, probably of a fatty or cholesterine nature, which occur inside or round the macular ring. Occasionally one or two may be seen in the neighbourhood of the retinal vessels, in front of which they lie. They are often congenital and glisten brilliantly. They do not affect vision. The name was first given by Marcus Gunn, who observed them in all the members of a family of that name.* I have frequently seen them, but the light must fall on them obliquely to be recognised.

Semi-translucent Nerve-Fibres.—These, when merely translucent and not opaque, are not pathological, and in no way interfere with a high degree of visual acuity—in fact, they form the normal condition in the highly pigmented negro eye (Pl. 2, fig. 4). They are sometimes seen in Europeans. They occur in the form of innumerable fine whitish, semi-translucent, straight lines of equal lengths, which start from the edge of the papilla, and become fainter as they are traced outwards, finally vanishing a few disc breadths from the papilla. They are very common in the lower animals, occurring in many of

* See 'Trans. Ophth. Soc.,' vol. iii, p. 110, and Adam Frost's 'Atlas,' Pl. iv, figs. 9 and 10, Pl. xi, fig. 30. Occasionally the entire fundus appears to be covered with these dots, which last apparently for a lifetime and undergo no change. They do not interfere much with vision. For an illustration of these see Frost's 'Atlas,' Pl. xi, fig. 31. Compare this with retinitis punctata albescens, p. 156.

the Rodents, Marsupials, and Monotremes, and almost universally throughout the reptiles and fishes. Translucent nerve-fibres is a congenital reversion of type which never affects vision, and must not be confounded with opaque nerve-fibres, which often seriously lowers acuity and causes scotomata, although they exist normally in many of the lower animals, among which the rabbit is the most familiar example.

Semi-translucent Müller's Fibres. — The sustentacular tissue of the retina forms the supporting fibres which protect and keep the retinal elements in position. It is almost invariably transparent, but cases have occurred in which the connective tissue of the two limitating membranes with which the terminals of the fibres are intimately combined were slightly opaque, and could be seen with the ophthalmoscope in the form of innumerable fine whitish lines, which stretched across the entire fundus in two parallel sets, the one running vertically, the other horizontally across the field like the web and woof of a tissue. It is a rare phenomenon, but is exceedingly striking when seen (see Frontispiece, drawn from a patient of the author's).

Congenital Pigment Dots.—A peculiar form of pigmentation is now and then met with in which a small section of the retina contains a large number of black pigment bodies arranged in groups of four, eight, or more close together but isolated from other groups. They somewhat resemble groups of gonococci. They lie sometimes in front of, but more often behind the retinal vessels. The eyes are otherwise quite healthy and the vision normal. Jaeger, Dodd, and Stephenson have depicted and described cases. See Frost's 'Atlas,' Pl. x, figs. 28 and 29, also Jaeger's 'Atlas.'

Opaque Nerve-fibres.—See end of Chapter IV.

CHAPTER IV.

THE OPTIC NERVE—ITS CONGENITAL PECULIARITIES AND VESTIGIAL RELICS.

Optic Nerve.—Physiological Cup.—Retinal Vessels.—Blood Supply of the Macula.—Congenital Anomalies.—Persistent Hyaloid Artery.—Absence of all Retinal Vessels.—Pecten.—Coloboma.—Development of the Eye.—Coloboma of the Disc.—Coloboma of the Choroid.—Hole in the Disc.—Opaque Nerve-fibres.

The Optic Nerve as it enters the eye is protected by two sheaths—an outer sheath continuous with the dura mater, and an inner sheath, a prolongation of the pia mater. Between the two is a lymph space, the sub-vaginal space, which is not free, but is loosely filled by a thin cellular network derived from the arachnoid, and the space between the latter and the pial sheath, which is very narrow, opens directly into the sub-arachnoid space of the brain. Both spaces terminate in a blind extremity around the disc, although a minute lymph space is continued on from them to join the perichoroidal space between the sclerotic and choroid, and this latter space communicates with the large lymph space between the layers of Tenon's capsule by means of the lymph channels surrounding the venæ vorticosæ.

There are also minute spaces running through the nerve trunk, which communicates with the

large sub-vaginal space and with the narrow spaces which run round the coats of the eyeball and drain the vitreous, choroid, and retina. Lastly, there is an important lymph space called the supravaginal space, which lies immediately around the outer sheath of the optic nerve, and is continued on into Tenon's space in front, and behind into the great subdural space of the brain. This explains how meningitis may follow a squint operation or an enucleation in which the wound has become infected.

The trunk of the optic nerve consists of a large number of nerve bundles, separated from one another by fibrous septa and connective tissue, but communicating by minute nerve-fibrils. The individual nerve-fibres are separated and supported by a delicate neuroglia tissue. The central artery (a branch of the ophthalmic), and its vein, enter the optic nerve about half or three quarters of an inch behind the globe, and pass up the centre of the nerve as far as the disc, where they each divide into their main branches. The vein either joins the superior ophthalmic vein or enters the cavernous sinus directly.

The fibres of the outer two thirds of the sclera are bent aside at a right angle, where they reach the nerve, thus forming its sheaths. The nerve, therefore, only penetrates the inner third of the sclerotic, through which the bundles of nerve tissue thread their way. This is known as the cribriform fascia, and can often be recognised in the centre of the disc with the ophthalmoscope as a series of pale grey spots or pits on a white background. The constriction of the optic nerve at this spot is due to the fact that the fibres lose their medullary sheaths where they pierce the cribriform fascia, and so take up less room. Occasionally the fibres carry their

medullary sheaths with them into the retina, and are visible with the ophthalmoscope as white woolly streaks (opaque nerve-fibres) (Pl. 4, figs. 7 and 8). At the lamina cribrosa the nerve is only supported by about a third of the sclerotic, and is thus a weak spot and tends to yield and be pushed back with an increase of tension (glaucomatous cupping) (Pl. 12, figs. 23 and 24). Moreover, any pressure on the head of the nerve tends to push the bundles against the cribriform fascia, which, being composed of unyielding fibrous tissues, squeezes the nerve, thus producing atrophy, just as a hernia becomes gangrenous by prolonged pressure against the rigid edge of Gimbernat's ligament.

The disc (papilla) is the most prominent object in the fundus (Fig. 36). It is usually either circular or slightly oval in shape, the longer axis being vertical. It varies from 1·25 mm. to 1·75 mm. in its horizontal diameter. In most cases three zones can be made out: (1) A central whitish pink, or pearly white zone of variable size, which is often depressed to a lower level than the rest of the disc. In a number of cases the pearly grey pittings of the lamina cribrosa are distinctly seen. More often the central zone is hidden behind the trunks and bifurcations of the retinal artery and vein. (2) An intermediate zone which extends from the inner zone to near or quite the edge of the disc. The two zones usually shade off insensibly, so that as a rule no sharp line of demarcation is noticed. (3) An outer zone which is often wanting. Sometimes it forms a narrow white ring, completely encircling the disc and extending as far as its edge. At other times it is seen on one side only, or it may be absent at the upper and lower parts of the disc. The more usual form is for the inner half of the disc, *i. e.* the whole space between

the vessels and its inner margin, to be of a rose red colour, while the rest of the disc on the macula side is of a buff or white colour, as if it were atrophied. Again, the whole disc may be chalky white, excepting a pinkish red crescent which encircles the

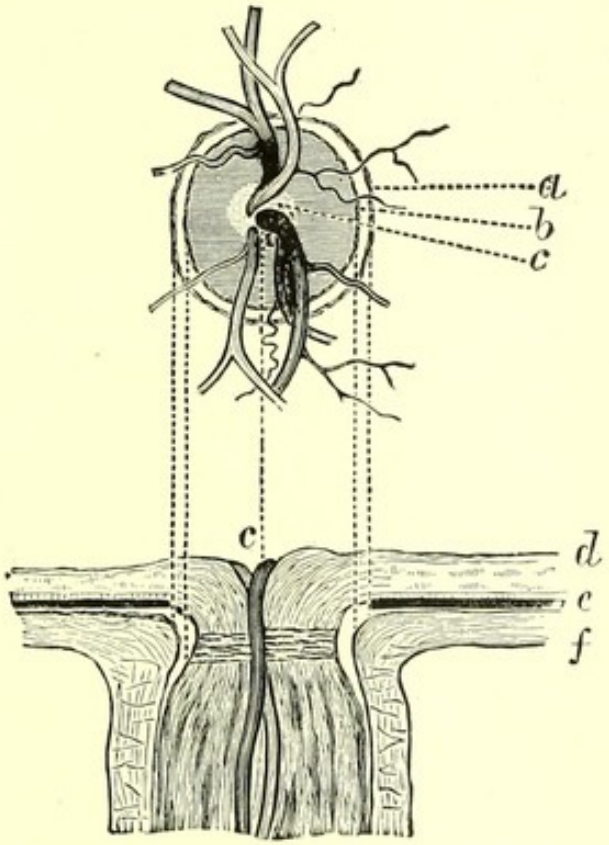

FIG. 36.—Normal disc showing the white ring due to absence of choroid at that spot, white sclerotic showing through.

a. Scleral ring.
b. Edge of disc.
c. Central depression and point of emergence of vessels.
d. Retina.
e. Choroid.
f. Sclerotic.

disc round its outer and a little of its upper and lower parts only. The beginner is apt to mistake this appearance for an atrophied disc; but in optic atrophy this pink zone is invariably absent. Along the outer margin of the third zone, just outside the true disc, a variable amount of black pigment is commonly present. Sometimes the choroid

does not extend as far as the dural sheath of the nerve, leaving a white crescent. This crescent becomes very exaggerated in high myopia.

Physiological Cup.—This forms a variety of the normal form of disc (Pl. 10, fig. 20), in which the rose-coloured *z*one terminates abruptly in a crescentic edge, usually along the inner side, the central part of

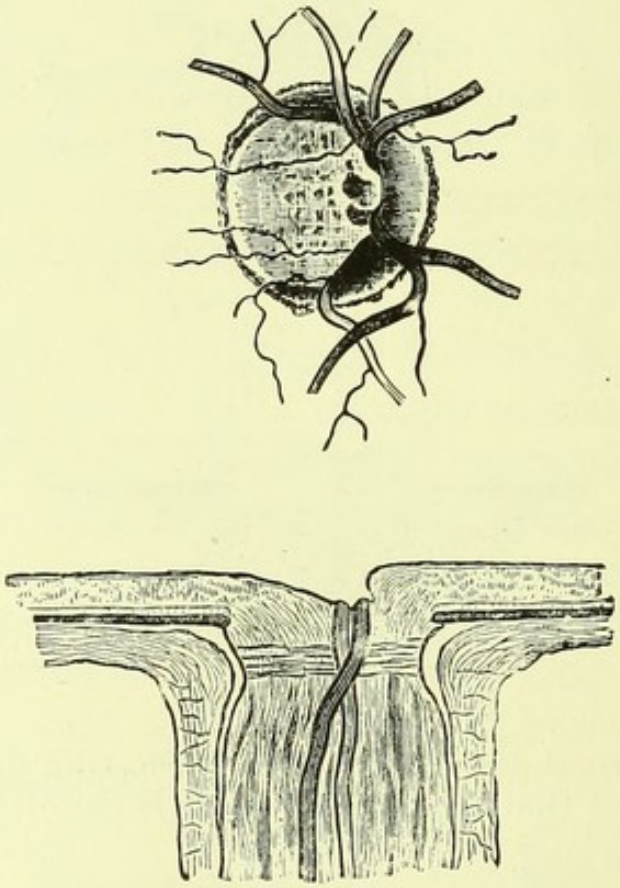

FIG. 37.—Physiological cup. The depression is confined to one side and does not extend to the margin of the disc.

the disc being chalky white, situated at a much lower level, and showing the lamina cribrosa mottling. The floor of the disc may be equally sunk all round, but usually it slopes up nearly to the level of the middle *z*one on the temporal side. In consequence of this sudden depression of the floor of the disc the vessels curl over the side, thus simulating a glaucomatous cup (Fig. 44); but it may be distinguished, first, by the fact that the vessels run a straight course

without curling over the periphery of the disc; second, by the presence of the rose-coloured crescent or zone, which is white or buff coloured in the diseased condition. Occasionally the pit does not really exist, the disc being level and covered over with perfectly transparent tissue as obtains in all the Felidæ (lions, cats, etc.). The pink colour is due to minute capillaries, which ramify between the nerve bundles.

The Retinal Vessels.—The retinal arteries are dis-

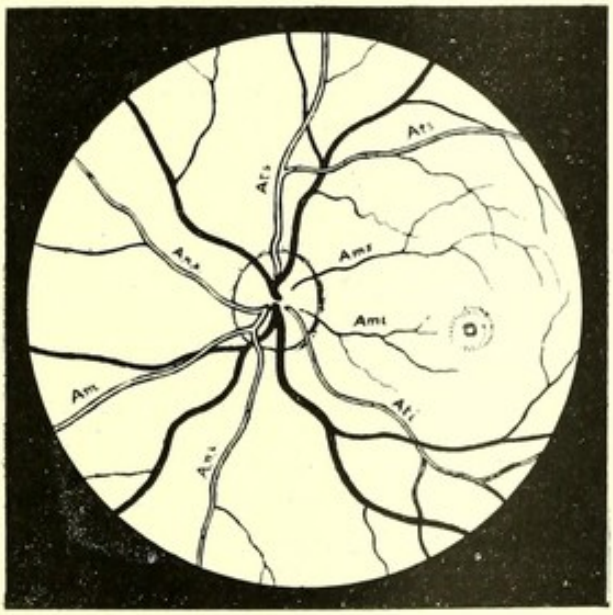

FIG. 38.—Vessels supplying the retina.

Ans. Arteria nasalis superior.
Ani. „ „ inferior.
Ats. „ temporalis superior.
Ati. Arteria temporalis inferior.
Ams. „ media superior.
Ami. „ „ inferior.

tinguished from the veins by being scarlet and much brighter than the veins, which are crimson lake in colour; they also have a calibre less by one third than that of the veins and possess a much more conspicuous central streak. The central artery and vein usually pierce the disc close together and near its centre. Each then divides into two short trunks, which immediately form two main branches, which ramify over the temporal and nasal halves of the

retina. Those on the inner side are termed the arteria nasalis, superior and inferior, and those on the outer side the arteria temporalis superior and inferior (Fig. 38). The two temporal branches each give off a large branch, which bends round and gives off fine twigs to supply the macula region. The position of the macula, when invisible, can readily be determined by fine branches, which are directed towards it from all sides, although none of them actually reach the macula itself. The variations in distribution and appearance on the disc of the retinal vessels are endless. Occasionally, instead of emerging as a single trunk, the arteries divide into several branches within the nerve, behind the lamina cribrosa, or the arteries and veins may be derived from the choroidal vessels. In either case they emerge from several places round the margin of the disc, bending over the edge in the form of large loops. This is the normal method of distribution in many of the lower animals, such as the Seals, Bears, Skunks, Flying Squirrels, etc. Now and then a cilio-retinal branch takes the place of one of the temporal branches of the central artery, and instead of passing along the retina, it bends over at the margin of the disc and disappears finally. Speaking generally the retinal arteries never anastomose, nor do the veins, except in rare cases, and then usually near the periphery.* When, therefore, a vein or an artery is plugged by a thrombus or an embolus, the entire area supplied by the vessel immediately becomes insensitive to light, nor does

* Three or four short ciliary vessels supply the optic nerve and its sheaths, and send minute branches which anastomose with the central artery at the disc or just inside the nerve. Occasionally a ciliary vessel makes its way from the optic nerve to the macula and apparently supplies the area between. This vessel sometimes anastomoses with a branch of the retinal artery near the macula.

it as a rule recover its function. Embolus, or thrombus, of the central artery or vein leads at once to total blindness in the eye affected.

Blood Supply of the Macula.—The blood supply of the macula region is far more abundant than at any other part of the retina, which is to be expected seeing the enormous amount of physiological work it has to do. Only at the actual centre of the fovea are there no retinal capillaries, an area equal to a tiny needle prick or about 0·15 square mm., *i.e.* a circle a quarter of a millimetre ($\frac{1}{100}$ inch) in diameter, but to make up for this the choroidal capillary network is very close meshed at this part.

Pulsation can very frequently be observed in the main trunks of the veins over the disc even in healthy eyes, but pulsation in an artery is always pathological, and is the result either of increased tension in the eyeball, or of valvular or kidney disease, or general anæmia. It is invariably seen during an attack of glaucoma, in the arteries where they cross the disc. The blood-corpuscles in the vessels in man are too small to be visible with the ophthalmoscope, but in the Amphibia, which all possess very large blood-corpuscles, their movements can be distinctly seen, and even the individual corpuscles noted. This is especially observable in the eyes of large frogs and toads. In these animals the vessels do not run in the retina, but close to it in the vitreous.

Light Streak.—A brilliant streak may be observed running along the centre of the vessels. It is best seen in the arteries. Loring says the streak is due to reflection from the blood column. It is only seen in the larger vessels.

Congenital Abnormalities and Vestigial Relics.—1) *Persistent Hyaloid Artery:* The retina is nourished

from two sources; the outer layers (viz. the bacillary and external nuclear layers) are supplied from the chorio-capillaris by direct osmosis through the lamina vitrea (membrane of Bruch); the remaining layers are supplied partly by capillaries from and partly by the branches of the arteria centralis of the retina. A large number of mammals (notably the Perissodactyla, Bats, Edentata, and many Rodents), all birds and most reptiles are entirely destitute of retinal vessels, while others, again (Marsupials and certain Rodents), possess a central meshwork of vessels, together with numerous minute independent vessels which emerge from the periphery of the disc. The central meshwork of vessels forms the vestigial relic of the pecten, a vascular organ covered with dense black pigment cells which emerges from the centre of the disc in all birds and many of the reptiles. In the Beaver it occurs inside a hollow cone—the remnant of Cloquet's canal. This serves as the support or core of the central hyaloid artery, which is the homologue of the falciform process in the fishes. Both the falciform process and the hyaloid artery, which together constitute the vessel with its surrounding sheath, form a capillary network at their termination at the lens which it serves to nourish in the fœtus. Now the hyaloid artery disappears in most mammals during fœtal life, but in some mammals this falciform sheath persists through life in the shape of a dense white fibrous cord stretching from the centre of the disc to the lens capsule. This is the case in many Ungulates and Rodents (Hog-deer, Alactaya, Mouse). It may either arise from a fibrous plate attached to the disc, or may be seen springing from the connective tissue around the main artery (arteria centralis retinæ), where it emerges from the disc. It is, therefore, in

reality the remains of the sheath which surround the hyaloid artery which was pushed forward during its development, and has eventually nothing to do with the hyaloid artery, which ceases to exist. *Occasionally it persists in man, and is then wrongly termed the persistent hyaloid artery* (Pl. 3, figs. 5 and 6). This latter, the true hyaloid artery, has quite a different appearance, forming a short club-shaped offshoot of the main vessel, which ends blindly in the vitreous, close to the disc (Pl. 5, fig. 9, and Pl. 8, fig. 15). It sometimes contains blood and even pulsates. I have observed the same thing normally in the Beaver. This hyaloid artery is never accompanied by a returning vein and is of no clinical importance, but is interesting from its intimate homology with the pecten.

(2) *Absence of Retinal Vessels.*—We have remarked above that retinal vessels are entirely absent in all birds, many reptiles, and some of the mammals. This condition has on very rare occasions been met with in man. Professor Berry informs me that he has met with such a case, and he says in his 'Ophthalmic Diagnosis,' p. 22, "complete absence of the retinal vessels has been met with as a congenital defect." Such cases undoubtedly point to a reversion of type to one of the above-named lower orders.

(3) *Pecten Remains.*—In reptiles and birds the hyaloid artery is superseded by a new development—the pectinal system. In some of the lower mammalia the two systems actually occur side by side, but both are rendered unnecessary in the higher mammalia by the development of a third system of supply—viz. the special retinal vessels which culminate in the arteria and vena centralis retinæ. The pecten may be observed in all stages of developmental recession among the mammals and certain reptiles—notably the Crocodilia—and

may occasionally be seen in an extremely degenerate form in man. This is the explanation of the shrunken fibrous deposits occasionally met with on the floor of the disc, and probably the small basket-like convolution of vessels occasionally seen springing from the main trunk at its emergence from the disc is of the same nature. The rare congenital pigmentation of the disc, which occurs as a more or less complete circle inside the margin, may also be

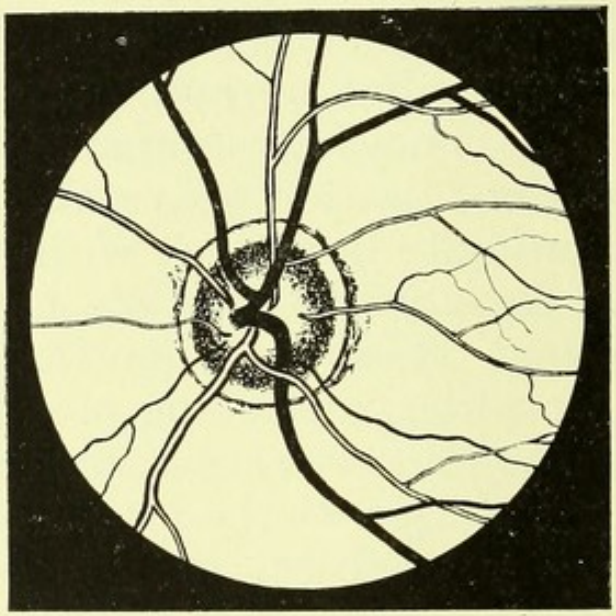

FIG. 39.—Pigmentation of the disc. Probably a vestigial degeneration of the pecten.

a pecten relic (see Fig. 39). It has occasionally been observed after a traumatic hæmorrhage as well.*

Coloboma.—A gap in, or absence of continuity of, any part of the choroid, retina, or iris is termed a coloboma. The cause of this defect may be explained as follows: the head-end of the medullary tube in the fœtus is due to the folding over of the sides of the primitive medullary groove which becomes dilated into three vesicles. The terminal one (first cerebral vesicle) expands laterally, forming two blind

* See Lechner's paper 'Nederlandsch Tydschrift voor Geneeskunde,' 1909, p. 797.

pouches, one on each side (optic vesicles). The end of this bladder-like expansion becomes pushed in, thus doubling upon itself and forming a cup when viewed from the outside. This is called the secondary optic vesicle, which becomes the retina proper, while the posterior cup forms the hexagonal epithelium layer. The tube behind this cup now contracts its ventral portion, forming the optic stalk, which ultimately forms the optic nerve. The secondary optic vesicle, or optic cup, gradually becomes moulded into the form of the eyeball, and its mouth is covered over with a layer of ectoderm, which thickens in the middle to form the rudimentary lens. If this primitive eye be now examined the mouth of the secondary optic cup will be seen to be open below with the spherical lens filling the gap at its upper part. This gap, which is merely due to a doubling in of the outer wall of the vesicle, and not to any want of continuity of tissue, extends not only along the whole ventral part of the eye, but also along the underside of the optic stalk. Both the optic stalk and vesicles are all formed from the ectoderm layer, but the vitreous and blood-vessels are formed from the mesoderm, and the object of the later closure of this gap is to allow the mesoderm cells to pass into the cup to form the vitreous and choroid, while the groove and the optic stalk is left open for the mesoderm cells to gain admission to form the central artery and vein of the optic nerve (Pl. 25, fig. 49).

Now, if any part of this cleft (choroidal fissure) does not quite close, either along the lower surface of the fundus or round the optic nerve at the disc, a breach of continuity in the tissues will be found, which in the latter case forms a coloboma of the disc, in the former a coloboma of the choroid or iris.

(4) *Coloboma of the Disc.*—This shows itself as a large white patch round the disc, which is much depressed. The form of the disc is more or less completely lost in the surrounding whiteness. The retinal vessels often curl over the edge of the crater. There is usually a considerable deposit of pigment in places. These changes are quite compatible with normal vision (Pl. 5, fig. 10; Pl. 6, figs. 11 and 12; also Pl. 25, fig. 49).

(5) *Coloboma of the Choroid.*—This may occur in any part of the field, but the place of election is at or near the macula, or along an area extending obliquely downwards and outwards (Pl. 7, figs. 13 and 14). A disc coloboma is often seen as well. The rest of the fundus is usually healthy. These colobomata are by no means so rare as is usually supposed. They all present certain features in common—viz. an oval, round, or irregular-shaped patch, which may be of almost any size. The margin of the patch is sharply defined, and usually loaded with pigment, which often extends some distance beyond the edge. The floor is usually glistening white or pale pink. Choroidal vessels form a network over the floor, which is often obscured in places by dense black pigment. This pigment is always in front of the retinal vessels and never behind. I stated in my original paper* that the lesion undergoes no change. I now find that considerable changes, both in form and size, do occur, but very gradually. Some of these extra-papillary types are either purely pathological from the first, or else form the focus of tubercular, and syphilitic and other changes, but some are undoubtedly due to arrest in the development of the parts through non-closure of the choroidal cleft, or

* 'Knapp's Archives,' vol. xix, p. 12. Thirteen drawings in colour are shown.

are simply nævi or retino-choroidal moles. In those cases in which the coloboma encroaches on, but does not actually involve, the macula, the central vision may be normal (Pls. 7 and 8, figs. 13 to 16).

(6) *Crater-like Hole of the Disc.*—This is a congenital anomaly very occasionally met with in an otherwise healthy eye. It generally takes the form of a punched-out hole in the disc, usually a little to the outer side of the points of exit of the main vessels. The hole varies greatly in depth and size in different cases; it may be equal to a fourth or a fifth of the diameter of the disc. It is generally deep and dark from the shadow cast. Sometimes a small ciliary vessel may be seen at or near the bottom of the pit. It is unconnected with the closure of the fœtal cleft, since it is unaccompanied by any signs of either coloboma or other indications of the fœtal vesicular fold. Moreover, the hole occurs normally in the Elephant, two drawings of which will be found in the writer's monograph ('Trans. Roy. Soc.,' 1901). Stephenson has a characteristic drawing of it in the 'Ophthalmoscope,' February, 1909, where references to many other cases are given.

(7) *Choroide tigrée* (see p. 99) must also be considered as a vestigial relic.

(8) *Opaque Nerve-fibres.*—We have pointed out that when the optic nerve reaches the lamina cribrosa the fibres lose their medullary sheaths. Sometimes they retain their sheaths, or else resume them on emerging from the lamina, and may then be seen by the ophthalmoscope as flame-shaped white tufts, which usually follow the vessels for a short distance, directly upwards or downwards (see Pl. 4, fig. 7). Occasionally the patches are very thick and opaque, completely burying the vessels in places, or the disc and a large area round it may be almost completely hidden

under the sheaths which become apparently fused into a solid white mass (Pl. 4, fig. 8). This might be mistaken for an inflammatory exudation, but it can be distinguished by the brush-like character towards the margins of the patch. When the area is extensive, the vision is usually very defective, or even *nil.*

Opaque nerve-fibres occur normally in several of the mammals' eyes, *e.g.* the Hare, Rabbit, among the rodents, and *Perameles lagotis* among the marsupials, also in the eyes of many fishes.

(9) *Semi-translucent Nerve-fibres* as a normal condition are extremely common in the lower animals (see end of Chapter III).

(10) Lastly, the central artery occasionally divides behind the lamima into numerous branches which emerge round the disc. This occurs in many of the mammals (see under "Retinal Vessels," p. 107).

Amblyopia (ἀμβλύς blunt, ὤψ the eye) is a condition of the eye in which vision is defective without any apparent lesion. By far the commonest form is amblyopia ex anopsia, in which the sight is defective entirely through want of use. This may be due either to old corneal opacities, to a squint acquired in childhood, or unilateral astigmatism. Such cases are partially blind, not from any defect in the retina or optic nerve, but to an arrest of the psychic functions of vision. With the ophthalmoscope the eye appears perfectly healthy and normal. Hysteria is also responsible for many cases. In addition to these psychological forms there are many other purely pathological varieties due to cerebral and spinal changes, and the effect of certain drugs, such as tobacco, alcohol, lead, etc. Day- and night-blindness also come under this head.

CHAPTER V.

GLAUCOMA.

Glaucoma.—General Sketch of the Disease.—Theories of Glaucoma.—The Writer's Theory.—Later Pathological Changes.—Secondary Glaucoma.

Glaucoma.—This insidious disease, which always tends to atrophy of the optic nerve, has essentially a mechanical cause, viz. increase of pressure from within. For the purposes of classification, we divide it into—(1) Primary glaucoma, in which it commences in an apparently healthy eye, and sooner or later affects the second one, and (2) Secondary glaucoma, which follows as a consequence of an existing disease, such as ectasia of the cornea or sclera, an incarcerated iris, seclusio or occlusio pupillæ, a swollen lens, an intra-ocular tumour, hæmorrhage, or as the result of changes set up after or during choroiditis. Secondary glaucoma is of local origin, and consequently only affects the diseased eye. We may also divide it into acute and chronic glaucoma according to whether the disease sets in with well-marked prodromal symptoms, followed, sooner or later, by an acute outburst, or begins gradually with very little tension, and absence of all acute inflammatory symptoms.

General Sketch of the Disease.

Primary inflammatory glaucoma may be divided into three stages:

(1) *Prodromal Stage.*—The patient suddenly notices that he does not see well with one or both eyes: everything appears clouded over. On looking at a light it appears surrounded by a halo, consisting of blue and yellow-buff rings (the latter outside). The bowels are usually constipated, and there is often frontal headache. The near point of vision recedes (premature presbyopia). On examination with a lens the cornea appears dull, as if breathed upon. The anterior chamber is probably shallow, and the iris will be found pushed forward. At the same time the pupil is dilated, and reacts to light with difficulty. There is increased tension and some ciliary injection. Arterial pulsation where the main branches cross the disc will be seen. After a few hours, or the next day, the symptoms will probably disappear, only to return again in a few weeks. The disc may already be seen to be red and clouded through the œdema.

(2) *Acute stage.*—This sets in suddenly, with intense shooting pain behind the eye and over the face and jaw. The vision becomes greatly impaired, and the field restricted. There is great ciliary injection, photophobia, and even œdema or chemosis of the conjunctiva. There is also considerable constitutional disturbance, often accompanied by vomiting and rise of temperature. The pupil is widely dilated and fixed, and the eyeball quite hard to the touch. The anterior chamber is usually shallow, and the iris dull and muddy. The cornea is cloudy through œdema of the layers, which prevents all details of the fundus being seen, and it is more or less insensitive to the

touch. Slowly, even without any treatment, the attack will pass off, and the symptoms entirely disappear, but the eye never remains as good as it was before the attack. The tension is permanently raised, the vision greatly impaired, and the field restricted. The excavation of the disc increases, and

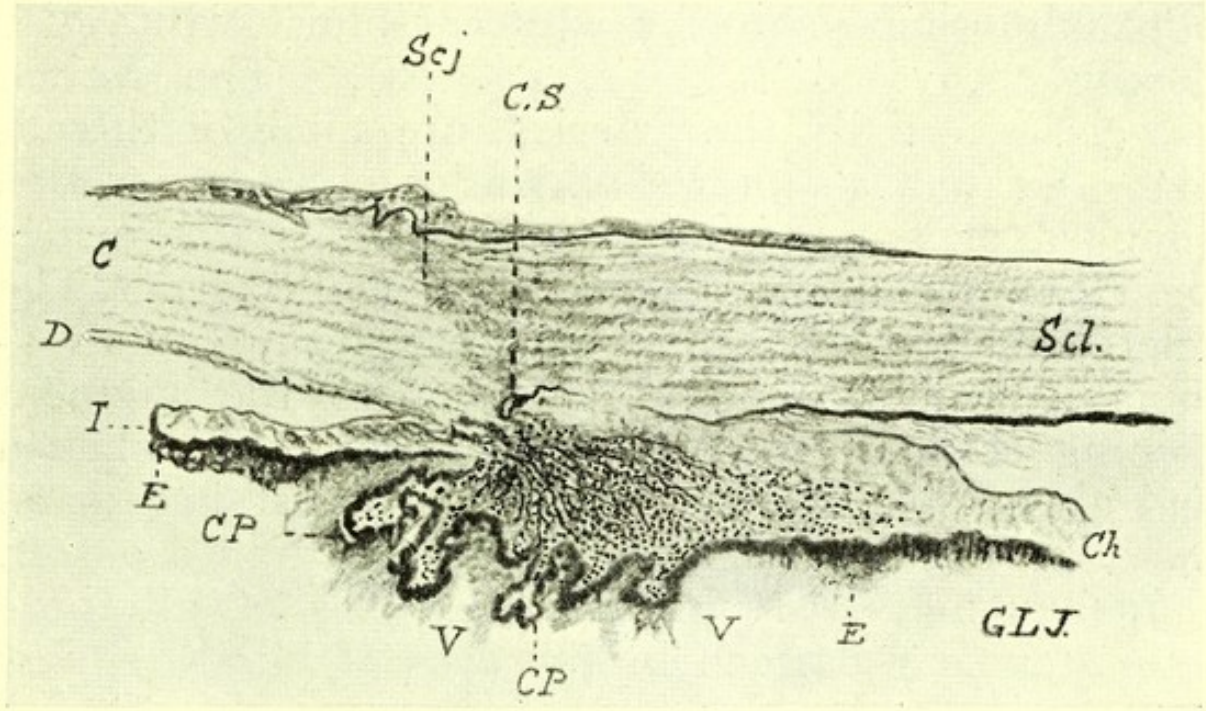

FIG. 40.—Microscopic section through the ciliary region of a case of acute glaucoma. × 30. From a photograph.

Scj. Sclero corneal junction.
C.S. Canal of Schlemm.
C. Cornea.
Scl. Sclerotic.
I. Iris.
CP. Ciliary process.
Ch. Choroid.
V. Vitreous.
EE. Exudates passing into the vitreous.

Note the thickened retracted iris, the highly stained nuclei of the proliferated cells, the abundant exudates, and the obliteration of the angle.

the fibres of the iris, by reason of the œdema, no longer appear sharp and clear, but become dull and swollen, and finally undergo atrophy. At the same time some ciliary injection permanently remains. After another interval a fresh attack will occur, with added damage, and this will be repeated until all sight has gone.

(3) *Glaucoma Absolutum.*—The eye at this stage is hopelessly blind and stony hard to the touch. The cornea is transparent, insensitive to the touch, and surrounded by a ring of large blood-vessels. The iris is almost completely atrophied, forming a greyish ring with a black pupillary border. The pupil, instead of appearing black, has a sickly green colour. The ophthalmoscope shows a white or bluish-white disc deeply excavated (Pl. 12, fig. 23). The retinal vessels, especially the arteries, are small or thread-like. Gradually the eyeball undergoes degenerative changes, accompanied by severe pain. Frequently the lens becomes opaque grey (cataract). The sclerotic becomes thinned in spots, forming staphylomata,* which are black owing to the pigment shining through. The cornea becomes roughened and glassy, like the surface of glass cast in a sand mould. The eye then gradually shrinks into a soft, shapeless mass. Numerous examples of all these later steps can be seen in the eyes of blind beggars in our streets.

Chronic Glaucoma (*Gl. Simplex*).—In this variety the above inflammatory condition is absent. The disease sets in insidiously. Beyond a slightly dilated sluggish pupil, a circumscleral congestion, and a dull cornea, one sees nothing to cause anxiety. Occasionally there is a decided increase in the tension, but unless coloured rings round a bright light be noticed, or the eye be palpated during an attack the disease may escape notice. The patient will see much better some days than others, *i.e.* during the intervals between the attacks, which gradually become more and more frequent. But test types will reveal a gradual failure of vision,

* From Σταφυλή, the uvula; ὄμα, a tumour. A name given to a local bulging of the globe.

and the perimeter a progressive contraction of the field. Notwithstanding the intervals of improvement, arterial pulsation over the disc, together with an excavation of the disc, will be perceived with the ophthalmoscope, which fixes the diagnosis at once; but at any moment this chronic state may develop into the more acute inflammatory form. In any case, the march of the disease, unless it can be arrested, is steadily but inevitably towards blindness.

Glaucoma is usually a disease of advanced life. and myopes generally escape owing to the axial

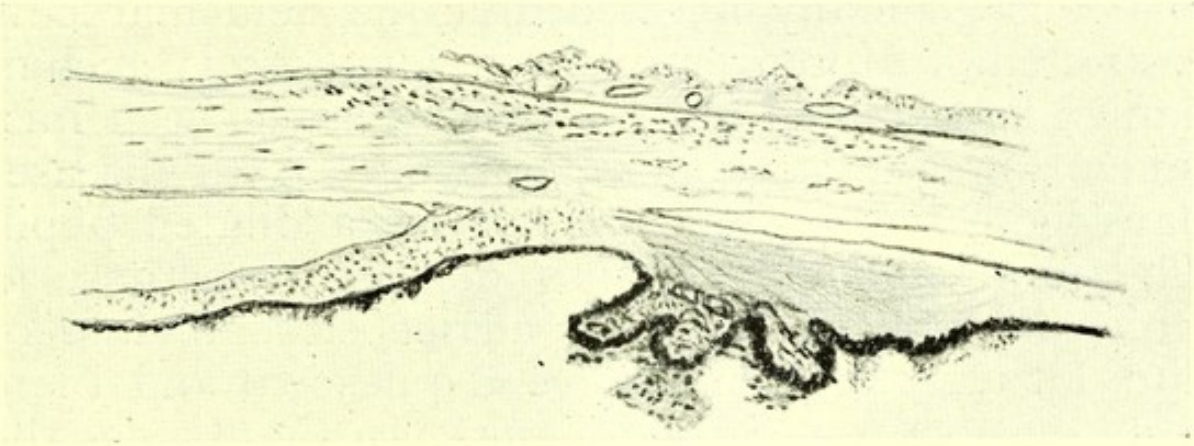

FIG. 41.—Microscopic section through the ciliary region of a case of chronic glaucoma. × 30. Note the obliteration of the angle, and a few exudates only, which ooze from the ciliary process and pigment-layer of the iris.

stretching of the eyeball, which allows of better drainage, and also admits of more fibrous-tissue shrinkage without injury to the nerve. When glaucoma occurs in children the coats yield to the internal pressure, leading to an expansion of the whole eye, which becomes very large and prominent (hydrophthalmos or buphthalmos). Since the eye expands while the lens keeps the same size, the suspensory ligament stretches, and the ciliary processes recede several millimetres from the lens. The latter, no longer held rigidly by the ligament, tends

to shift its position and become tremulous. The iris is also pulled away from the lens, so that it no longer rests against it. It then loses its support and trembles on the slightest movement of the child's head.*

Diagnosis.—Glaucoma is sometimes mistaken for other diseases. Thus, if a patient is seized with vomiting, fever, and acute pain in the head and brow, glaucoma may be mistaken for intestinal obstruction or some gastric or bowel disturbance. Again, the vomiting in hysteria or migraine may lead the student astray, as, especially in these cases, the vision is generally affected and the field markedly restricted. Moreover, violent vomiting is accompanied by a dilated pupil. Glaucoma has been mistaken for glioma and *vice versâ*. Both diseases have increased tension and usually a dilated pupil. The most difficult cases to diagnose are those in which the patient seeks for advice either before or after an attack, when the eye is quiescent and there is no tension or sign of cupping. In these cases the previous history of the case will guide the practitioner. The age of the patient, history of pain over the orbit, premature presbyopia, ciliary congestion, history of rings round a light, a dilated pupil and a constricted visual field are all in favour of glaucoma. An inexperienced student will sometimes mistake glaucoma for cataract or the reverse, especially when the cornea is cloudy.

Theories of Glaucoma.—The immediate cause which induces glaucoma must be either due to increased secretion or diminished outflow. Clinical facts militate against the former supposition, since

* It is only right to point out that many writers consider buphthalmus a separate disease, and not a variety of glaucoma due to obstruction of the exit channels.

glaucoma is not primarily an inflammatory disease such as would bring about an increase of secretion. Besides, iritis and choroiditis often lead to increased secretion, but only rarely to a rise of tension. We may therefore assume that restricted outflow is the immediate cause of the symptoms. This is produced, in the first place, by anything which tends to close the angle between the sclerotic and the iris, since it is in that corner where a large portion of the waste fluids escape, viz. between the meshes of the pectinate ligament (spaces of Fontana), and thence into Schlemm's canal. Secondly, by anything which obstructs the iris crypts, which affords a channel of escape for the aqueous only second in importance to the pectinate ligament. This is one of the reasons why instillation of atropin is so harmful in glaucoma and so likely to bring about an attack in a predisposed eye, since by dilating the pupil, and thus contracting the iris, access to the iris veins through Fuchs' crypts is denied. It also explains why eserine is so beneficial by opening up the crypts and so facilitating the out-flow, as well as by pulling the base of the iris away from the angle. Priestley Smith accounts for glaucoma as follows: The eyes most subject to glaucoma are usually somewhat smaller than normal. Now, since the lens increases in size as adult age advances, while the eye remains the same, the lens extends to the ciliary processes, and thus restricts the outflow of lymph from the vitreous into the anterior chamber, thereby raising the pressure in the chamber, and since something must yield, it pushes the lens and iris forward. This renders the anterior chamber more shallow, it narrows the angle, and squeezes the base of the iris against the sclera, thus increasing the mischief by blocking up the outlet, and thereby raising the tension. This

theory, though supported by a certain amount of clinical evidence and microscopic sections, nevertheless fails to account for those rather rare cases in which the chamber is large and deep. Besides, occlusion of the circumlental space, which plays the chief *rôle* in Priestley Smith's theory, cannot produce an inequality in the hydrostatic pressure of the two chambers for any great length of time owing to the connection between the two through the anterior ciliary veins and scleral venous ring.

Some pathologists ascribe the increased tension to a sclerosis of the minute arteries of the globe; others, again, to a diminution in their calibre through vasomotor excitation, but in either case it will lead to congestion and stasis of the venous current.

Atropine, by dilating the pupil, causes the iris to become thicker at its base and so blocks up the first-mentioned channel of escape, and at the same time prevents the escape of aqueous through Fuchs' crypts into the iritic veins by squeezing up the passages, and thereby obliterates the second mentioned outlet for the waste fluids. Of course, in an otherwise healthy eye the channels of escape are too large to be affected by it.

The Writer's Theory.—The writer ascribes, at any rate, one of the causes of the increased tension to a general increase in the fibrous tissue of the eye, which tends slowly to contract, and by so doing obstructs the excretory channels of the angle and the lumen of the veins in the iris and ciliary region. This starts the rise of the tension, which sets the match to the wood and puts the whole in a blaze. Then follow the changes which lead to a strangulation of the fibres of the optic nerve and all the other troubles. Speaking broadly, the tendency of the cells of our body with advancing

years is ever towards fibrinisation and rigidity as opposed to elasticity, as everyone is familiar with in the gradual failure of accommodation as one grows older. This diffuse transformation of the cells into fibrous tissue and diminution of elasticity only ceases at death. Now a very little contraction of this tissue, or the presence of any obstructing body, will considerably diminish the lumen of the waste channels. For example, anything which causes the ciliary processes to swell may set up glaucoma (in a suitable eye) by their pushing the root of the iris forwards against the ligamentum pectinatum and thus obliterating the sinus and choking the overflow. This, again, will throw extra work on the iris crypts and veins, which is the alternative channel of exit for the waste fluids. Now if the eye for some reason or another arrives at that dangerous condition when a very small obstruction upsets the balance between inflow and outflow, any obstruction to the exit of fluid, either from mechanical hindrance, or from a rise of blood-pressure, which in a normal eye would have no effect, will, in the condition referred to, raise the tension. This at once starts a vicious circle by setting up venous stasis, both in Schlemm's canal and in the veins of the iris which receive the waste products through the crypts.* The stasis, in turn, causes inflammatory changes, giving rise to general inflammatory œdema. The œdema shows itself in all the tissues of the eye. Then the cornea loses its sensibility, by pressure on the nerve-endings, becomes dull and cloudy, owing to minute drops of exudation

* The immense importance of the crypts and the intimate connection of Schlemm's canal with the scleral venous ring and the venous capillaries of the iris and ciliary muscle has been brought to light by the researches of Henderson.

forming between Bowman's membrane and the corneal epithelium, which disappears as soon as the internal pressure is relieved. The entire uveal tract suffers, becoming congested and sodden. This is especially evident in the iris, which becomes discoloured, and the individual fibres lose their sharpness. The pupil becomes dilated and inactive owing partly to the atrophy of the sphincter and circulus minor, which become narrower, and partly to the paralysis of the ciliary nerves. At the same time the lens and iris are pressed forward, with the result that the chamber becomes shallow. Thus we see how pressure paralyses the nerves, and by interfering with the circulation, how it causes œdema and atrophy of the tissues and cloudiness of the cornea. At this early stage the disc is merely red, cloudy, and swollen through œdema, but there is no excavation. On testing, the acuity is found to be considerably diminished and the visual field contracted, especially on the inner (nasal) side, since the nerve-fibres and blood-vessels are longest on the outer side of the retina and therefore most affected by pressure. These are the earlier symptoms of acute (inflammatory) glaucoma. In the chronic form (Gl. simplex) the tension is only slightly above normal, the cornea remains clear, and there is, in the early stages at least, little or no contraction of the field.

To what cause are we to attribute the atrophic changes in the nerve, in those cases where there is no evidence whatever of increased tension, except on the hypothesis of a constant pressure on the optic nerve, owing to the gradual contraction of the fibrous tissue?

Later Pathological Changes.—The subsequent pathological changes consist in alterations in the sclera, which becomes denser, some of the fibres

undergoing fatty degeneration. Albumen finds its way into the aqueous, just as it does into the urine during inflammatory œdema of the kidneys. The veins of the choroid and iris become distended, often leading to small extravasations of blood. The ciliary body, and especially the processes, become swollen and sodden by the venous congestion. They force the base of the iris forward, causing it to become adherent to the sclera owing to exudations thrown out. Thus the base of the iris becomes drawn forward and attached to the sclera in front of Schlemm's canal, blocking the outlet more than ever. All this œdema chokes the vessels of the ciliary body, and leads to its atrophy, which progresses until the ciliary processes almost cease to exist. At the same time the iris veins get compressed, the aqueous can no longer find its exit through the crypts, the only remaining door of escape becomes closed, the pressure rises, and an acute attack of glaucoma at once supervenes.

The disc now presents a very characteristic appearance (Pl. 12, figs. 23 and 24). The whole floor is pushed back so as to leave a deep pit, which is undercut. This causes the white scleral ring to hang over the pit, whereby the retinal vessels, when they reach the edge of the disc, become suddenly lost to view, and reappear as they curl round the scleral ring. The lamina cribrosa is very obvious and deeply pitted with grey spots, due to the contrast between the atrophic fibres and the surrounding sclerosed tissue. The retinal vessels usually appear pushed over the nasal side of the disc, so that often there are no vessels to be seen at the temporal (macula) side. There is often a second ring of partly atrophied choroid outside the white innermost ring.

If the attack is an acute one, the tension very

high, and the anterior chamber shallow, the writer prefers to instil a $\frac{1}{4}$ per cent. solution of eserine, and then passes a sterilised Beer's knife or a broad needle into the vitreous. The knife is entered just behind the ciliary body as far as the centre of the globe, and then slowly withdrawn.* It can be done in a moment without preliminary preparation. This will relieve the tension for the time being, and it will give the patient time to think over matters, and allow you to perform an iridectomy the next day, which the patient will probably not consent to on the spur of the moment. A further reason for performing a preliminary operation is that it will enable the pupil to contract, and will diminish the chances of rupturing the capsule when you let out the aqueous, owing to the pressure in both chambers being lessened. If you do rupture the capsule, the wisest course to take is to spoon out the lens in its capsule straight away. If the case assumes the chronic form it is sometimes advisable to instil a 0·1 per cent. solution of eserine twice daily, and keep the bowels relaxed, as you can always perform an iridectomy if there is any return of tension, or the field of vision becomes at all contracted. In some cases the writer has found an operation to be unnecessary, the tension remaining normal for many years. But such cases are quite the exception.

The best thing to do in the early stages of glaucoma is to perform a good broad iridectomy from 1 *to* 2 *mm. external to the corneal margin, since the tension, and with it all symptoms, will disappear for the time being, and in about half the cases operated on the vision will return to the acuity it had just before the attack, and will remain so.* In fact, the patient may with justice be said to be cured.

* For a detailed description of this operation consult the writer's work, 'Glaucoma and its Treatment,' H. K. Lewis, London.

In the remaining half of the cases the result will be far from satisfactory, and the eye will steadily go from bad to worse. The tension after the operation, or after repeated instillations of eserine, is liable to rise at any moment, and a further operation, such as the formation of an artificial fistula, sclerotomy, rupturing the pectinate ligament, removal of a piece of the sclera by trephining, or even the removal of the lens in its capsule may be required, or at least thought to be necessary, in order to reduce the tension. In very many cases of Gl. simplex the tension appears to have been entirely removed by the iridectomy, and does not occur again, and the surgeon begins to congratulate the patient on his cure. But he has raised a false hope. The visual acuity and field continue slowly to shrink week by week, nor does it cease until it ends in total blindness. Primary glaucoma, in which the progressive atrophy of the nerve is unarrested by the daily instillation of a myotic, by an operation, or by lowering the blood-pressure, is one of the most terrible diseases for the surgeon to witness in the whole of ophthalmology. He can do absolutely nothing to help his patient, week by week he sees the vision becoming dimmer, and the field more restricted, until at length it terminates in absolute blindness.

Secondary Glaucoma.—This form of glaucoma appears as a consequence of pathological changes in the course of other diseases or as the outcome of an injury or operation. The following are the most prominent exciting causes of secondary glaucoma.

(1) *Incarceration of the Iris* in a cicatrix at the angle of the chamber, whereby it becomes blocked up by exudates or adhesions.

(2) *Seclusio papillæ.*—This invariably brings about an increase of tension. The iris becomes adherent to the lens capsule all round and completely shuts off

the vitreous cavity, whereby the tension rises in the latter as the fluids have no way of escape.

(3) *Luxation of or Injury to the Lens.*—This causes the lens and iris to swell, and is one of the chief sources of danger attendant on needling a lamellar cataract. The tension should therefore be felt at least every twelve hours after the operation, so as to be ready to evacuate the lens particles the moment the tension rises unduly.

(4) *The Increase in Size of Intra-ocular Tumours* always tends to increased tension.

(5) *High Myopia with Choroiditis.*—This induces a non-inflammatory glaucoma, in some cases by reason of the hæmorrhages which occur, in other cases by closure or rupture of the choroidal vessels, and lastly by gradual degeneration of the choroid and ciliary body and especially of the choroid around the disc, where the atrophy takes the form of a depressed ring (Hirschberg).

(6) *Deep-seated inflammation of the sclera* brings about increased tension owing to the stasis in the blood-vessels (especially in the scleral veins). This causes exudation into the scleral tissue, which lessens its resisting power and causes local bulgings (staphylomata).

(7) *Any severe inflammation of the uveal tract* such as iridocyclitis may cause increase in tension. Secondary glaucoma runs the same course as the primary form.

The first thing is to remove the cause and open up the drainage either at the angle, or by tapping the cornea as a temporary measure. Swollen lens matter must be evacuated either by puncture or by a free corneal incision. A dislocated or luxated lens must be removed. In seclusio pupillæ an iridectomy should be performed at the earliest opportunity or the eye will be irretrievably lost.

CHAPTER VI.

DISEASES OF THE OPTIC NERVE.

Inflammation of the Optic Nerve.—Intra-ocular Neuritis.—Choked Disc.—Neuro-retinitis.—Retrobulbar Neuritis.—Toxic Amblyopia.—Leber's Disease.—Atrophy of the Optic Nerve.—Physiological Cup.—Atrophic Cup.—Glaucomatous Cup.—Synopsis of the Various Forms of Excavation of the Disc.

Inflammation of the Optic Nerve.—The nerve may become inflamed in any part of its course, it may spread through the retina as well (neuro-retinitis), or it may be, though rarely, limited to the sheath and neighbouring parts of the retina (peri-papillitis). For clinical purposes we cannot do better than adopt von Graefe's classification into (1) *intra-ocular neuritis or papillitis*, in which the inflammation lies in front of the lamina cribrosa, and (2) *retrobular neuritis*, in which the nerve is affected in the orbit, or originates at the nerve centres and travels along the nerve to the eye. Both these diseases may be bilateral.

Intra-ocular Neuritis (optic neuritis, papillitis, Stauung's papille, or choked disc) is characterised by a dilated pupil and a greatly swollen, inflamed disc. The swelling may be diagnosed by parallactic displacement* and by the necessity of focussing-up to

* For definition of parallax see end of this chapter.

see the head of the disc. The colour is generally red, mottled with white patches and small hæmorrhages. The margins of the disc are ill defined and merge into the surrounding parts. More often the nerve-fibres of the disc are strikingly prominent and radiate out in a brush-like manner considerably beyond the confines of the papilla, which are entirely

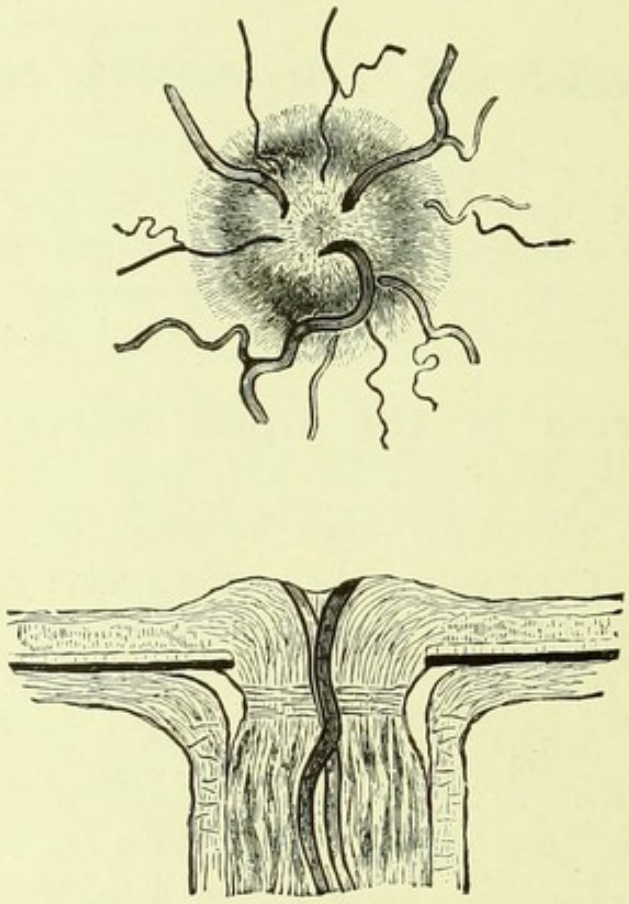

FIG. 42.—Acute papillitis showing the swollen disc and tortuous vessels.

hidden. The veins are large, tortuous, dark, and hazy, or entirely obscured in places (Pl. 13, fig. 26). All possible varieties of the above description occur. Sometimes only a part of the disc is hidden by the swelling, the rest being quite visible or merely veiled over. The vitreous is often hazy from a fine deposit over the front part. This is sure to be overlooked unless carefully focussed up. The beginner is very apt

to confuse papillitis with a simple hyperæmia and swelling of the papilla, and also with a variety of the disc occasionally met with in hypermetropic eyes, in which the margin imperceptibly fades away into the surrounding fundus, so that the edge of the papilla cannot be defined (Pl. 13, fig. 25). The history of the case, together with the occurrence of full acuity of vision when corrected with glasses, usually serves to distinguish them. In papillitis the vision is usually markedly impaired, often merely perception of light, the field is contracted, and the pupil dilated. Nevertheless, intense papillitis does sometimes occur with excellent vision, but in pseudo-papillitis, as the innocent form is called, the vessels are not engorged or tortuous and there are no hæmorrhages.

The pathological condition differs widely from that found in glaucoma. In the latter the œdema and inflammatory symptoms are due to pressure on the coats and head of the nerve from within the globe, whereas in choked disc the symptoms are caused by toxic products, which are carried down the nerve sheaths to the papilla, and so cause inflammation, or (as in the case of a brain tumour) the displaced cerebro-spinal fluid accumulates in the intravaginal spaces and nerve sheaths by pressure from behind, causing a stasis of the lymph current at the lamina and resulting in ecchymosis and œdema of the papilla, with consequent compression of the vessels. The prognosis of choked disc is always a grave one. The neuritis slowly, sometimes months after, clears up, in most cases only to leave an atrophied disc and complete blindness (Pl. 14, fig. 28). A few cases, mainly due to menstrual disturbances, rapidly develop with intense papillitis and then clear up, leaving little or no legacy

behind (Pl. 14, fig. 27). The causes of intra-ocular neuritis are usually diseases of the brain or its membranes, especially tumours, tubercular meningitis, and hydrocephalus—in fact, anything which produces engorgement of the retinal vessels, either through inflammation or direct pressure on the brain from a tumour, will produce this condition. As soon as the pressure causes a stasis of the lymph in the intervaginal space, it is at once communicated to the lamina cribrosa lymph-channels, the œdema of which compresses the main retinal vessels, venous engorgement throughout the head of the nerve ensues and the papilla becomes strangulated (choked disc).* In severe cases extravasations of blood and hæmorrhagic splashes occur, and the disc projects like the head of a mushroom, the surrounding retina being thrown into folds, while the nerve-fibres are swollen and sodden with exudates.

The causes of optic neuritis are exceedingly abundant. The most important are injuries, especially blows on the head, meningitis, syphilis, tumours in any part of the brain, sunstroke, chills, exposure accompanied by extreme exhaustion, the specific fevers, influenza, loss of blood, anæmia, chlorosis, menstrual disorders, kidney affections, diabetes, orbital injuries, or disease and thrombi or emboli of the central vessels of the retina. Other causes are diseases of the orbit, antrum, and frontal, ethmoidal and sphenoidal sinuses.

Lastly, there is the mysterious Leber's disease, which is a hereditary symmetrical optic neuritis occurring mostly in males on reaching adult life,

* A tumour, an abscess, or a large hæmorrhage in any part of the cerebrum or cerebellum may, and usually does, produce an optic neuritis, and the nerve affected will nearly always be on the same side as the lesion.

the descent taking place through the unaffected mother.

In optic neuritis the condition of things is so serious that one is justified in resorting to any means which will relieve the pressure on the nerve and its vessels. Some cases, especially those of syphilitic origin, will yield to large doses of iodide of potassium or mercury. In many cases drugs will be found useless, especially those due to brain pressure. Of late years cerebral pressure has been reduced by lumbar puncture. This is sometimes successful, and should be tried before more severe measures are adopted. If it fails, trephining over the squamous bone for preference and on the same side as the papillitis should be immediately performed. The dura mater is then incised and the temporal lobe gently lifted. This will allow of the intra-peduncular space being tapped. Or the occipital bone may be trephined and the cisterna underneath the cerebellum opened up. In either case the pressure will probably be relieved, *but if you decide that the operation should be performed, the sooner it is done the better for the patient.*

Neuro-retinitis is an extension of optic neuritis along the retina, and will be considered under "Retinitis."

Retrobulbar Neuritis (Neuro-retinitis descendens). —This may start from the nerve centres and travel along the nerve, or it may arise in any part of it behind the eye. We distinguish two main forms—acute and chronic. The former begins with a sudden impairment or loss of vision in one or sometimes both eyes. The pupil may be dilated and fixed. The ophthalmoscope reveals nothing except a slight papillitis; often that is wanting. There are general symptoms, such as severe headache,

nausea, tenderness of the eyes on pressure. The prognosis is usually good, the sight recovering gradually and often completely. The disease may be engendered by a violent chill, or any of the acute specific fevers, and it has been observed in several cases to have followed a severe attack of influenza.

Toxic Amblyopia.—This is the chronic form of the disease, and is due to the cumulative action of certain poisons, especially nicotine (tobacco amblyopia), alcohol, and lead. Diabetes is also a frequent cause. An epidemic of blindness due to atrophy of the nerve broke out among a herd of cattle in Monmouthshire, which the writer traced entirely to the drinking-water supplied from a lead cistern. The ophthalmoscope reveals nothing except a papillitis or a pale outer quadrant of the disc. This latter is seen in tobacco and alcohol amblyopia, and the pale section is due to the papillo-macular bundle (which contains the fibres of the macula area) being affected. This degenerated bundle of fibres has been traced by Uhthoff in alcohol amblyopia through the optic nerve, and may be recognised by differential staining as an oval or circular patch near the centre of the nerve just behind the lamina cribrosa. The symptoms are nyctalopia (the patient seeing best in dull light) and central scotoma for red and green—in other words, the patient is blind to these colours in the direct line of sight, and defective central vision. Pipe tobacco, especially shag, is the chief cause of the tobacco amblyopia in England, and liberal quantities of alcohol accentuate it. Large doses of quinine (45 grains and upwards), extract of male fern, and pomegranate-root have been known to set up an acute form of toxic amblyopia accompanied by an extreme contraction

of the retinal vessels, together with destruction of many of the ganglion cells.

I have never met with a case which has completely recovered, unless the tobacco and alcohol have been stopped at a fairly early stage, but considerable amelioration will always be obtained if the patient becomes a total abstainer from the exciting cause. Subcutaneous injections of strychnine are sometimes beneficial.

Atrophy of the Optic Nerve.—This, as we have seen, is the usual termination of optic neuritis, severe retinitis, and glaucoma, but it may occur as a primary non-inflammatory affection. In this case we notice the papilla becomes gradually paler, at first here and there in spots, at the same time the lamina cribrosa more distinct and fenestrated. The minute capillary vessels of the disc disappear, and the retinal arteries (but not the veins) become thinner. The vessels are often bordered by whitish lines (perivasculitis). Gradually the disc becomes excavated and the parallactic displacement may be measured, the deterioration of sight keeping pace with the atrophic changes observed until all sight has left the eye, when the disc will appear chalky white, grey, or of a pearly-bluish white colour (Pl. 11, figs. 21 and 22). At this stage the nerves will have lost their medullary substance and will be found converted into fine shrunken fibrillæ without any double contour. At the same time the rest of the fundus appears more vividly red than normally. Although blindness is invariably accompanied by a white disc, still, cases are occasionally seen in man in which a chalky-white disc has been accompanied by excellent vision. For example, one may occasionally see a white disc in disseminated sclerosis. This is the normal appearance of the

disc in a large number of animals, notably those which are protected by Nature, such as the Rhinoceros, Armadillo, Porcupine, Ant-eater, and Echidna. A white disc, therefore, is strongly presumptive of atrophy and blindness, but not necessarily so. The causes of non-inflammatory atrophy are for the most part brain and spinal affections, especially tabes dorsalis, disseminated sclerosis, general paralysis, sleeping sickness, and cerebro-spinal tumours. In all spinal cases remember to look for signs of atrophy of the optic nerve, with corresponding loss of visual acuity; also for the Argyll-Robertson pupil,* a fixed and greatly contracted pupil, or unequal pupils, absence of knee and ankle reflexes, and contracted visual field. Colour-blindness comes on early in the disease, red first, and then green. Blue rarely ceases to be recognised and only dies out with the sight. The prognosis is usually very bad, especially in adults. In children, if the exciting cause is removed, sight may occasionally return. Cases have been known to recover after injuries and concussions of the body from a fall, the sight returning completely after a long period of total blindness.

If no cause for the disease can be found, you should make the "complement-fixation" test for syphilis,† and if you get a positive reaction (viz. no sign of hæmolysis) you will not go far wrong if you set the disease down to syphilis and treat it accordingly, even if the patient persistently denies ever having contracted it and you can find no indication of a chancre. Atrophy is often the only symptom observed in a case of tabes, both acquired

* The Argyll-Robertson pupil is a characteristic and early symptom of locomotor ataxy, in which the pupils respond to accommodation, but not to light.

† See Appendix.

and congenital. A blow on any part of the skull may be followed by optic atrophy of one or both eyes, which sometimes develops without any previous optic neuritis sufficiently marked to be noticed.

Lastly, daily exposure to a dazzling light, as is the case with workers at glass furnaces, has been found to lead to optic atrophy and blindness of both eyes.

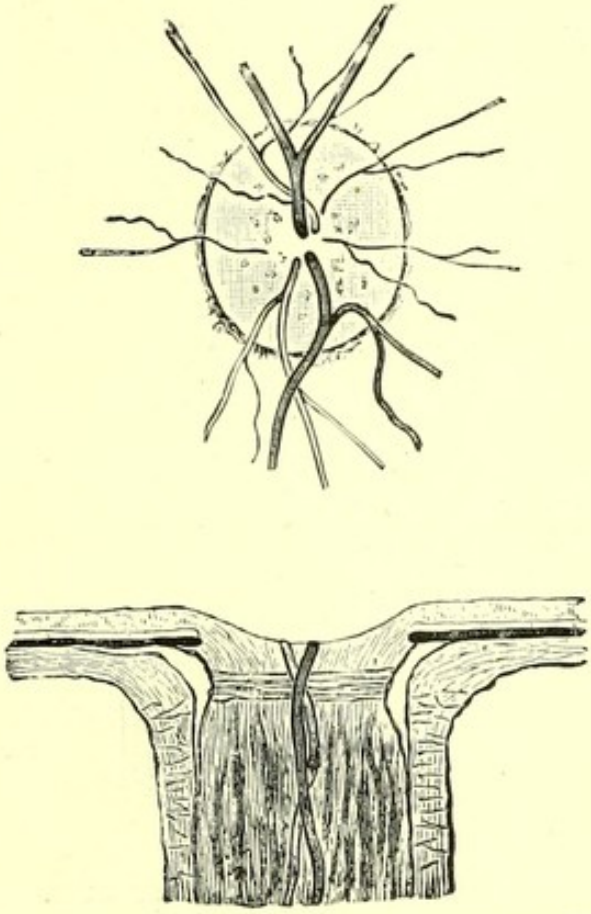

FIG. 43.—Atrophic disc showing shallow cup.

Excavation of the Disc.—Three varieties of excavation are met with, and can be readily diagnosed, viz. the physiological cup, the atrophic cup, and the glaucomatous cup. The first variety exists normally, in perfectly healthy eyes, and is due to the fact that the fibres of the nerve separate out to spread over the fundus at a lower level, *i.e.* behind the retina. In this case there is cupping, which is

often very marked and deep, but it never extends throughout the papilla, the rim, or at least the outer half being of a healthy pink colour, and for the most part on a level with the retina. The vessels never curl over the peripheral margin of the disc. The lamina cribrosa is normally placed and level, although often chalky-white and deeply

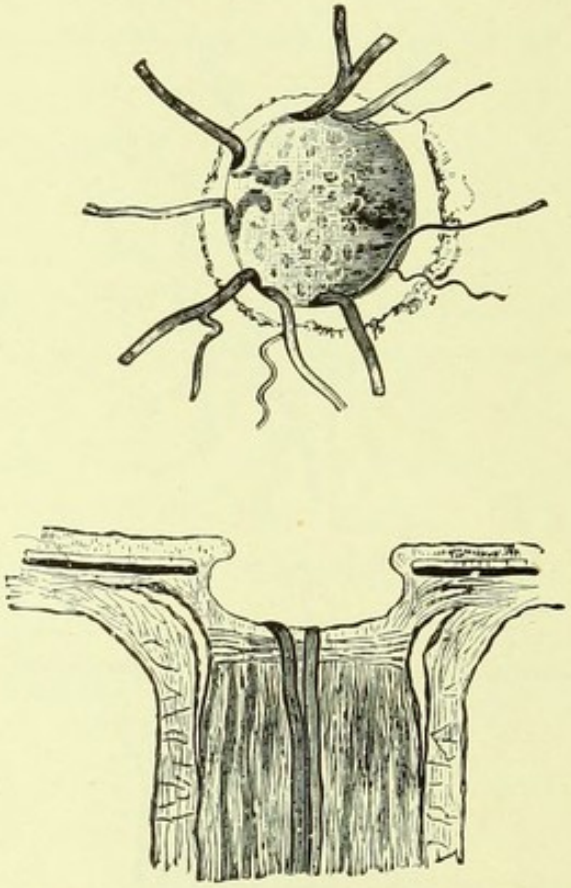

FIG. 44.—Glaucomatous cup.

pitted (Pls. 9 and 10, figs. 17, 18, 19, 20; also Pl. 6, fig. 11).

The second variety of excavation is uniformly pale, or sometimes a chalky-white colour. It is slightly sunk everywhere, owing to the shrinkage and atrophy of the nerve-fibres as they pass over the retina. The disc is therefore uniformly depressed, and lies close on to the lamina cribrosa, which is flat and *in situ*. The disc is usually very sharply defined.

The vessels pass over its edge, with little or no dip (Pl. 11, figs. 21 and 22).

The third variety is characterised by the lamina being concave and greatly depressed, so as to form a true cup. In glaucoma of long standing it may be greatly undercut, so that the retinal vessels are lost to view at the edge of the disc, coming into view again as they curl over the rim. It may be distinguished from the physiological cup by the fact that the excavation embraces the entire disc, and from both varieties by the concave shape and recession of the lamina. The disc is often pinkish in the earlier stages, but gradually whitens and thins down, exposing the pitted lamina on its floor as the disease continues. The vessels often appear pushed over to the nasal side. The arteries are usually smaller, and the veins fuller than normal (Pl. 12, figs. 23 and 24).

The following table shows the differences at a glance:

Physiological Cup.	*Atrophic Cup.*	*Glaucomatous Cup.*
1. Excavation confined to central half or a portion of disc only.	Excavation (if any) occupies the entire disc.	Excavation occupies entire disc.
2. Excavation often undercut, but this never extends to outer margin of disc.	Excavation shallow, flat, and never undercut.	Excavation usually undercut, often extensively so, and extends to the outer margin of disc.
3. Some portion of the disc (usually the inner half) is a natural pink colour.	Disc *never* a natural pink colour at any part, but uniformly creamy or chalky-white, and margin sharply defined, except in retro-bulbar atrophy, when it is blurred.	Disc may or may not be pink according to the state of the disease. At a late stage it becomes white or bluish.

Physiological Cup.	*Atrophic Cup.*	*Glaucomatous Cup.*
4. Lamina normally placed and flat, chalky and spotted. Parallactic* displacement over central portion of disc.	Lamina normally placed, white and flat, not spotted, not much parallactic displacement as a rule. In retrobulbar cases the lamina is concealed by fibrous tissue.	Lamina greatly depressed, sunk, and concave, often chalky or bluish-white, and spotted, parallactic displacement over whole floor of disc.
5. Vessels never curl over the outer edge of the disc, but they curl over the inside lip of the disc.	Vessels rarely curl over the edge of the disc, and then only slightly.	Vessels curl over the edge of the disc. Usually a marked feature.
6. Vessels spread out normally as a rule.	Vessels spread out normally.	Vessels often pushed over to nasal side of the disc.
7. Vessels normal in size.	Arteries and veins usually contracted, or even thready.	Vessels may be normal in size or arteries smaller and veins larger than normal.
8. Acuity normal on correction with glasses.	Acuity always greatly reduced, or *nil*; cannot be improved with glasses.	Acuity often greatly reduced, but central vision may be normal.
9. Visual field normal.	Visual field always contracted.	Field very contracted, often confined to macular area (5°).

Injuries of the Optic Nerve.—Injury of the nerve is generally followed immediately by blindness of the eye. More rarely the blindness is only partial. At first little or nothing abnormal can be noticed with

* Parallactic displacement (or simply parallax) is the displacement of one object with respect to another object behind it when observed from different standpoints. For example, when you stand directly in front of a clock, at nine o'clock the minute hand will point to XII. If you move to the right it will point to one minute to nine. If you move to the left it will point to one minute past nine. This is due to parallax, and the amount of parallax increases with the distance apart of the two objects.

the ophthalmoscope unless the central artery or vein is injured, but in the course of a few weeks simple atrophy of the disc may be observed. This is due to the atrophic changes descending along the nerve. The visible changes are similar to those met with in simple atrophy from other causes, and these changes are observable in the vessels as well. The arteries shrink up into mere threads, while the disc becomes chalky white and sharply defined.

Tumours of the Optic Nerve are rare. They arise either primarily from the sheaths or neuroglia of the nerve (fibromata, sarcomata, and myxosarcomata), or secondarily by extension from the neighbouring parts. Blindness always sets in early and is a characteristic sign. At the same time an acute neuritis is set up with excessive venous engorgement. If the tumour is encapsuled and can be removed along with the globe and as much of the nerve as can be reached by the scissors, the chances of recovery are good.

CHAPTER VII.

DISEASES OF THE RETINA.

Normal Retina.—Nyctalopia.—Hemeralopia.—General Characters of Retinitis.—Albuminuric Retinitis.—Diabetic Retinitis.—Lipæmic Retinitis.—Leukæmic Retinitis.—Syphilitic Retinitis.—Retinitis Circinata.—Retinitis Proliferans.—Retinitis Pigmentosa.—Simple Atrophy of the Retina.—Detachment of the Retina.—Hæmorrhage of the Macula.—Massive Exudation of the Retina.—Arterio-sclerosis of the Retinal Vessels.—Hæmorrhagic Retinitis.—Embolism of the Central Artery.—Thrombosis of the Central Vein.—Tay's Disease.—Progressive Degeneration of the Macula.—Excavation of the Macula.—Glioma of the Retina.—Pseudo-glioma.—Commotio Retinæ.

THE healthy normal retina is invisible. If any part of it can be seen, it is due either to a congenital abnormality, to disease, or detachment. The visual purple, which, as we have stated, surrounds the outer halves of the rods, does not contribute to the colour of the retina proper, and only to a slight degree to that of the fundus. Anæmia and chlorosis, which present such marked changes in the colour of the gums and face, effect no alteration of colour in the fundus beyond a general pallor of the disc. A change of colour in the fundus must therefore be traced to other diseases, such as embolism of the central artery, retinal or optic nerve atrophy, commotio retinæ, to the contraction of the retinal arteries and capillaries or as the result of acute poisoning

(quinine, ergotine, etc.). A temporary alteration of colour may occur, however, during severe attacks of migraine, or irritation of the cervical sympathetic.

Nyctalopia, or the condition of seeing better in a dim light (day-blindness), is often due to a physical cause, such as central scotoma, which occurs in tobacco amblyopia, polar cataract, and central opacities of the lens. In these cases the pupil naturally dilates in a reduced light, and the patient notices his defect of sight much less, and therefore thinks he sees better. Other cases are due to hyperæsthesia of the retina, which may be caused by hysteria, or an error of refraction or accommodation, which produces an intolerance of light. In a dim light, or by wearing dark spectacles, this trouble vanishes.

Hemeralopia, or the condition of seeing better in the daytime (night-blindness). This occurs when the peripheral part of the visual field is deficient, either from peripheral opacities in the media (as in some forms of cataract and corneal disease), or in retinitis pigmentosa, in which the outer (percipient) layers of the retina being degenerated, a greatly increased stimulus is necessary to enable persons to see, which stimulus at night-time, or in a dim light, is wanting. Scurvy, xerosis, and all diseases associated with mal-nutrition may induce the defect.

Hyperæmia of the Retina, although mentioned as a separate disease in many text-books, is so bound up with retinitis that it may be considered as one of its symptoms. The colour of the fundus outside the disc undergoes no alteration any more than in anæmia, but other changes occur which are exceedingly conspicuous, and which we will now consider.

Retinitis.—The changes seen in severe forms of retinitis are due to disturbance of the circulation.

The retina no longer remains transparent, but becomes dull and cloudy, and the outline of the disc indistinct. These changes are the result of exudations from the vessels. As the inflammation intensifies these exudates increase, the red corpuscles escape through the capillary walls, and splashes of blood may be seen here and there, following the general direction of the vessels, which resemble brushes, as if combed out into short fine streaks. Lenticular, or irregular-shaped glistening white, or yellowish patches, often of considerable size, are frequently seen. The vessels distend and increase in length, thereby becoming tortuous. The chief reason for the peculiar changes observed in retinitis is due to the fact that the retinal vessels do not anastomose. Hence, any thrombosis or obstruction of an artery leads to inflammation, hæmorrhage, and exudation and œdema, together with obliteration of the distal branches over the area of supply. In a word, the changes are due to infarcts, and are similar, at least in some of their characters, to those which occur in the lungs, liver, and brain. Since the macula ring is surrounded by a chain of retinal capillary loops, changes in them, leading to embolism and exudation at the macula and its vicinity, are very frequent, and should always be looked for. As a rule the exudates are not confined to the retina, but ooze out into the vitreous. The first symptom the patient will notice is a cloud in front of his sight, which will be the main cause of his seeking advice. Should the inflammation continue the vision will become greatly impaired, and the perimeter will reveal scotomata corresponding to the white patches and hæmorrhages. If the retinitis begins to clear up and the woolly patches and hæmorrhages become absorbed before atrophy of the retina or papilla

sets in, provided there is no marked macula change, the prognosis is hopeful; otherwise the sight will be irreparably lost. The retinitis of pregnancy is one of the least serious forms of the disease.

Causes.—Retinitis is most commonly the result of grave metabolic changes in the system, such as are brought about by various forms of renal affections, among which Bright's disease is the most important; also from syphilis, diabetes, menstrual disorders, pernicious anæmia, pregnancy, and leucocythæmia, or it may follow changes in the vascular system, especially arterio-sclerosis leading to hæmorrhage of the choroidal vessels (retino-choroiditis), embolism of the arteria centralis, or thrombi in the retinal veins (septic poisoning). In these cases the retinitis is usually bilateral. Unilateral retinitis, on the other hand, may be due to a local embolism, or to injury. Thus gazing at the sun or the arc light may induce it, producing maculitis with intense and persistent after-images, and in severe cases permanent destruction of the nerve ends similar in character to the changes in the organ of Corti, which result from the noise of violent explosions, or from long-continued hammering, as in boilermakers' deafness. Again, blows on the eye may cause a cloudy swelling over a considerable area of the retina, which usually undergoes resolution after some time (commotio retinæ).

General Diagnosis.—Severe and advanced cases of retinitis can always be recognised, but at the very commencement of the disease, or in very mild forms, there is often an element of great uncertainty. Thus blurring of the papilla may be due to a high degree of astigmatism, or simple hypermetropia, or slight opacity of the media. Any of these causes may give rise to a disc showing capillary congestion,

with its edges more or less blurred and ill-defined, and indistinguishable *per se* from a mild form of papillitis. Again, opaque nerve-fibres, especially in extreme cases, are apt to be confused by the student with the white woolly patches of albuminuric retinitis which sometimes surround the papilla. But in the congenital abnormality there is no sudden falling off of acuity, the disease is often unilateral, and the patches are usually confined to the vicinity of the disc. There are no hæmorrhages or glistening patches, or striæ near the macula. The hyaline thickening of the retinal vessels is absent, nor are the latter tortuous outside the opaque patches. Moreover, albuminuric retinitis occurs very late in the history of the disease, which makes the diagnosis much easier. Neuroretinitis is only a form of retinitis, in which the papilla is markedly inflamed.

Albuminuric Retinitis.—Of all retinal affections this is by far the commonest as well as the most diverse in its appearances. Although usually found during the later stages of kidney disease, especially the chronic contracted form, it may arise during the albuminuria of pregnancy, diphtheria, and scarlet fever. The most important changes are to be sought (1) at the papilla, and (2) around the macula.

The papilla varies greatly in appearance. It may be swollen, and its margin ill-defined, or lost in striæ which radiate in short lines into the surrounding fundus, or it may be only slightly hyperæmic (Pl. 15, fig. 29). Lastly, the disc may be enveloped in a white, opaque, glistening or woolly effusion, which extends a variable distance into the fundus, partly concealing the main vessels.

The macula is often surrounded by a characteristic irregular crescent of chalky-white patches or dots or radiating streaks. Occasionally, instead of dots or

streaks, a group of white flocculent patches are seen. The radiating streaks form the most characteristic lesion of all (Pl. 15, fig. 30). The rest of the fundus presents various appearances. Sometimes it is quite normal, more often single or multiple hæmorrhages are visible, which usually follow the main vessels, with here and there white flocculent (woolly) patches. These may be due to—(1) partial organisation of effused leucocytes; (2) exudation of fluid between the nerve-fibres; and (3) to varicose swellings of the nerve-fibres themselves. Often the veins are swollen and dark, and the retina appears of a greyish tint from the exudates. Hyaline thickening of the vessels is common. The arteries have an unusual glint due to the brilliant light streak. They are often bordered by white lines of effusion. Sometimes the walls of the vessels become changed into white chalky-looking bands. In other cases a vein may appear broken at the spot where a thickened artery rendered almost invisible by hyaline degeneration crosses it, but does not stop the circulation. The appearances may assume two general forms, according as the disease is acute or chronic.

In the *acute* form the disc is œdematous, red and hazy, or striated, and the fundus strewn here and there with white patches and flame-shaped hæmorrhages (Pl. 15, fig. 29). A sudden blindness (uræmic amaurosis) may supervene. The blindness comes on in a few hours, accompanied by great constitutional disturbances, headache, vomiting, convulsions, etc. As the uræmic poisons become excreted from the system the sight may gradually return. The pupil acts to light, showing that the blindness is psychic and not optical.

In the *chronic* form the patches are very small, of a white glistening appearance often rendered still

brighter by crystals of cholesterin. They are caused by fatty changes which have taken place in the effused products. The disc is better defined in outline, less œdematous (Pl. 15, fig. 30), and the hæmorrhages few or wanting.

Albuminuric retinitis in chronic renal disease is a grave sign. The patient almost always dies within eighteen months or two years after its first appearance. The writer can only recollect one case in which the patient was alive five years afterwards.

Pathology.—This may be summed up in three words: inflammation, exudation, and degeneration. The morbid products engendered by the perverted metabolism first of all produce inflammation of the endothelium of the vessels, with thickening of the walls, stasis, thrombi, local hæmorrhages, and exudation. The coagulation of the exudates and changes in the nerve-fibres give rise to the white patches previously mentioned, and the fluid exudation causes the papilla to swell and its outline to become hazy. The fluid exudates are distributed throughout the retina and choroid, and cause first inflammatory and then degenerative changes. Thus, the vessels of the choroid undergo sclerosis, and later on hyaline, colloid, and fatty degeneration of the endothelium, with infiltration of lymphoid cells. The lamina vitrea is filled with colloid products. The hexagonal layer proliferates, and the pigment gets distributed throughout the retina. The rods and cones, deprived of their proper nourishment and pressed upon by the exudates, break down and become matted together. Cyst-like spaces and hyaline deposits, derived from the white blood-cells are formed in the outer reticular layer and the ganglion cells, which become squeezed into the nerve-fibre layer and undergo hyalin degeneration.

The latter become thickened, and form varicosities and fusiform swellings. Here, too, the hæmorrhages occur. Müller's fibres also become sclerosed, and then undergo fatty degeneration. Local foci of fatty and hyalin degenerative products account for the small glistening dots, while the white striæ round the macula are probably due to similar changes in the fibres of Müller and nerve-fibres as they bend round the mouth of the foveal pit, and then spread centrifugally outwards. We see, therefore, that the general tendency is towards total degeneration of the retina and the retinal vessels, in consequence of the perversion of the nutrition of the parts.

The course of retinitis is always a prolonged one. Often many months elapse before the symptoms subside, and generally speaking, the longer it takes the more will be the impairment of the sight afterwards. Diffuse pigmentation and atrophy of the retina are among the commonest sequelæ.

Retinitis Diabetica.—In this disease (which is quite rare) the changes are much fewer and less conspicuous. The papilla is usually normal, and the visible ophthalmic changes are mostly limited to small hæmorrhages, which are sometimes very numerous, and may even invade the vitreous, and to white deposits around the macula. These occur in the form of spots and small patches arranged in an irregular crescent. Occasionally there is a large patch as well. The appearance closely resembles the albuminuric form, but may usually be distinguished by the absence of marked papillitis.

Retinitis Lipæmica.—This is a variety of retinitis diabetica which is characterised by a very striking salmon-pink fundus (Pl. 21, fig. 42). The retinal arteries and veins are indistinguishable and of a buff

or straw colour, becoming pink near the disc. They appear to be somewhat flattened and larger than normal, but are not tortuous or varicose. The disc has a peculiar dark grey colour with a pinkish margin. The blood is very pale and milky from an immense quantity of fine granules of a peculiar fatty substance which does not stain with osmic acid. It is probably related to albumen or cholesterin.

Retinitis Leuchæmica is also characterised by the peculiar orange-pink hue of the fundus. This change of colour is attributed to the great preponderance of leucocytes over the red corpuscles. In addition to the colour the vessels will be seen to be enormously dilated and tortuous, and the veins appear of an unusually pale red colour. The extraordinary distension of the vessels may be explained by the great excess of leucocytes, which are larger than the red ones and tend to block and distend the channel. The walls of the veins lose their elasticity and so stretch under the pressure. A large number of small whitish spots are seen scattered over the fundus, or confined to the periphery. Hæmorrhages are common, and the spleen and lymphatic glands will be found enlarged.

Retinitis Syphilitica.—This may be congenital or acquired. It is often (and always in the diffuse form) an extension of the choroidal or choroido-iritic affection. Hæmorrhages are seldom met with. There are two varieties—circumscribed and diffuse.

The *circumscribed* takes the form of a thick yellowish or yellow-white exudate, which occupies the whole macula area, or is even larger. This gives rise to a central scotoma. More rarely it appears to spread out from the side of one of the big retinal vessels. The exudate may become absorbed, or it may be converted into a large greyish ill-defined scar. The optic nerve is slightly congested

and veiled. There are rarely any hæmorrhages. Relapses are frequent. Metamorphopsia is a distortion of the image seen by the patient, which is occasionally met with in this and many other forms of retinal disease affecting the macula. The distortion is due either to the cones becoming squeezed together, so that more cones cover a given area than normally, which magnifies that portion of the image seen (megalopsia), or, as is more usual, the cones are pushed aside, and objects which fall on that region appear smaller (micropsia).

The *diffuse* variety, which is much more common, affects the whole retina, which is grey and cloudy. Round about the macula other spots of a deep grey colour are to be seen. As it always starts from the choroid, or more exactly from the choriocapillaris, it is the deeper layers of the retina which are chiefly affected, and the hexagonal layer suffers most, since this part of the retina is nourished exclusively by osmosis from the choroidal vessels. The pigment breaks up and becomes deposited, irregularly invading the inner layers, as in retinitis pigmentosa. The vitreous is hazy from a dust-like deposit of exudates, or even an organised floating membrane. In this form the patient suffers from night-blindness. The hereditary (congenital) form is described under choroiditis.

Retinitis Circinata (Fuchs).—This is characterised by a crescent-shaped or oval belt, made up of innumerable chalk-white dots or patches, which surround the macula, and lie behind the retinal vessels. It occupies a considerable area of the fundus. It is usually accompanied by profound changes at the macula (Pl. 17, fig. 34), and is often surrounded by patches of a milky-looking exudate beneath the vessels. The white spots are probably due

to hyaline and fatty degeneration of the red corpuscles (Ammann). The significance of this condition is not yet known.

Retinitis Proliferans.—This is characterised by dense bands of connective tissue, which forms scars extending a long way over the fundus, often as far as the ora, or even into the vitreous. The origin of these scars, in some instances at least, is doubtful, but in many cases they have been traced to extensive pre-retinal hæmorrhages, which are probably due to the action of a toxic influence, such as is produced in the course of gout, syphilis, Bright's disease, or diabetes. These weaken the coats of the vessels and cause them to break down and bleed. These hæmorrhages form clots, which subsequently become organised into a dazzling white scar-tissue (Pl. 17, fig. 33).

This is the more probable, as fresh hæmorrhages are often met with in various parts of the field. Other cases are due to hæmorrhages just behind the membrana limitans, which they push forward. At the same time lines of connective tissue may be seen along the sides of the large vessels, and especially at and around the disc. It is in this region that the largest hæmorrhages occur, and consequently the most abundant formation of scar tissue. In fact, this disease may be said to take origin from the vessels at the disc and surrounding parts. Noyes believes R. proliferans to be a variety of hereditary syphilis, since later on spots of choroidal atrophy surrounded by pigment appear (choroiditis disseminata). The bands are sometimes flattened out into membranes, which curve round, enclosing oval spaces in the fundus, which are strewn with moss-like pigment (Pl. 17, fig. 33). Now and then the bands over the hæmorrhage appear glossy, which has been ascribed to a localised detachment of the retina.

At other times they form long white cords of scar-tissue. Usually these bands lie *on the retina* between it and the vitreous, and this is the form of disease first described by Manz. In some cases these cicatricial formations do not occur in front of, but *behind* the retinal vessels, and are derived from the adventitia of the larger branches. Whether this forms a separate disease or is merely a variety of the same affection is not clear. This variety is called plastic or cicatricial retinitis by some writers to distinguish it from R. proliferans. There is no recognised remedy for this disease.

Retinitis Pigmentosa.—This disease is essentially a progressive chronic atrophy of the retina, which always affects both eyes simultaneously, and is unaccompanied by signs of inflammation. It is difficult to say whether it commences in infancy, but the first indications are generally noticed after puberty by the patient complaining of inability to see at dusk (hemeralopia) The perimeter, if well illuminated, shows a normal field, but it falls off immediately the light is reduced. Slowly year by year the symptoms increase, until the patient can only see clearly in brilliant daylight. The field then becomes contracted in full daylight, and ultimately all sight is lost.

The ophthalmoscope in the early stages reveals a zone of fine black branched pigment, resembling bone corpuscles, which encircle the extreme periphery of the field (Pl. 16, fig. 31). As time progresses the pigment increases, invading the fundus more and more, spreading towards the pole and following the course of the vessels, especially the veins, and consequently the direction of their blood-current. Sometimes the pigment, instead of forming branches, is distributed over the fundus in fine dots, like pepper-

dust (Pl. 16, fig. 32). The choriocapillary layer is the first to degenerate, and as this nourishes the hexagonal layer, the latter breaks up and disappears, and the pigment gradually makes its way to the surface, so that it comes to lie in front of the retinal vessels. This dissolution of the hexagonal layer causes the choroidal network of vessels to become very conspicuous and sharply defined. At the same time the walls of the retinal vessels become thicker, and the light-streak vanishes. Later on their calibre gets reduced, until they become mere reddish threads. The optic nerve then atrophies, the disc assuming a peculiar yellowish waxy look (Pl. 16, fig. 32). The connective tissue hypertrophies, which causes all the layers of the retina to become disorganised and atrophied, the hexagonal and bacillary layers being the first to degenerate. The choroid becomes filled with colloid deposit, and the retina slowly changes into a layer of connective tissue. Two other diseases of congenital origin have been described as related to retinitis pigmentosa. The first is known as retinitis punctata albescens (**Mooren's disease**). It is characterised by an immense number of discrete white dots scattered over the entire fundus. They lie behind the vessels. There are no other changes of importance, but the vision is somewhat impaired. The other variety is almost identical with retinitis pigmentosa, save that there is an entire absence of pigment. Both these forms are very rare, and the pathology is entirely unknown. See also note on p. 100.

Origin.—Retinitis pigmentosa, as far as we know, is unconnected with any disease.* It is essentially

* There is, however, a disease of syphilitic origin which closely resembles the true retinitis pigmentosa, so much so that some writers look upon syphilis as a cause of this disease. But this does not affect the general statement given in the text.

hereditary, often appearing in several members of the family, and usually dominant, that is to say, occurring through successive generations directly from parent to child. About a quarter of all cases are connected with consanguineous marriages. Moreover, it sometimes occurs along with other congenital anomalies, such as harelip, supernumerary fingers, toes, and nipples, persistent hyaloid sheath, or branchial clefts. In examining the *Galagos*, a family of the Lemuridæ, which rank next to the monkeys, I noticed they all possessed a fundus strikingly like that of an advanced stage of retinitis pigmentosa, in the fact that the entire circumference of the field was strewn with the characteristic bone-corpuscle pigment branches. Now the *Galagos* are all-night animals, never opening their eyes until sunset. I noticed the same appearance in all the Crocodilia, and many other orders, which invariably seek their prey at dusk. It occurred to me that this disease might be a reversion to an ancestral form, in which the eye was adapted to dim lights only. I therefore secured some *Galagos*, and exposed them to a continuous bright light, so that they could not avoid the light. Gradually atrophy of the nerve supervened, and in less than six months they all became blind. I then tried another experiment on one of three members of a family afflicted with retinitis pigmentosa, in about the same stage of advancement, by fitting him with goggles having coloured spectrum-blue glass fronts, which cut off both ends of the spectrum and all extraneous white light, so that only the blue, green, and yellow rays were transmitted. I examined him constantly for three years, and I found the disease made practically no advancement, while his brother and sister

were markedly worse. If this should be established in other cases, even if the theory of reversion be found untenable, it will at least show that the light rays of one or other end of the spectrum act injuriously on such eyes, and tend to degeneration of the retina, as was found to be the case with the *Galagos*, and further, that we have a means of arresting the disease indefinitely. It might be objected that patients suffering from this form of retinitis see best in daylight, whereas the *Galagos* see best at night. But we do not know that they see better at night-time; we only know that the daylight is injurious, and that the intense light-stimulus tends towards retinal atrophy in the *Galagos*, as well as in patients having this disease. But to carry out the preventative treatment it is essential that the goggles should exclude all unfiltered light, which should never be allowed at any time to reach the eyes. If the ultra-violet rays are the cause of the degeneration of the retina, it is probable that Drs. Schanz and Stockhausen's (No. 5) Euphos glass, or else Flemos glass, which entirely absorb the ultra-violet rays, will serve the purpose as well, or even better, than a spectrum-blue glass. (See footnote, page 162.)

Simple Atrophy of the Retina.—This is a similar disease to the last, only there is no appearance of pigment, otherwise the symptoms are identical. Probably Mooren's disease (retinitis punctata albescens), in which the fundus outside the macula is strewn with small, round, chalky-white dots, is of the same nature, in fact some pathologists consider them both as mere varieties of retinitis pigmentosa. The subjective symptoms are the same, except that the disease, instead of pursuing its course to blindness, remains stationary.

Detachment of the Retina.—Two distinct forms of detachment occur: One, which is seldom met with, in which the hexagonal layer is separated from the choroid; the other in which the retina is detached from the hexagonal layer, which remains *in situ*. In the former case visual acuity is diminished, but not lost, since the hexagonal layer continues to nourish the outer layers of the retina. Hypermetropic and irregular astigmatism are at once revealed. We shall only consider the second form.

Cause.—Detachment may follow a blow on the eye, a fit of coughing, a sudden concussion from a fall, or during gymnastic exercises. Loss of vitreous from any cause may readily lead to detachment, and even cutting through the cornea for an iridectomy has been known to produce it by the sudden release of the aqueous. High myopia, owing to the hyalitis which so often accompanies it, is a frequent cause of detachment. In fact, any severe inflammation accompanied by exudates into the vitreous may bring it about. Syphilis, so prominent a factor in many deep-seated eye diseases, is undoubtedly responsible for some cases, and detachment is occasionally met with in the course of other diseases, such as general dropsy, the albuminuria of pregnancy, Bright's disease, rheumatic fever, etc. The patient is usually myopic, or, at any rate, rarely hypermetropic, and the ocular tension is subnormal to begin with. The retina may be detached in two ways: first, by being pulled away from its bed by shrinking of the vitreous, the formation of false membranes or bands of new formative tissue, such as occur in hyalitis and cyclitis, or as the result of hæmorrhages in front of the retinal vessels and their occasional sequel, retinitis proliferans (Pl. 17, fig. 33); or secondly, the retina may be pushed from

its bed as the result of choroidal hæmorrhages, and the formation of exudates and lymph accumulations in albuminuric retinitis and other forms of deep-seated inflammation (Pl. 26, fig. 51).

Usually detachment takes place spontaneously and quite suddenly without warning. The patient wakes up in the morning and notices that part of his visual field is cut off, or a cloud appears in front of the objects seen. In slight cases of detachment the ophthalmoscope shows a row of faint ripples in the fundus arranged in parallel lines, somewhat resembling the appearance of a sandy beach when the tide is running out (Pl. 26, fig. 53). In more advanced cases the retina appears as a dirty-looking grey membrane ; sometimes it is nearly transparent, and in old cases white, especially on top of the folds. The retina is thrown into wave-like folds, and the vessels on it have lost their bright streak, being of a dull chocolate colour, and tortuous (Pl. 27, fig. 54). Since the retina is pushed forward into the vitreous, the detachment should first be examined with the indirect method, otherwise it might be overlooked. To see all parts of the retina the focus must be adjusted by convex lenses, which need varying for each part in turn. Sometimes the detachment is so faint that it can only be recognised at one particular angle, or it may be limited to a single narrow ridge. The tendency is for the detachment to increase both in size and prominence. It may occur at any part of the retina, but the lower half, if not detached at the time, usually becomes so at a later period by gravitation of the effused fluid. The macula is not generally involved at first, but the central vision is often obscured by a fold of the retina overlapping it. By focussing up, vitreous opacities and gauze-like membranes are often

observed. If the macula is involved, the fovea will appear bright cherry red, surrounded by a hazy whitish zone, just as in embolism (Pl. 26, fig. 52). Since the retina is firmly bound down at the ora, the retina is liable to be torn near, but not at, the periphery. This can be recognised by the whitish free border and dark background. The retina is so firmly bound down round the disc that no amount of force can separate it there.

Progress.—When detachment is due to a blow or shock, there is a reasonable chance of partial or even complete recovery, but when due to the other causes above mentioned, temporary benefit is all that can be hoped for, since relapses almost invariably occur, ending in total blindness over the detached area, but later on the detachment may spread over the entire retina. Detachment is frequently produced by exudates, serous effusion, or hæmorrhage, which push the retina forward, but a great many cases are undoubtedly due to contractions of the vitreous in places, either from the organisation of exudates, or a fibrillar thickening or condensation occurring in its structure, or else from the formation of a thin false membrane on the surface of the retina, whereby the retina is first torn, and then pushed away from its bed by the percolation of fluid underneath. This fluid accumulates behind the detachment, and one of the objects of the surgeon is to promote its escape by retinal puncture, scleral puncture, and particularly by bisection, as practised with great success by Deutschmann, so that the retina may have a chance of returning to its natural bed. Some surgeons endeavour to excite adhesions between the detached surface and the hexagonal layer, by setting up a localised inflammation by cauterisation, or by injec-

tion of Morton's fluid* or Lugol's solution, neutral saline solutions, or pilocarpine, either into Tenon's capsule or else behind the detached retina; operations, however, which are always attended with a certain amount of risk. Latterly, Deutschmann has injected sterilised rabbits' vitreous. Unfortunately, whatever treatment is carried out, the exciting cause still remains, and as that cannot be got rid of, relapses usually occur. Moreover, cataract supervenes in about 20 per cent. of all cases, so that the prognosis is very gloomy. The favourable results tabulated by some surgeons are apt to be misleading, since they are made too soon after the operation. No case should be entered as cured unless the re-attachment has lasted at least twelve months, and this one may not have the opportunity of seeing. The writer discovered the remarkable fact that the wearing of spectrum-blue glass† goggles increases the visual field, often to a surprising extent, the areas of perception extending nearly as far as the normal boundaries. He obtained an increase of field in thirty-five out of forty-two eyes so treated, but it is difficult to see how this is effected, since the detachment is never restored to its position, nor does it apparently undergo any change. Possibly the increase of the visual field may be accounted for by the accumulation of visual purple under the less active or green rays, thus allowing the enfeebled and semi-detached retina to receive impressions which it would not otherwise respond to.

* Morton's fluid consists of a solution comprising iodine gr. x, iodide of potassium gr. xx, and glycerine ℥j. It was originally employed by Morton for the treatment of spina bifida.

† This glass lets through the middle of the spectrum, rays of wave lengths longer than those at the D line being absorbed, as are those outside the H lines. The glass can be obtained from Pillischer, Ellis, Curry and Paxton, and several other opticians in London.

Hæmorrhage of the Macula Region.—Hæmorrhage confined to the macula region is fairly common. It is often due to a blow on the eye, but may be spontaneous. The extravasation oozes out beneath the hyaloid membrane, spreading into a uniform patch, which covers and surrounds the macula area, extending nearly as far as the disc (Pl. 23, fig. 45). It is usually convex below and nearly level above. The blood forms a distinct swelling, becoming darker below, where it gravitates down. The patient sees the centre of the field through a red veil. Sometimes the fovea is visible behind it. In a few days the blood begins to absorb, and the upper part changes to a slate-grey or straw-colour, while the retinal vessels begin to be seen through it. The blood may either completely absorb, leaving a normal fundus and full acuity of vision, or fatty degeneration may occur, giving rise to a large number of yellow dots, or sometimes white patches. Sometimes cholesterin crystals are deposited, or pigment patches may form, not from the effused blood, but through the pigment coming forward from proliferation of the hexagonal pigment layer. The prognosis is favourable in many cases, especially when due to *contra coup*.

Hæmorrhagic Retinitis.—Hæmorrhages of the retina occur in the course of many diseases, especially in kidney affections, but this term is especially applied to very extensive hæmorrhages spreading over the entire retina, which appear independently of kidney disease or diabetes. The hæmorrhages are usually confined to one eye. The disease, therefore, is more likely to be due to venous thrombosis or a local weakness of the vascular coats than to diseases of the blood or its vessels generally. The central visual field is greatly reduced, but, curiously, the general field is not restricted.

The hæmorrhages are for the most part flame-shaped, and therefore confined to the surface, or trickle into the vitreous. They are radially distributed along with opaque patches. There is little or no papillitis, although atrophy of the disc follows. The veins are dark, swollen, and tortuous, and often lined with white streaks. Occasionally the hæmorrhage is confined to one particular section of the retina.

Massive Exudation of the Retina.—This chronic disease was first described by Fuchs. It is characterised by the presence of a large, thick plastic exudation beneath the retina and also by the extremely dilated and tortuous condition of the vessels. It is this diseased condition of the retinal vessels which causes them to break down and give rise to a hæmorrhage or exudates between the retina and choroid. The former partly organises, thus forming the characteristic yellowish waxy exudate. Detachment and subsequent glaucoma are common. Most of the reported cases occurred in young women, and often in sisters or members of the same family. The disease appears to be unconnected with tubercle or syphilis. The disc is inflamed and swollen, but at the same time the sight may be good. Cases have been described by Griffith, Ormond, Coats, Wood, and others.

Arterio-sclerosis (Angio-sclerosis) of the Retinal Vessels.—This condition of the retinal arteries is merely a localised visible indication of a general disease. It consists of a general thickening of the walls of the blood-vessels, by which the lumen is reduced in size. It first affects the smallest terminal branches of the vessels (end arteries), such as are found in the eye, brain, and kidney. Those in the retina are, of course, the only ones we can study during life. Examined with the ophthalmoscope, we notice the following characters: (1) The fine

branches of the retinal artery are curled (cork-screwy) in places. (2) The veins are flattened where an artery crosses over them. (3) The main trunk of both artery and vein as they issue from the disc have a thick, white, double contour, with a bright light streak, which gives them a very striking appearance. One may trace this white sheath for a considerable distance along the branches (**peri-vasculitis**). A similar appearance is found in anæmia of the retina. (4) Lastly, the disc is of a deep brick-red colour. The small veins lose their middle coats, becoming mere tubes of connective tissue, and have lost all tone and resilience, resembling a punctured bicycle tube. In the very early stages the arterial tension is said to be low, but it becomes high as the disease advances.

Cause.—The chief causes of arterio-sclerosis are syphilis, auto-intoxication, gout, acute specific fevers, and lead poisoning. It appears closely connected with chronic digestive troubles.

Prognosis.—The importance of early recognising this disease lies in its serious nature. When the retinal arterial changes are well marked there is a danger of retinal hæmorrhages and apoplexy. Many cases are accompanied or followed by Bright's disease, and the disease may terminate fatally at any time.

Embolism of the Central Artery of the Retina.—In valvular disease of the heart a small clot may find its way to the central artery and plug it. This obliteration, which occurs suddenly and without warning, is made known to the patient by immediate and total loss of sight over all parts of the affected retina, except the extreme periphery, which is nourished by the chorio-capillaris and thus escapes from the consequences of cutting off the main blood supply. On ophthalmoscopic examination the fundus shows extreme anæmia. The optic nerve appears

white or very pale, the arteries are much reduced, to mere threads, their small branches being invisible. The veins are little altered except at the disc, where they taper down. After some hours the retina becomes opaque, cloudy, and creamy-white. This whiteness is most marked around the macula, which has a cherry-red colour, and is rendered

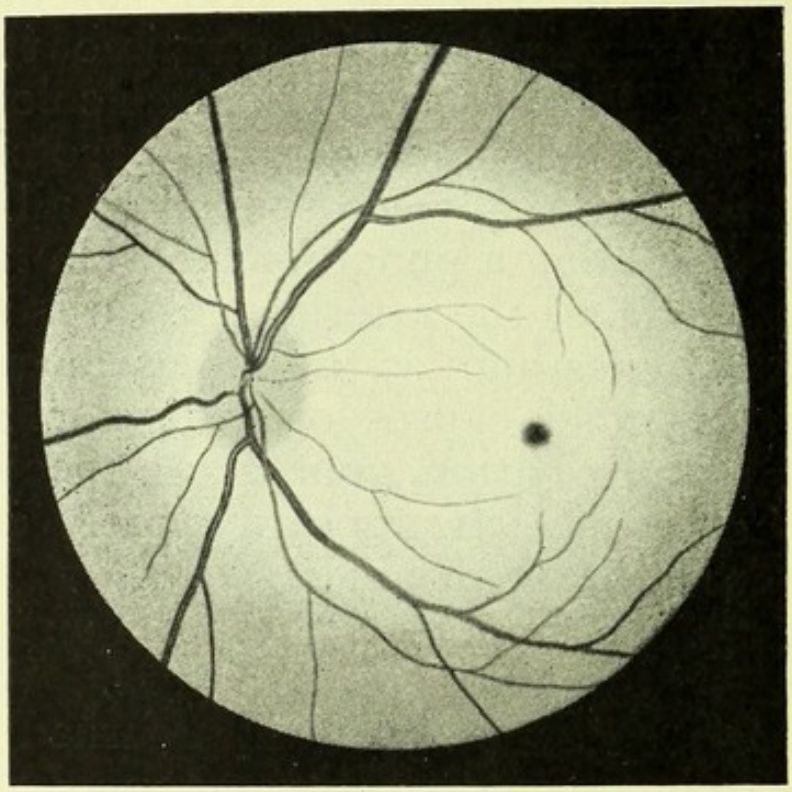

FIG. 45.—Embolism of the retina. This photograph of an actual case shows a very extensive white bloodless area embracing the disc and the macula, which appears in the photograph as a black dot. The arteries appear as fine threads in striking contrast to the large veins.

a very conspicuous object against the contrasting creamy-white background (Fig. 45). Sometimes the blood in both arteries and veins is broken up into short lengths like a column of mercury in a broken thermometer. Ultimately the internal ocular pressure squeezes this blood out, the vessels become mere threads bordered by white lines, and the papilla white and atrophic surrounded by a clean cut rim.

The retina loses its cloudiness and becomes invisible. If the clot is very small it may travel on past the central artery and plug one of the branches. In this case similar appearances will be seen, but they will be confined to the area of distribution of the branch. The perimeter shows a blind area (scotoma). Very exceptionally the clot is first lodged in the main trunk and a little later gets pushed on into one of the branches out of the main trunk, with the result that vision returns over a large part of the field. In fact one or two cases have been recorded in which the surgeon has pressed on the eyeball as hard as possible with his thumb and suddenly released the pressure, with the result that the clot has been shifted or broken up and the sight has returned. But to be successful it should be done within a few hours after the accident. Repeated massage of the eyeball has also been effective.

Thrombosis of the Central Vein is characterised by enormous increase in calibre of all the retinal veins, which are tortuous, and disappear in the retina at intervals. They pulsate on pressure. On the other hand the arteries become thready. Small venous hæmorrhages occur all over the fundus (Pl. 22, fig. 43). It is remarkable that the sight is only slightly affected at first, but gets worse with repeated hæmorrhages, which unfortunately frequently occur. The prognosis is also very bad, most of the patients dying within a year or two. Occasionally the hæmorrhages become absorbed, leaving white atrophic spots behind (Pl. 22, fig. 44).

Tay's Symmetrical Disease of the Macula—This curious and rare disease, first described by Waren Tay, is a disease of early infancy, often attacking several children in a family. The child, previously healthy, becomes lethargic, but does not suffer pain.

The muscles of the back and limbs become weak and atrophic. On ophthalmoscopic examination the macula area and some distance round it, is found to be hazy and of a pale whitish colour, the fovea appearing as a cherry-red spot in the centre (Pl. 21, fig. 41). In other words, the disease resembles that of thrombosis of the central artery excepting that it is confined to the central part of the fundus, the rest of the eye appearing normal. Gradually atrophy of the nerve sets in, and the disc becomes white and the arteries thin. Both eyes present the same appearance. The child dies from general debility within a year or two at the most. Atrophy of the ganglion cells of the retina, accompanied by degeneration of the optic nerve and of the cerebral cortex and spinal cord, are found after death, but the choroid does not appear to become affected.

Progressive Degeneration of the Macula.—This disease, like the last, is bilateral, hereditary, and affects several children in the same family. It begins by an irregular pigmentation over the macula area. Then a number of yellow or orange specks appear, which spread over the whole area, and at the same time the pigmentation increases in little clusters round and between the specks. At the fovea the pigment forms a large greyish spot surrounded by a circle of pigmentation. The pigment spreads out for some distance beyond the macula ring. Gradually the disc becomes pale, and the sight becomes reduced to counting fingers, with central scotoma. It does not appear related to syphilis. It was first described by Stargardt ('Graefe's Archiv f. Oph.,' September, 1909).

Excavation of the Macula.—This disease is generally produced by a blow on the eye—at least that was the cause in most of my patients; but it may arise from other causes, such as syphilitic retinitis,

choroiditis myopica, chlorosis and foreign bodies in the vitreous. The ophthalmoscope shows a round or irregular shaped hole occupying the centre of the macula area. It is usually from about half to a third the diameter of the disc (Pl. 26, fig. 52). The sharp edge of the retina can be clearly seen surrounding the hole, which looks as if punched out. The floor is of a deep red colour. Often the retina for some little distance around is œdematous, and more or less opaque and hazy. In some cases a retinal vessel may be seen dipping into the pit, and pigment is often scattered round the edge of the floor. Central vision is abolished over a much larger area than the hole. Along with the degeneration of the macula layers, the bundle of nerve-fibres, both afferent and efferent, which are peculiar to the macula, likewise degenerate, and this ultimately leads to a marked pallor of the temporal quadrant of the disc. This quadrant is the wedge-shaped portion of the disc which lies between the superior and inferior retino-temporal arteries, and as it contains the nerve supply of the macula, it tends to become pale and atrophic in diseases of that part. Thus one may often see the same thing in toxic amblyopia (see p. 136).

Microscopical examination shows degeneration of the ganglion cells and nerve-fibre layers from œdematous pressure, while hardly a trace of the cones remains around the hole.

Glioma of the Retina.—This is the only new growth formed by the retina. It occurs exclusively in children, mostly of the fair-haired type, and usually affects one eye only. It seems to run in families. The disease is usually advanced and the eye blind before the child is brought to the clinic, and one notices a brilliant, golden yellow reflex. On ophthal-

moscopic examination a whitish nodular mass is seen behind the lens. If seen a little later vessels will be noticed growing over the tumour, and the tension is increased, with pain. In the third stage the tumour has spread backwards along the nerve, and forwards, bursting through the coats of the eye. At this stage probably nothing can be done beyond removing the eye for relief of pain, and even if the walls of the orbit be removed, the commissure and neighbouring glands will have already become involved. The growth proceeds very slowly at first, but after a long period of stagnation it suddenly spreads with great rapidity. To save the child's life *the eye must be removed before the tumour has made its way through the globe.* The tumour starts from one of the nuclear layers of the retina, and is made up of small, round, or cylindrical epithelial cells with large nuclei, held together by a delicate tissue. Glioma may be mistaken for metastatic choroiditis (pseudo-glioma), but in the latter case there will most likely be a history of meningitis, spinal trouble, or relapsing fever. The lens will be pushed forward and the periphery of the iris drawn back by pressure of the mass behind, and there will also be signs of old iritis. The disease begins with conjunctivitis, which glioma does not, *and further, as we shall see, the tension is subnormal instead of being increased.* Extensive cyclitis may engender fibrous deposits behind the lens, which might also be mistaken for glioma.

Pseudo-glioma is an ambiguous term implying an appearance of the eye resembling a true glioma of the retina, but which, according to Treacher Collins' researches, may be applied to at least three distinct diseases, viz. :

(1) A persistent fœtal condition of the fibro-vascular sheath of the lens.

(2) A tubercular growth bulging forward from the choroid.

(3) A metastatic choroiditis accompanied by purulent exudation into the vitreous, commonly occurring in children who are suffering from meningitis.

In the first form the ophthalmoscope shows a shiny white reflex with a blood-vessel seen here and there. The hyaloid artery persists, surrounded by a thickened mass of embryonic cells. The presence of the hyaloid artery, the fact that the lens and its capsule are imperfectly developed, and the tension is normal or subnormal, distinguish it from glioma.

The second form is very difficult to diagnose from a true glioma, owing to the tension often being raised, but in practice it does not so much matter, as the eye is a source of infection both to the body generally and to the other eye, *and therefore should be excised in any case*, when, of course, the true nature of the disease can be readily identified by examination under the microscope. The retina is usually pushed forward and wrinkled, the ophthalmoscope shows a yellow reflex, and v. Pirquet's or Calmette's tests will show the characteristic reactions. On opening the eye after enucleation nearly the whole vitreous is found replaced by a soft greyish-yellow mass covered with the remains of the retina. The iris and ciliary body will be found infiltrated with small round cells. The optic nerve is injected and permeated with round and giant cells.

The third form, which accompanies cerebro-spinal meningitis, can be diagnosed by the latter disease alone. The meningitis may spread to the eye between the sheaths of the optic nerve, carrying the septic inflammatory matter into the retina, and also to the middle ear and mastoid as well. The inflammation passes right along the retina to the ora serrata,

causing proliferation of the epithelial pigment cells, which grow into the retina and soon undergo fatty or hyaline degeneration.

Speaking generally, if the anterior chamber is shallow throughout and the retinal vessels appear on the surface of the tumour, while at the same time the tension is raised, the disease is glioma.

If the chamber is deep at the periphery, owing to contraction of the exudation, and shallow at the centre, from the lens being pushed forward, and if the tension is not raised, the case is one of pseudo-glioma.

Commotio Retinæ.—This is characterised by a visible change in the fundus, accompanied by a train of symptoms which result from a violent blow on the eye, such as is caused by a champagne cork or a blow from a stick or fist. First a scleral injection is noticed. The pupil is somewhat contracted, and fails to yield either to light or atropine. This may be accompanied by a mild form of iritis. Within an hour or so after the accident a greyish, cloudy, circular patch, varying from the size of the disc to half the area of the fundus, makes its appearance. Often a number of patches appear in a cluster, separated from each other by the normal coloration of the fundus. After a day or two the greyish cloud becomes lighter, often nearly milk white. The retinal vessels pass in front of the patches. The appearances gradually vanish, until after two or three days the fundus assumes its natural colour. Vision, which at first is very defective, slowly improves, but acuity is not restored until days or weeks after the disappearance of the disease. Irregular astigmatism due to a temporary unevenness of the capsule of the lens is occasionally produced. The cause of the whitish patch is due to infiltration in the substance of, or immediately behind, the retina.

CHAPTER VIII.

DISEASES OF THE VITREOUS AND CHOROID.

Diseases of the Vitreous.—Choroiditis.—Suppurative Choroiditis.—Plastic Choroiditis.—Choroiditis Disseminata.—Tubercle of the Choroid.—Diffuse Miliary Tuberculosis.—Large Tubercular Deposits.—Macula Choroiditis.—Senile Choroiditis.—Colloid Degeneration.—Choroiditis in Myopia.—Crescents.—Rupture of the Choroid.—Detachment of the Choroid.—Sarcoma of the Choroid.—External Injuries of the Eye as a Cause of Intra-ocular Disease.

Diseases of the Vitreous.

The vitreous is a brilliant colourless jelly, identical in appearance and consistency with the white of a fresh egg. It appears to be homogeneous, but if hardened by reagents it can be shown to possess a definite framework or reticulum. Traversing its centre along the optic axis is a canal (canal of Cloquet) which can only be demonstrated in hardened specimens. The hyaloid artery and falciform ligament run along this canal during the fœtal development of the eye. These structures disappear as soon as the lens and its capsule are completely formed. The vitreous is nourished by the uvea, and especially the ciliary processes and pars ciliaris retinæ, and hence, in choroiditis, cyclitis, and retinitis, the vitreous suffers from deficient nourishment and becomes more or less turbid from the inflammatory products (blood,

serous effusion, and pigment particles) which find their way into it. These consist of opacities in the form of threads and flocculi and black pigment-spots, as well as extravasations of blood, which may become completely absorbed or organise into delicate membranes. They sometimes become detached and float about in the vitreous, which they do the more readily since the vitreous loses its consistency and degenerates into a watery fluid. More commonly the membranes are anchored to the source of inflammation and merely wave up and down in the vitreous. Sometimes serous exudates collect against the hyaloid membrane, either forming a faint, translucent membrane, which effectually prevents the details of the fundus from being seen (diffuse opacity of the vitreous), or they may collect in the form of a fine stippling either in the vitreous itself or over the capsule of the lens (vitreous dust).

When the vitreous becomes fluid, which it tends to do during the progress of high myopia with choroidal changes or after cyclitis or detachment of the retina, it shrinks, the tension is lowered, and it loses its consistency and becomes a pale yellow colour containing pigment particles. Occasionally, during fluidification, a large number of golden crystals are seen, which fall like a shower of golden dust when the patient raises his eye or winks quickly (**synchisis scintillans**). These consist of cholesterin and tyrosin particles. They are not infrequently found in elderly people with fairly good vision, and by themselves need not cause any anxiety.

Muscæ Volitantes consist of semi-transparent cells like rows of pearls, which float slowly downwards when the patient winks. They cast shadows on the retina, and are only seen by the patient when he

looks at a bright light, such as the field of a microscope or telescope. They are too small to be seen with the ophthalmoscope, and are perfectly harmless in themselves unless they become very numerous and prominent. All myopes are troubled by them if they work much at the microscope. The treatment is to persuade the patient to ignore them, to improve his health by outdoor exercise, and to avoid sedentary occupations. Dark glasses, spectrum blue, or euphos glasses are useful if he is exposed to bright light. When working with the microscope the patient should place one or more blue glass discs underneath the stage. This is advisable at all times even if no muscæ are seen.

Diseases of the Choroid.

Choroiditis may be conveniently divided into suppurative and non-suppurative or plastic forms.

Suppurative Choroiditis is a disease produced by pyogenic germs, but the fundus cannot be examined with the ophthalmoscope owing to the severity of the symptoms and the opacity of the media. The infection may either be carried to the eye from without, or arise inside the eye through a septic embolism. The disease exceeds even adult purulent ophthalmia in the violence of the onset. It sets in with œdema of the lids, excessive conjunctivitis and puffiness round the cornea. Exudates begin to pour into the vitreous. Violent cyclitis and iritis are set up. The iris becomes swollen, sodden, and synechiæ form. The aqueous becomes turbid, and a hypopyon forms in the anterior chambers. All the signs of plastic choroiditis are present. The tension rises accompanied by fever, pain, and vomiting. If the symptoms decline the eye softens, and general atrophy sets in

followed by blindness. Extreme cases assume the form of panophthalmitis. The eye then becomes a mere bag of pus, and requires immediate removal before the contents exude. Another form of this disease results from metastatic infection from cerebro-spinal meningitis, or one of the acute specific fevers (choroiditis metastatica), which leaves the eye blind, and presenting an appearance similar to that of glioma ; in fact it is often called a pseudo-glioma.

Plastic (Exudative) Choroiditis.—This form of choroiditis is as quiet and insidious in its progress as the former is violent. Unfortunately for the surgeon most forms of plastic choroiditis give rise to so few symptoms that one hardly ever sees the disease in the early stages. It is, therefore, often very difficult to diagnose the disease until it is fairly developed. The typical form of plastic choroiditis is choroiditis disseminata.

Choroiditis Disseminata.—The first indications are the formation of isolated patches or spots of yellow-looking exudates which permeate the choroidal tissue beneath the retinal vessels, and which may become slightly cloudy with a grey veil (Pl. 18, figs. 35 and 36). These clusters of inflammatory exudation stop the nourishment of the tissues in contact, and as they become re-absorbed they leave behind a bleached spot which continues to atrophy until nothing remains but a sharply defined ring of pigment surrounding the white sclera, which is seen shining through. This pigment continues to proliferate for some time afterwards. The retina also atrophies over these exudates. These form small scotomata or blanks in the visual field. Unless they are large or situated at the macula the patient is unaware of them, but during the exudative stage the inflammatory products filter into the vitreous, producing cloudiness with great

deterioration of vision. If the exudation takes place at the macula, and is only slight, so as not to destroy the retinal cones, distortion of vision may occur (metamorphopsia) in which portions of the objects seen appear larger or smaller than they should do. The macula is unfortunately a favourite seat of these exudates, and when they cluster thickly they may fuse together, giving rise to a large oval gap which forms one variety of macula coloboma (Pls. 7 and 8, figs. 13 to 16). Much pigment is prolificated round the margins, and networks of new-formed vessels often spread inside the gap, lying over the broad orange choroidal vessels, which may be seen stretching across it (see Pl. 8, figs. 15 and 16). If the gap includes the macula, central vision is of course lost, but I have seen several cases where the gap was quite close to the macula without affecting central vision at all, although the diseased part occupied a larger area than the disc. This disease is generally traced to syphilis, either acquired or congenital, usually the latter. The small isolated crater-like spots are mostly found in the latter form. They vary from one fifth to one third the diameter of the disc. In many cases of choroiditis disseminata the fundus appears quite normal, but careful examination will always reveal two or three crater-like atrophic spots surrounded by the characteristic pigment-ring in some part of the fundus. The sight may be normal, but is usually defective and due to changes which can rarely be seen with the ophthalmoscope. Old spots are indicated by the complete atrophy of the choroid, showing the chalky white sclera inside the ring, by their sharp outline and dense black pigment. Often they herd together, fusing into irregular patches loaded with pigment (Pl. 19, figs. 37 and 38). Recent spots show merely dirty, yellowish areas of exudate,

free from pigment, and without any sharp demarcation. In the final stage, which is quite uncommon, general retinal degeneration occurs, the retina becoming in places converted into fibrous tissue over the involved areas with diffuse pigmentation.

Tuberculosis of the Choroid.—The eye is now known to be far more commonly the seat of tubercle than was thought to be the case even a few years back. This is due partly to improved diagnosis, many appearances, as, for example, some forms of choroiditis disseminata, being now proved to be tubercular although unrecognised as such formerly, and partly to our being able to recognise tubercle by means of v. Pirquet's test and the opsonic index. These tests are highly important, since there is no certain way of distinguishing tubercles of the choroid from syphilitic deposits by means of the ophthalmoscope, while the prognosis as regards life is widely different in the two diseases. The two chief forms are—(1) diffuse miliary tuberculosis, and (2) large tubercular deposits of the choroid. Tubercle of the retina is exceedingly rare; when seen it is nearly always due to an extension of the disease from the choroid.*

Multiple or Diffuse Miliary Tuberculosis.—Tubercle deposits may be found in the choroid in about a third of all cases of miliary tuberculosis and tubercular meningitis. They most commonly occur in children, and are often confined to one eye. One notices a number of white or yellowish elevated specks round the macula area or near the disc, and often with a pinkish indistinct border (Pl. 24, figs. 47 and 48). The deposits arise in the choroid and make their way to the front through the hexagonal layer. They

* See 'British Journal of Children's Diseases,' June, 1906. (Adlard & Son.)

may be diagnosed from the syphilitic form by the absence of pigment, ill-defined border, and general dull colour. Sometimes we see them accompanied by optic neuritis, when the sight will be affected, otherwise the sight is fairly normal. The presence of tubercular spots or patches is an extremely unfavourable sign, as they invariably indicate general tuberculosis and point to a rapidly fatal termination.

Large Tubercular Deposits.—These assume various types. One type occurs in childhood in the form of a solid, oval, or irregular-shaped, whitish-yellow or fawn-coloured growth, which is seen in the fundus, usually not far from the macula, and often appears dusted over with minute pigment granules. It obviously lies behind the retinal vessels. Its origin and mode of growth is similar to the miliary tubercle, and in some cases it is undoubtedly due to a confluence of small tubercles, but when once formed they do not grow any larger. In these cases isolated miliary tubercles will be found in the neighbourhood. One may occasionally meet with a single tubercular deposit at or near the macula, nearly as large, or even larger, than the disc. Sometimes these formations spread backwards through the sclerotic or forwards through the retina. In either case they break down and undergo caseous degeneration. Miliary tubercles will generally be found elsewhere in the brain membranes, lungs, or abdomen, and signs of either dulness over the lungs, drowsiness, or abdominal pains are often present. In the most favourable cases the deposit is thin and undergoes absorption with atrophy of the contiguous retina and choroid, leaving a white base which may be partly covered with cicatricial tissue, or partial atrophy of the choroid occurs, with the result that the base is pinkish and shows a sparse network of

choroidal vessels. Another variety met with consists of a huge yellowish-white deposit spreading over a large part of the fundus, and lying behind the retinal vessels. The margin of the deposit is either broken up into innumerable islands of the same material, or the patch may be surrounded by small miliary tubercles which appear in the fundus like pinheads. The ends of the fine vessels become lost to view in small hæmorrhages. The retina is often detached and pushed forward by the exudates. In all cases the diagnosis should be confirmed by v. Pirquet's reaction.

Choroiditis of the Macula.—Large gaps in the macula region are not so rare as was formerly thought to be the case. Many cases which have been diagnosed as coloboma* have ultimately turned out to be due to central choroiditis of either tubercular or syphilitic origin, or occurring in young women as the result of menstrual irregularities. In one form the choroid becomes thinned over a circular or oval area as large as or larger than the disc (Pl. 8, figs. 15 and 16). A few of the choroidal vessels may be seen stretching across the patch. Some of these cases are undoubtedly due to an old strictly local and quiescent tuberculous patch or node, as has been recently demonstrated by Butler.† In the next variety the large oval patch shows complete atrophy of the choroid, with a sharp overhanging edge, as if the area had been punched out (Pl. 7, figs. 13 and 14). A rich growth of pigment surrounds the patch for some distance, enclosing here and there smaller atrophic spots in its meshes. This form may be congenital, or due to intra-uterine inflammation, and is often met with in eyes which

* See "Extra-papillary Colobomata," 'Knapp's Archives,' 1890.
† See his paper in the 'Ophthalmoscope,' June, 1910.

have been arrested in development (microphthalmus), but a similar disease may be seen in adults and old people, and the process of breaking down of the choroid observed. Occasionally smaller circular patches are formed at the macula, which are arranged like a pavement of cobblestones; we find it in choroiditis disseminata. It is probably due to congenital syphilis, and may be seen even in early childhood. Some of these central atrophic patches are undoubtedly true colobomas, since the same eye shows both traces of the original choroidal cleft and coloboma of the iris as well. Others, again, show a nævoid-like entanglement of the choroidal vessels. Tubercles at the macula are not rare. Syphilitic deposits (gummata ?) at the macula have occasionally been seen. They disappear readily under large doses of iodide of potassium.

Senile Choroiditis is a general term for several distinct forms of disease. In one form an irregular-shaped or circular patch slowly forms at the macula of each eye, accompanied by thickening of the sheaths of the vessels and local pigmentation (choroiditis guttata) (Pl. 20, fig. 40). There is a central scotoma, but as the disease does not spread peripheral sight remains. Another form is characterised by partial atrophy of the hexagonal layer, which causes the choroidal vessels to show up very clearly. The pigment generally is displaced and scattered irregularly through the retina. This disturbance of pigment always reduces acuity, and is generally accompanied by some night-blindness. It affects both eyes. Lastly, peculiar bunches of small, round, greyish-white bodies may be sometimes found on or round the disc in old people. They look exactly like the spawn of a fish grouped together in bunches. These bodies are probably due to a **colloid degenera-**

tion of the lamina vitrea of the choroid. The disease does not *per se* affect the sight.*

Choroiditis preceding and accompanying Myopia.—Myopia is essentially caused by a stretching of the coats of the eye, due in some cases, at any rate, to a very chronic inflammatory process. This stretching produces an axial elongation, as well as a slight general enlargement of the globe. Although the tendency to myopia is hereditary,† children are very rarely, if ever, born myopic. There is, however, a close connection between choroiditis and myopia. Although the vision may be normal, or even supernormal, when corrected with glasses, a myopic eye can never be regarded as a perfectly healthy organ, and requires constant medical supervision, especially during childhood and youth. During the stretching of the globe, which invariably takes place in progressive myopia, the weakest spots naturally suffer most. These are—first, the tissues round the sheath of the optic nerve at the papilla; secondly, the macula region, and the area between the two; and lastly, the ciliary region between the ora serrata and the sclero-corneal junction. All three coats at these parts tend to yield, the two first named as the result of congenital weakness or want of resistance of the tissues, the latter as the result of chronic inflammatory changes, both of which diminish the elasticity of the coats and their resistance to internal pressure, causing a slight bulging backwards (staphyloma posticum). During the stretching along the optic axis, resistance is encountered at the

* For characteristic illustrations of this disease see 'Arch. of Ophth.,' vol. xviii, Part II.

† This statement must not be taken to mean that myopia can be acquired and then transmitted to future generations, a fact which, although commonly stated, has never been satisfactorily proved to be the case.

places where the choroid and retina are attached. These are the ora serrata and the papillary ring. The strain pulls the head of the nerve outward and a little downwards towards the posterior pole, causing the choroid to yield at its attachment round the nerve sheath, so that it forms a crescent-shaped patch of thinned atrophic choroid outside the outer margin of the disc (myopic crescent) (Pl. 23, fig. 46). The lymph space between the sheaths of the nerve also stretches, and gets pulled aside, giving rise to a local widening of the white crescentic border of the disc (scleral ring). As the stretching proceeds the choroid atrophies until its vessels become clearly defined, and appear as if they spread over part of the disc. Then a second wider crescent forms on the outer side of the first, and at the same time the first crescent spreads further and further round the disc, until the latter is completely surrounded by a ring of atrophied choroid. Sometimes the border of the disc becomes lost in this ring, especially on the outer side, so that, to a beginner, the disc appears very much larger than it really is. A more careful examination will, however, readily reveal its limits. The disc, being tilted, appears foreshortened laterally, which foreshortening is intensified by the fact that the sclerotic and choroid around the inner margin are pulled up to its edge. Often these whitish or pink crescents have a greyish tinge from disturbance of the pigment, or are pigmented, especially along the atrophied margin. The depression, due to the bulging, can be seen by the parallactic movements, and also by the change of lenses needed to bring the different parts into focus. This posterior staphyloma always gives rise to a variable amount of irregular astigmatism, which may be roughly measured by direct ophthalmoscopy.

The myopic crescents are usually directed obliquely outwards from the disc towards the fovea. A downward crescent (Pl. 7, fig. 14) or a large atrophic ring round the disc denotes a congenital origin, and an arrest in the closing of the fœtal cleft. The absence of a crescent in myopia is a very favourable sign, and indicates that the myopia is not of a progressive nature.

In high degrees of myopia tiny bubbles or droplets are apt to form behind the anterior capsule of the lens, followed by slight opacities, which, in some cases, terminate in true cataract (Hirschberg).

Macula Changes.—These only occur in high myopia, and indicate the so-called "**malignant myopia.**" The macula appears congested. Here and there the choroid atrophies, showing whitish, irregular patches, which tend to become confluent. Soon slight hæmorrhages appear, leaving pigmented patches after their absorption, with loss of central vision. Often patches of atrophic choroid appear between the disc and the fovea, and occasionally in other parts of the fundus. In this condition the vitreous becomes slightly turbid, with numerous floating opacities. The tension is then found to be subnormal, and there is great danger of retinal detachment supervening. In addition to the above changes a peculiar inky-black spot, about a quarter the diameter of the disc, sometimes develops in the macula region or fovea. It is accompanied by a corresponding central scotoma.

All chronic forms of choroiditis, and especially the form just discussed, require careful hygienic treatment, so as to place the system in the most favourable condition for building up the health. The bowels and digestion require constant attention; reading and indoor occupations should be, as far as

possible, avoided. The myopia should be either considerably under-corrected or two pairs of glasses worn, one for reading and one for distance, so as to prevent the tension on the choroid and retina which the act of accommodation produces. The astigmatism must be very perfectly corrected. In schools the desks and forms, and especially the lighting of the rooms, require great attention, so as to prevent all inclination on the part of the scholars to stoop over their work.

Rupture of the Choroid is due to some local injury. Blood is invariably extravasated and generally pours into the vitreous and obscures the view. After absorption has taken place an irregular yellowish streak or band is seen at the seat of injury. Usually the lesion is curved, and extends for a considerable distance across the fundus (Pl. 25, fig. 50). Later on the stripe appears white from the sclerotic shining through, and abundant pigment will be seen scattered along the edges. Its position can be seen by the retinal vessels, which pass uninterruptedly over the lesion, unless the retina is torn with it.

Detachment of the Choroid.—This is very rarely seen during life, but is often met with in old, shrunken eyes when cut open. It may be detached by an effusion of blood underneath the choroid after a blow or hæmorrhagic choroiditis. The ophthalmoscope may reveal a dark circumscribed swelling in the fundus, while the retinal vessels will appear unaltered in colour. But the diagnosis is extremely difficult, since the media are almost certain to be cloudy, and often the retina is detached as well. It may also be due to a sarcoma pushing the retina and choroid in front of it. In any case some considerable time must elapse before any opinion can be arrived at as to its nature.

Sarcoma of the Choroid.—A tumour behind the retina is almost certain to be a melanotic sarcoma. About one in ten cases will be found to be non-pigmented, *i.e.* the so-called white sarcoma. The chief thing is to arrive at an early diagnosis, as delay in removal of the eye increases the risk to the patient's life. It arises spontaneously, without any cause, in a person otherwise healthy, and usually over thirty-five years of age. At the commencement of the disease the tension is normal, and the only symptoms are a slight blurring of vision accompanied by a scotoma or slightly restricted field. On ophthalmoscopic examination a slight shallow detachment of the retina, usually in the lower half of the eye, or else a hæmorrhage will be noticed, or a bluish-grey retinal patch will be seen in the fundus, which may present neither ripples nor folds. Gradually the sight fails. The retina becomes pushed forwards and the detachment increases until it may even become total. But as a rule the growth is very slow at the commencement, and one, or even two, years may elapse before the detachment becomes markedly larger. As soon as this takes place the eye undergoes a rapid change. All vision is extinguished, the tension increases, the media becomes clouded, and the lens cataractous, while the eye becomes inflamed and painful. If the eye is allowed to remain the growth makes its way through the coats and the eye protrudes. The tumour then rapidly fills the socket, and secondary growths are formed in other parts, especially in the brain and liver, with speedy fatal results. Unfortunately the diagnosis in the early stages is often difficult if not impossible, since it may be confounded with simple detachment, or detachment due to subretinal hæmorrhage or effusion. If, however, *a circumscribed, smooth, rounded swelling be*

seen with the ophthalmoscope with the retina adherent, which continues slowly to increase in size, it is almost certain to be a sarcoma, and this is confirmed if glaucomatous symptoms supervene. If the swelling remains stationary and the tension normal, an exploratory puncture with a sterilised, grooved needle should, if possible, be made through the sclerotic behind the upper border of the protuberance (since the detachment is always on the lower side of the tumour). This will decide whether it is a simple subretinal effusion or not. In any case, if glaucomatous symptoms arise, the eye had better be removed at once as it will be useless for vision, while the chances of metastatic growths ensuing will have been greatly reduced by enucleation should it turn out to be a sarcoma. Of late years the diagnosis of orbital sarcomata has been facilitated by the use of transillumination instruments (diaphanoscopy), especially in cases in which the media prevented the ophthalmoscope from being used. A number of types of this instrument are made, among which Meyrowitz's, Würdemann's, Lange's, and Sachs' seem to embody the latest improvements. They consist essentially of a metal cylinder or box containing a powerful glow-lamp, and sometimes kept cool by means of a water-jacket. The end of the instrument, which transmits the beam of light, is applied directly to the scleral conjunctiva or the upper or lower lid after applying a solution of cocaine. The observer makes the examination in front of the patient in a dark room, and gazes into the pupil, which has been previously dilated. The instrument is especially useful to obtain information of the ciliary region and peripheral parts of the fundus where the ophthalmoscope will not reach. Foreign bodies in the vitreous can sometimes be readily seen by this method. If the water-cooled lamp be used it may

be placed inside the patient's mouth in a dark room, and the frontal, ethmoidal, and maxillary cavities illuminated. This is especially useful in cases where a tumour or pus is suspected to exist, and to directly or indirectly affect the globe or optic nerve. We owe the use of this valuable method in this country largely to Stephenson.

External Injuries to the Eye as a Cause of Intra-ocular Disease.—The question put by Perlia in 1905, "Can an injury to the eye start a constitutional infection which has hitherto lain dormant in the system in the same eye?" has been exhaustively answered in the affirmative by the remarkable researches of Birkhaüser in Siegrist's Klinik, who has shown that *any* injury to the eye, whether from a blunt instrument or the knuckles, or even from a blow produced by a handful of sand, insufficient in itself to cause any rupture of the parts, will sometimes be followed after a few days or weeks by an outbreak of tubercles or tubercular growth in the iris, ciliary body, or choroid in a strumous or tuberculous child, or by an interstitial keratitis in a patient suffering from latent congenital syphilis. He has further shown that a number of diseases originating in or carried to one eye may pass along to the other eye, either by the lymph-channels or through vaso-motor influence, in the same way as has long been known to be the case in sympathetic ophthalmitis. Further, he has shown that a non-penetrating injury to the eye may prove the immediate exciting cause of a number of diseases of the deeper parts of the globe in patients who are not tainted with any hereditary disease. For example, a patient who a few weeks previously had contracted syphilis, got some particles of cigar ash into one of the eyes. This was followed next day by

a violent attack of iridocyclitis accompanied by exudates, which caused adhesions between the iris and the lens capsule, together with intense ciliary injection. The opposite eye remained healthy from the commencement. This shows how secondary lesions may break out in distant parts of the body by slightly reducing the resisting power of that part. It is evident that the above facts should render the medical adviser doubly careful in giving an opinion as to the course and prognosis of any injury to the eye, however slight, when the patient happens to be affected with either a tubercular or a syphilitic taint.

APPENDIX.

Purkinje-Sanson Phenomenon.—Localisation of Opacities.—Definition of Tension.—Wassermann's Serum Reaction Test for Syphilis.—Determination of the Opsonic Index.—von Pirquet's Skin Reaction Test.—Calmette's Conjunctival Reaction Test.—Ehrlich's Specific Remedy for Syphilis.

To Ascertain if the Lens is Present.

This is done by means of *the Purkinje-Sanson images*. Hold a candle in the dark room a little in front and to one side of the eye, and on looking closely two and sometimes three images will be seen. First, an erect bright image reflected from the front surface of the cornea; second, a faint, often imperceptible image, rather larger than the first one and reflected from the front of the lens; and third, an inverted image, bright, but very small, which is reflected from the back of the lens. This last image can always be seen, and on moving the candle from side to side it travels in the reverse direction. This shows at once whether the lens has been removed or not, a fact not otherwise so easily ascertained as one would imagine.

To Ascertain on which Side of the Lens an Opacity Exists.

First see whether it is fixed or movable. If the former, it is probably in the front or posterior

capsule. If movable it is in the vitreous. It is often a little difficult to be certain on which side of the lens the opacity lies. Test by parallactic method. Illuminate the opacity with a lens and observe its position when looking directly through the centre of the pupil. Now move your head sideways and observe its displacement relative to the centre of the pupil. If it is not displaced at all, it lies on the anterior capsule. If it is displaced in the opposite direction, it lies in or on the cornea. If in the same direction, it is situated behind the anterior capsule at a distance proportional to the amount of displacement. Another way is to focus the ophthalmoscope on to the pupillary edge of the iris; if without shifting the instrument the opacity is sharply in focus, the opacity lies in or on the anterior capsule. If the opacity is out of focus, reduce the power of the lens behind the mirror until it is in focus. This will give roughly its position. But it is often extremely difficult to focus it up, and both the ophthalmoscope and the oblique light methods should be used.

Tension of the Globe.

Always commence the examination of the eyes, no matter what the disease may be, by ascertaining the tension of each one. This is done by palpating the globe over the upper closed lid, pressing alternately with the forefinger of each hand. The scale of tension is reckoned as follows:

T + 3. Stony hardness. It generally indicates a lost eye, and only an immediate operation can save the sight under any circumstances.

T + 2. Very hard, but a slight indication of yielding may be felt. It always indicates a serious condition

as regards future vision, and requires immediate surgical interference.

T + 1. Distinctly harder than a normal eye.

T + ½. Just perceptibly harder than a normal eye.

T − ½. Just perceptibly softer than a normal eye.

T − 1. Distinctly softer than normal.

T − 2. A soft eye. Vitreous undergoing degenerative changes. Sight no longer normal. Floating opacities usually seen in the vitreous. The patient should be guarded against liability to detachment of the retina.

T − 3. A very soft eye. Resembles a punctured tyre. Vision very defective or *nil*. Indicates a fluid vitreous containing many floating opacities, the result of old hyalitis or cyclitis. It is frequently accompanied or followed by detachment of the retina.

The internal pressure of the healthy eye as represented by the tension is equal to a column of mercury 1 inch in height.

Wassermann's Serum Reaction Test for Syphilis (Fleming's Modification).

Prepare: (1) An alcholic extract of heart muscle. Grind up one part of sheep's, rabbit's, or guinea pig's heart muscle with five parts of absolute alcohol. Digest for one hour at 60° C. Allow to stand for a short time till the supernatant fluid becomes clear, then pour off this and keep it as stock. Before use dilute this to 10 per cent. with saline solution (1 per cent.).

(2) Wash sheep's corpuscles by repeated centrifugalisation, and dilute to 10 per cent. with saline solution (1 per cent.).

(3) Draw off the patient's blood in an ordinary blood capsule, such as is used for an agglutination

or opsonic test, and allow to stand for a short time till the contraction of the clot separates the serum.

Take two pipettes, one marked at 10 cubic mm. and the other at 40 c.mm., and with these pipettes make the following mixtures:

(1)	Heart extract	.	40 c.mm.					
(2)	„	„	.	„	and	patient's	serum	10 c.mm.
(3)	„	„	.	„	„	normal	„	„
(4)	Normal saline	.	„	„	normal		„	„
(5)	„	„	.	„	„	patient's	„	„

Incubate these for one hour at 37° C., then add 10 c.mm. of 10 per cent. sheep's corpuscles in saline solution to each of the tubes, and continue the incubation for another hour. If the patient has syphilis there should be no hæmolysis in tubes 1 and 2, but hæmolysis in 3, 4, and 5.

By hæmolysis is meant the escape of the hæmoglobin contained in the blood-corpuscle, thereby colouring the liquid.

Explanation of the Test. — Hæmolysis of the sheep's red blood-corpuscles is effected by the combined action of two bodies—complement and amboceptor—which are normally present in fresh human serum. Neither of these two bodies is, however, capable of causing hæmolysis in the absence of the other. In addition to the hæmolytic amboceptor there is present in the syphilitic serum a syphilitic amboceptor, which, in the presence of a heart extract, has the property of firmly joining up this last with the complement (so as to form a united body) during the first incubation period, thereby preventing the complement acting in conjunction with the hæmolytic amboceptor when the sheep's corpuscles are subsequently added. In the normal serum no such syphilitic amboceptor is present, so that the complement is not bound up, and is there-

fore free to act on the blood-corpuscles and dissolve out the hæmoglobin.

Determination of the Opsonic Index (Wright and Douglas method).

Wash some normal human blood-corpuscles, free from serum, as follows: Dilute the blood about ten times with citrate of soda solution (1·5 per cent.) to prevent coagulation, centrifuge it and remove the supernatant fluid, replacing it with normal salt solution. Shake gently and again centrifuge it. Make an even emulsion of organisms by drawing up and ejecting with a bulb capillary pipette several times an emulsion made by putting a small loopful of organisms from an agar culture into about half a cubic centimetre of normal saline.

Obtain the patient's and normal blood in ordinary blood capsules and allow to clot, then separate the serum.

Take a straight capillary pipette, make a mark on the stem about half an inch from the end, and with the aid of a rubber teat take up equal volumes of the washed corpuscles, bacterial emulsion, and patient's serum. Mix these on a slide, draw the mixture up into the pipette, seal the end in the flame, and put in the incubator at 37° C. for fifteen minutes. Repeat the performance, substituting normal serum for that of the patient. After incubating for the requisite time, make films of the contents of the tubes on slides. Fix by passing the slide through a Bunsen or spirit flame, stain by the ordinary methods for the organism in question (formol fuchsin is a useful one), and count the number of organisms which have been taken up in the first 100 leucocytes (phagocyte cells) on each slide. The opsonic index is the figure

obtained by dividing the number of organisms found ingested when the patient's serum was used by the number obtained when normal serum was used. Thus, if the number of organisms in 100 of the patient's leucocytes was 560, and that of normal leucocytes was 600, his opsonic index would be 0·9.

V. Pirquet's Tubercular Skin Reaction.

Scrape off a small area of epidermis on the inner surface of the arm with a sterilised lancet or cutting needle, so as to expose the moist cutis vera. Rub in a drop of tubercular solution (Koch's Alt tuberculin ; Hoechst preparation will do) with the blunt head of a sterilised needle or probe. Make a control on another raw surface with glycerine and water. If tuberculous disease is present, a pink areola will form round the first inoculation at the end of forty-eight hours, which is followed after twenty-four hours more by a livid papule. This should last at least seven days. The control should show no reaction whatever. The severity of the reaction is in inverse ratio to the severity of the disease (McNeil). This reaction is considered more reliable than Calmette's.

Calmette's Conjunctival Reaction.

This consists in instilling two drops of ·5 per cent. or 1 per cent. solution of dried tuberculin in distilled water inside the lower lid. Reaction if tuberculosis exists should set in about six or eight hours afterwards, accompanied by conjunctivitis, slight photophobia, lacrimation, hyperæmia, and thickening of the conjunctiva of the lower lid,

plica, and caruncle, and occasionally slight muco-purulent discharge. The inflammation continues from three days to a fortnight. A good many cases of severe conjunctivitis, keratitis, and ulceration of the cornea have been reported recently, so that v. Pirquet's test, which cannot affect the eye injuriously, is much to be preferred.

Prof. Ehrlich's Specific for Syphilis.

Quite recently (at the last Medical Congress) Paul Ehrlich reported a remarkable specific remedy for the active (contagious) stages of syphilis. It consists of a complicated compound of arsenic, Dioxydiamido-diarsenobenzol, but better known by the easily remembered figure No. 606, or by its German name, Ehrlich-Hata Mittel. By means of experiments on guinea-pigs and apes the exact dose for patients has been arrived at. The fluid for injection is prepared as follows: Half a gramme of the arsenic compound, which is sent out in the form of a yellowish powder in sealed tubes, is placed in a sterilised mortar and rubbed up with 0·33 gr. of caustic soda, and then 10 c.c. of distilled water is added and carefully neutralised with a drop or two of acetic acid. The whole is then drawn up into a syringe and injected in a direction from above downwards deep into the subcutaneous cellular tissue between the patient's shoulder-blades. This is the entire treatment. No further injection is needed. As a matter of precaution the patient is kept in bed until the fever has subsided. Very much larger doses have been administered into apes previously inoculated with syphilitic virus, with perfect safety. It has been found that a single injection in man, guinea-pig, or ape will *at once* kill all the spirochætæ in the system, and the lesions

will commence healing up rapidly wherever they may be situated. Further, Wassermann's serum reaction, which previously gave a positive reaction (*i.e.* no signs of hæmolysis), from that time showed a negative reaction (*i.e.* more or less complete hæmolysis), while the body-weight increased week by week. The injection is rather painful but not invariably so, and the patient becomes feverish. The pain and fever last about three days, after which they both rapidly subside. As a rule the injection is unattended with danger. Only in the case of infants infected with congenital syphilis has it as yet proved fatal, and that not from the drug itself, but from the fact that their livers were so loaded with spirochætes that these organs were unable to eliminate the dead bodies even with the help of the leucocytes. From all accounts it had even less objectionable features than mercury, and as far as the writer can ascertain, no case of atrophy of the optic or other nerve has occurred as has happened with atoxyl and other drugs. It seems to be especially valuable in those severe cases which will not tolerate mercury in any form. The drug will not be placed on the market until repeated trials have placed its efficacy and harmlessness beyond all doubt, but it may reasonably be expected to be obtainable early in the coming year, 1911.

INDEX

ADLARD AND SON, IMPR., LONDON AND DORKING.

PLATE I.

FIG. 1.—NORMAL FUNDUS OF A LIGHT-HAIRED EUROPEAN. RIGHT EYE.

This picture represents a very characteristic orange-red fundus. The scarlet arteries always appear lighter than the crimson veins. The macula may be identified by its darker red colour. In the centre the fovea centralis is seen as a light spot. The disc has a healthy pink colour with a moderately deep white central excavation (physiological cup). The clearly defined edge of the disc is striated all round with semi-translucent fibres. As the subject was an emmetrope over twenty-five years of age the macula ring is not in evidence. (See Chapter III.)

FIG. 2.—FUNDUS OF AN ALBINOTIC EYE. ERECT IMAGE. LEFT EYE.

The general background is white or a pale pinkish-orange, the white colour being due to the sclerotic shining through. The choroidal vessels abound all over the field. They are a bright orange colour, and are especially fine and numerous over the macula area, and for a considerable distance around it they form a dark red mass. The optic disc will also be seen to be much darker than normal, which may be partly due to contrast with the pale background. The retinal vessels may be traced from its centre, and lie in front of the choroidal vessels. The branches of the retinal vessels are few in number, whereas the choroidal branches are exceedingly numerous, and divide into leashes of vessels which pursue a nearly parallel course, and are nearly all of the same size. A second group of vessels, larger and paler, can be seen behind the chorio-capillaris layer. They belong to the layers of Haller and Sattler. (See Chapter III.)

PLATE I.

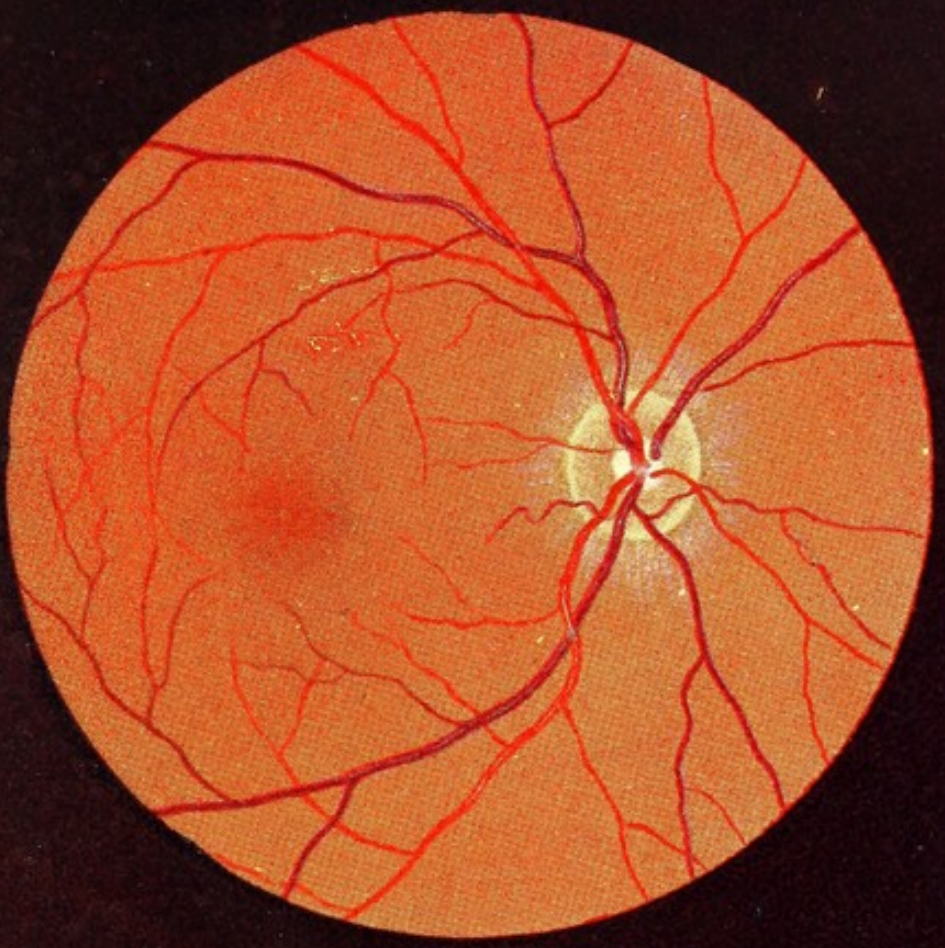

Fig. 1.

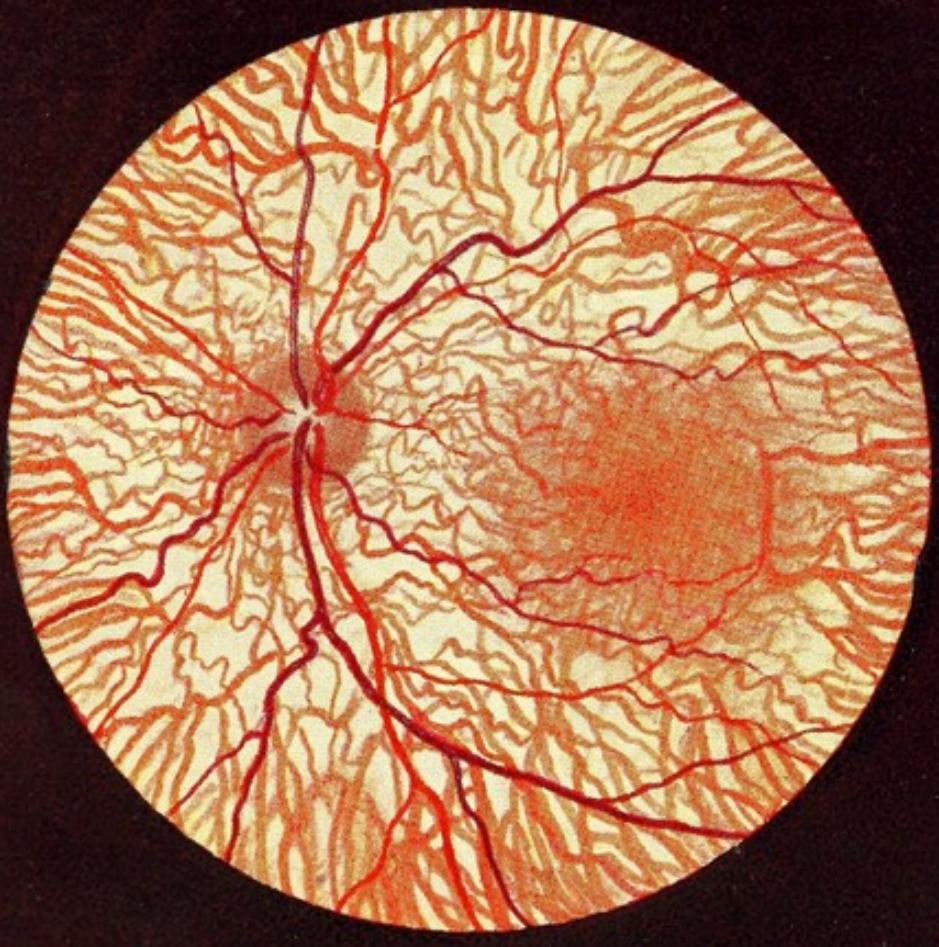

Fig. 2.

A. W. Head.

PLATE 2.

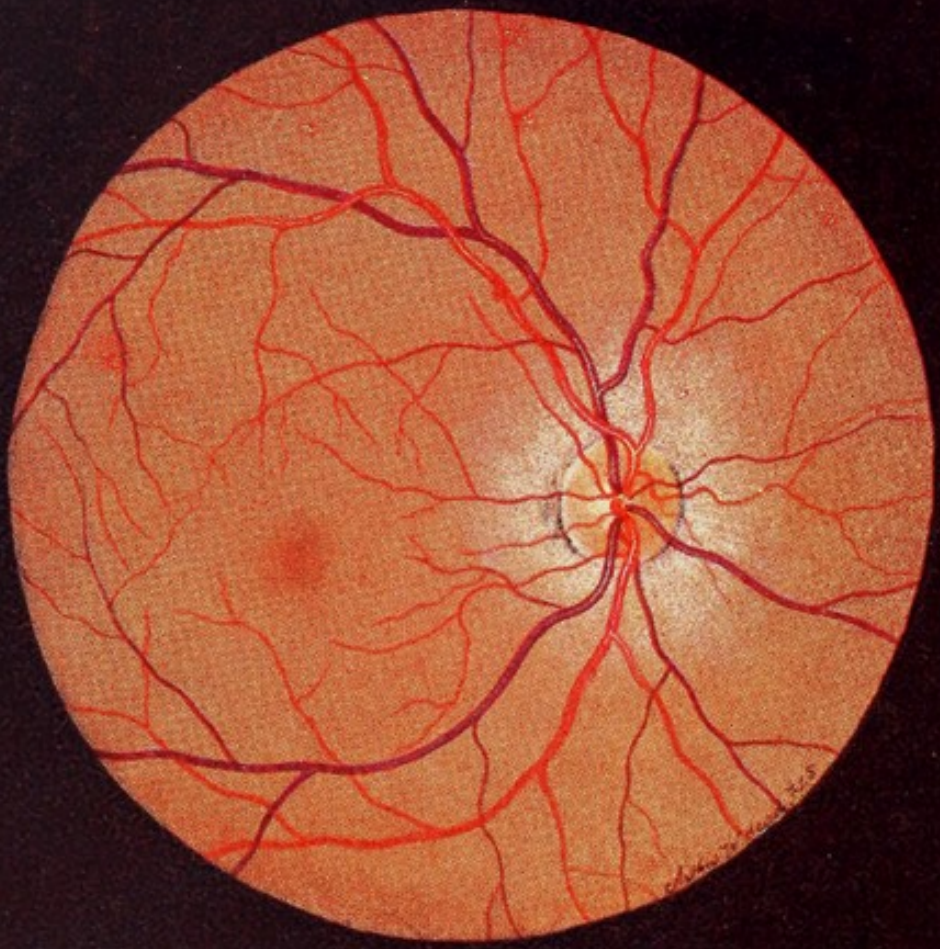

Fig. 3.

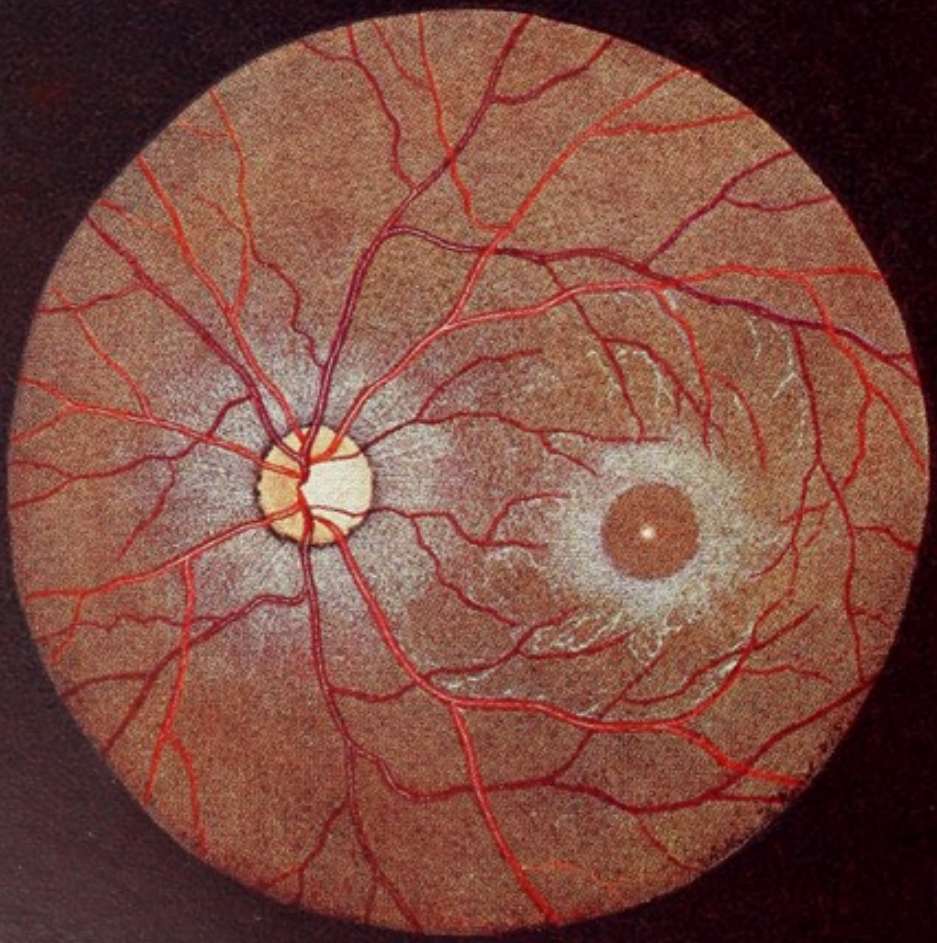

Fig. 4.

A. W. Head.

PLATE 2.

FIG. 3.—NORMAL FUNDUS OF A SPANISH GIRL WITH BLACK HAIR AND OLIVE COMPLEXION. RIGHT EYE.

The colour of the fundus is intermediate between that of Fig. 1 and Fig. 4. A fine pigmentary mottling may be noticed over the entire fundus. This is due to the excess of pigment in the hexagonal layer, each minute dot representing a single hexagonal cell. These dots are very fine, and seen by the erect method resemble tiny specks of coal-dust scattered all over the background. The optic disc is a uniform bright orange-pink colour, bordered with black pigment and striated all round. The macula area is very dark red. Fovea not visible; distribution of the retinal vessels typically normal.

FIG. 4.—DO. OF A CONGO NATIVE BOY, SHOWING RETINAL REFLEX. LEFT EYE.

The eyeground is a reddish-chocolate colour. The entire field glistened with reflexes, which were especially evident around the macula and the vessels which were directed towards it. This sheen is very difficult to reproduce by lithography, and in nature has the appearance of a torn spider's web, wet with dew, glistening in the sunshine. The fovea centralis is seen in the centre of the macula ring as a pale yellowish round dot. The optic disc is bordered round its edge with dense black pigment, and shows a typical cup on the outer side pitted with grey dots due to the tissue of the lamina cribrosa showing through. The inner or nasal side of the disc is a bright orange-red; the nerve-fibres seen radiating round the disc and macula are semi-translucent and typical of all very dark fundi. The vision was supernormally acute, $= \frac{6}{1 \cdot 5}$, or the fourth line of Snellen's type below the normal $\frac{6}{6}$. He could see three of Jupiter's moons with the naked eye, as evinced by his marking their position on paper, which was confirmed by a field-glass, and could spell correctly the words of the leading article of the 'Times' at ten feet (3 metres). (See Chapter III.)

PLATE 3.

FIG. 5.—LEFT EYE, SHOWING TRACES OF THE FALCIFORM LIGAMENT OR SHEATH, WHICH EXTENDS FROM THE DISC TO THE POSTERIOR CAPSULE OF THE LENS.

The colour of the optic disc is a typical red, and shows very distinctly the pale sclerotic ring around its border. On the temporal side are several patches of black pigment, and arising from the centre of the disc is a long white ligamentous cord, consisting of connective tissue which projects forward through the vitreous, and will be seen in the drawing extending forwards towards the lens, where it tapers to a very fine white thread. At the base of the ligament is a considerable amount of tissue of a brilliant white. This is the degenerated fibrous tissue of the remains of the pectinate system of vessels. The fundus is otherwise quite normal. (See p. 109.)

FIG. 6.—DISC SHOWING A PERFECTLY DEVELOPED PERSISTENT FALCIFORM LIGAMENT. LEFT EYE.

It will be noticed that the disc is entirely covered with a dazzling white conical mass of fibrous tissue, from which a large number of fine fibres spread out. This conical mass of tissue terminated in a strong, white fibrous cord, which split into three smaller cords which could be traced to the sides and centre of the lens capsule. Such an arrangement is the normal condition in many ruminants, notably in the Cervidæ and Antelopelidæ, and certain rodents. The so-called persistent hyaloid artery occurs in man and the Primates in three distinct forms, all of which are rudimentary. The *first* is a short pulsating cæcal vessel filled with blood which springs from the main trunk of the arteria centralis retinæ. *This is the true persistent hyaloid artery.* The *second* is a hollow, trumpet-shaped tube, free from blood, and known as the canal of Cloquet (see Alt's 'Monograph,' Plate 3, p. 422). The distal end is affixed to the lens capsule, and is known as the campanula Halleri (see 'Comparative Anatomy of the Mammalian Eye,' pp. 46–53, and Plate 27, fig. 3). The *third* form consists of a white fibrous cord, which arises from the disc, and terminates either free in the vitreous or is attached by two or three threads to the lens. It is this form which forms the remains of the falciform ligament which occurs normally in the fishes, and is figured in this plate. Observe the ciliary arteries which pass out of the disc along its margin. (See pp. 109, 110, and 111.)

PLATE 3.

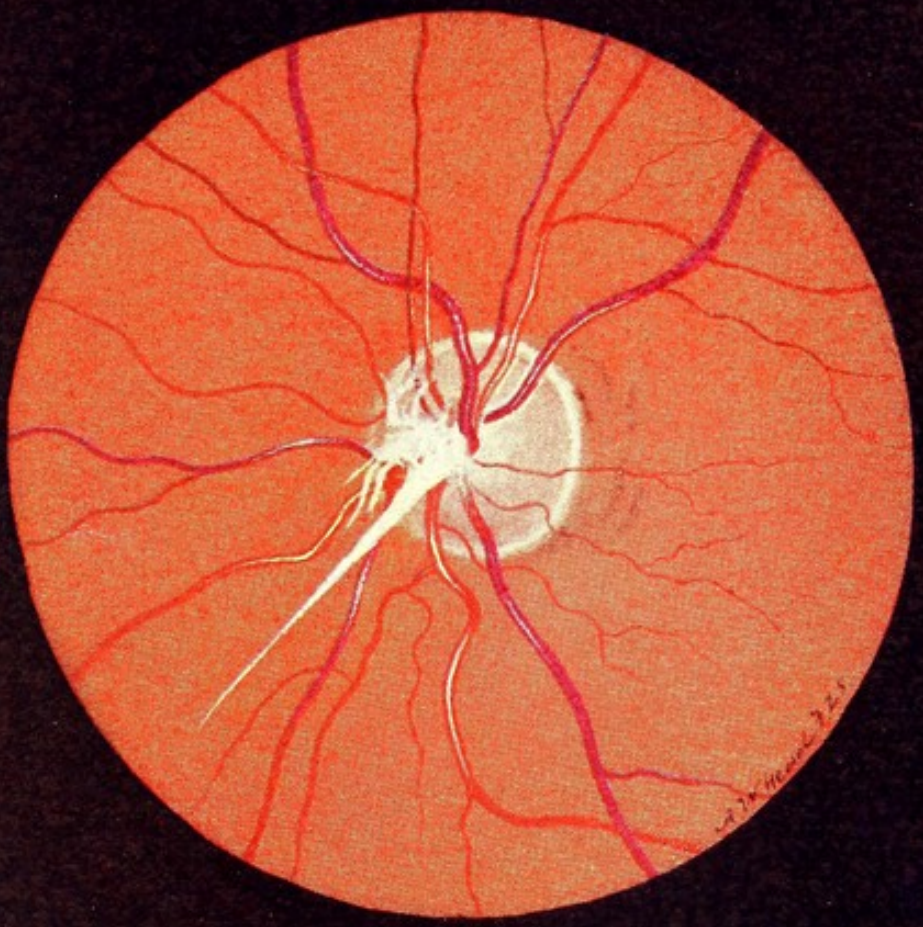

Fig. 5.

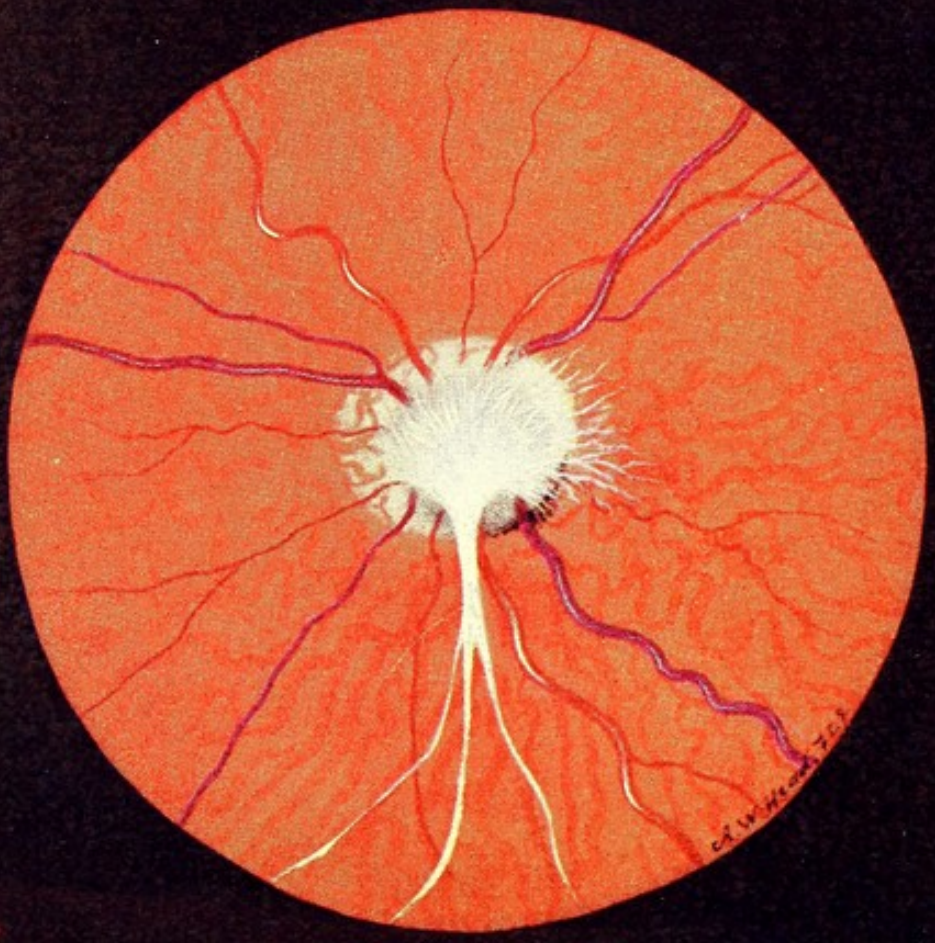

Fig. 6.

A.W. Head.

PLATE 4.

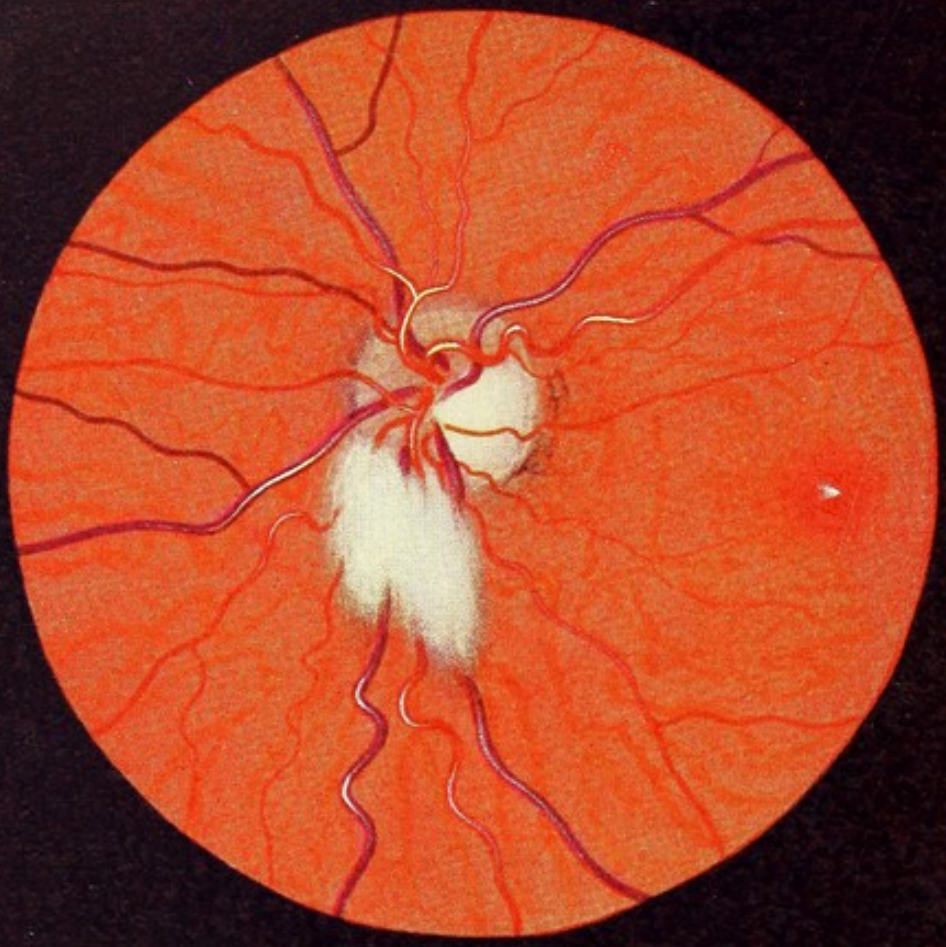

Fig 7.

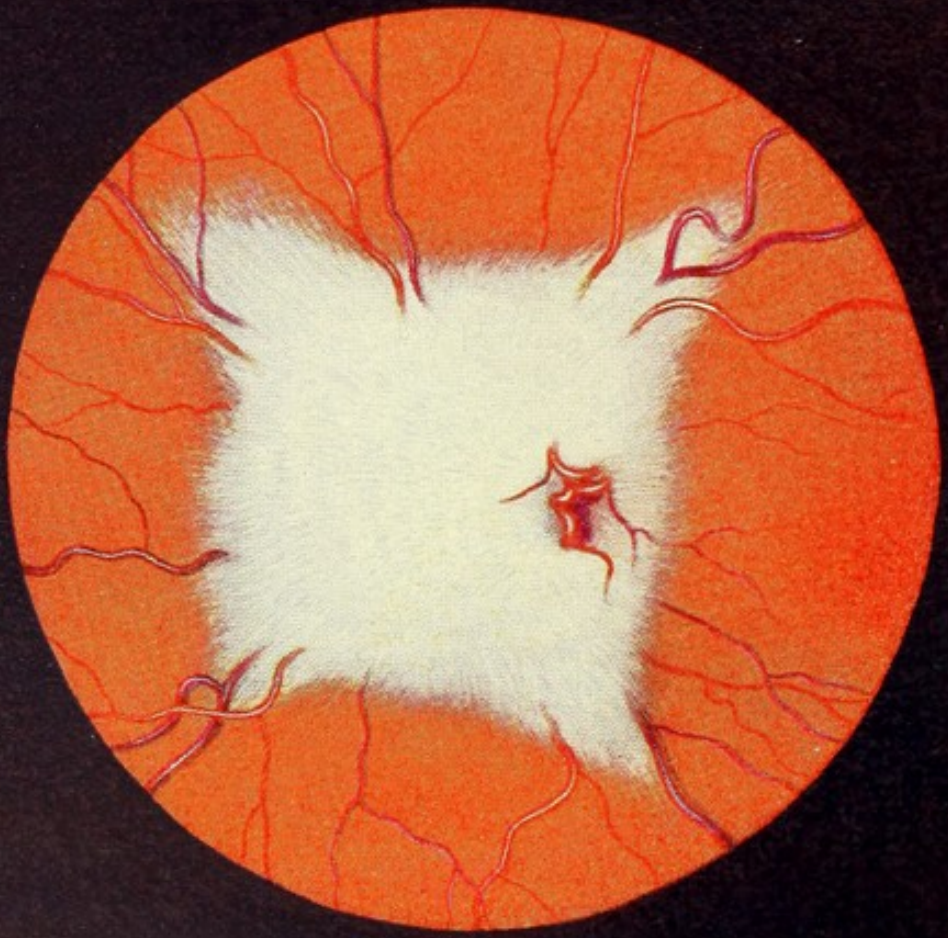

Fig 8.

A.W. Head

PLATE 4.

FIG. 7.—OPAQUE NERVE-FIBRES IN A YOUTH. SMALL TUFT BELOW DISC. LEFT EYE.

This shows a small tuft of opaque fibres on the lower margin of the disc, which otherwise is typically normal, with a well-marked physiological cup. The fibrillar arrangement is clearly seen along the edge of the patch. This conceals a portion of three of the main vessels. The choroidal vessels are seen all over the fundus on a lower plane. A complete scotoma was mapped out over the area corresponding to the patch. The macula side of the disc is bordered by black pigment. The vessels below the patch are rather tortuous. The cause of the opacity is due to the persistence of the medullary nerve-sheaths. The sight was not affected. V. = $\frac{6}{6}$ and J. 1.

FIG. 8.—DO., ON THE INNER SIDE OF THE DISC. VERY EXTENSIVE. LEFT EYE.

This shows a very extensive mass of brilliant white opaque nerve-fibres completely covering the disc and extending about four disc diameters over the fundus. The main trunks of the retinal vessels as well as the disc are entirely hidden by the fibres, which are so dense as to resemble the white fur coat of an Arctic fox. The macula is the typical dark red colour, the fovea centralis being invisible. A large scotoma was mapped out, but with difficulty, as the sight was very defective. V. = $\frac{6}{60}$. From a youth, aged 19 years, who sought advice for amblyopia. The other eye was normal. (See p. 116.)

PLATE 5.

FIG. 9.—PERSISTENT HYALOID ARTERY. GREATLY ENLARGED. RIGHT EYE.

Right eye magnified about twenty-eight times. The disc has a very typical form, with a physiological cup on the temporal side. A large artery is seen to emerge from the centre of the disc, and had the appearance of an inflated sausage-skin. It is obviously a branch of the main trunk of the central artery, and penetrates for some little distance into the vitreous. It was filled with blood, which could be seen pulsating rhythmically with the heart. This is the true form of persistent hyaloid artery. On the macula side of the disc are some coal-black pigment spots. The optic nerve-fibres are semi-opaque, and extend for a short distance round the disc. The vision was reduced to P.L. owing to a degenerative patch occupying the macula region (macula coloboma). The macula sector of the disc (papillo-macula bundle of nerve-fibres) was extremely pale. (Pp. 109 and 110.)

FIG. 10.—LARGE CONGENITAL CRESCENT BELOW THE DISC.

This occurred in an otherwise healthy eye. The disc appeared tilted downwards so that its horizontal diameter was nearly double the vertical. The upper part is bright orange-red in colour and finely stippled with red dots. The lower margin of the disc appeared hazy, due to its being out of focus, and was bordered by a crescent-shaped or nearly hemispherical coloboma, which appeared glistening white, owing to the absence of the retina and choroid over its area, which allowed the sclerotic to shine through. It is stippled with fine grey dots. A ring of black pigment surrounds its outer margin. The choroidal vessels are clearly seen all over the fundus, even up to the disc. V. = $\frac{6}{9}$. (Pp. 112 to 114.)

PLATE 5

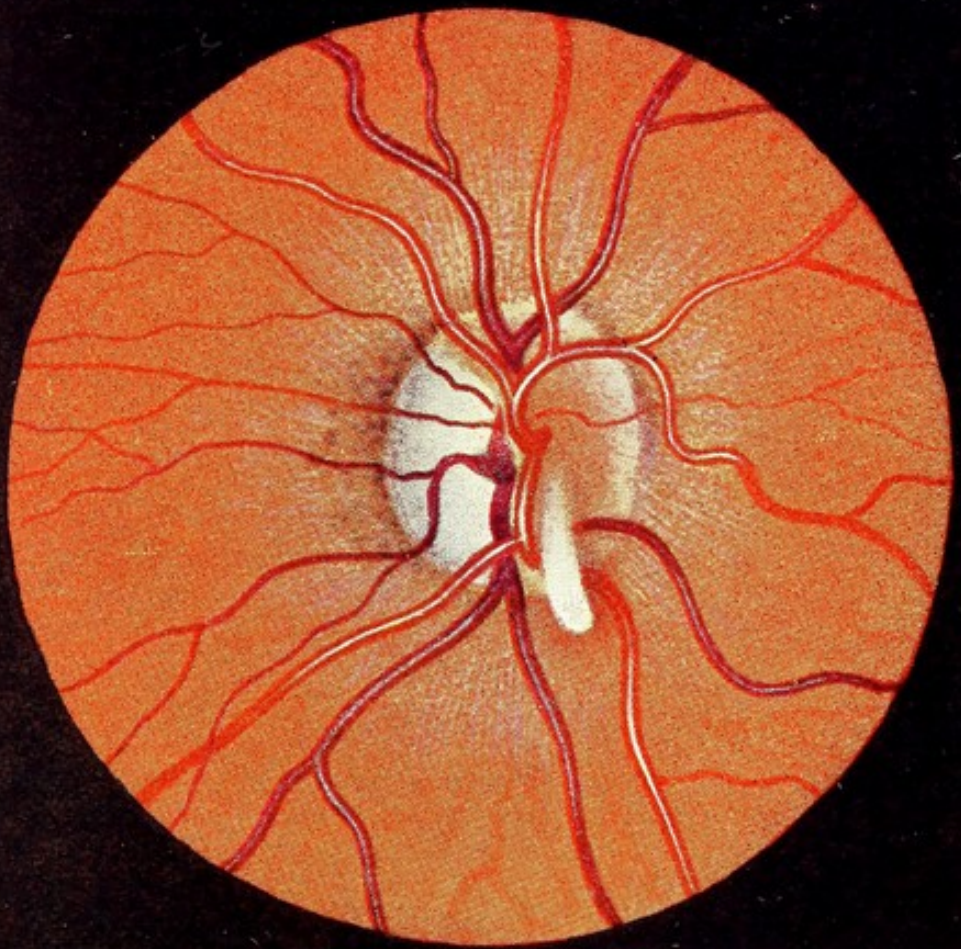

Fig. 9.

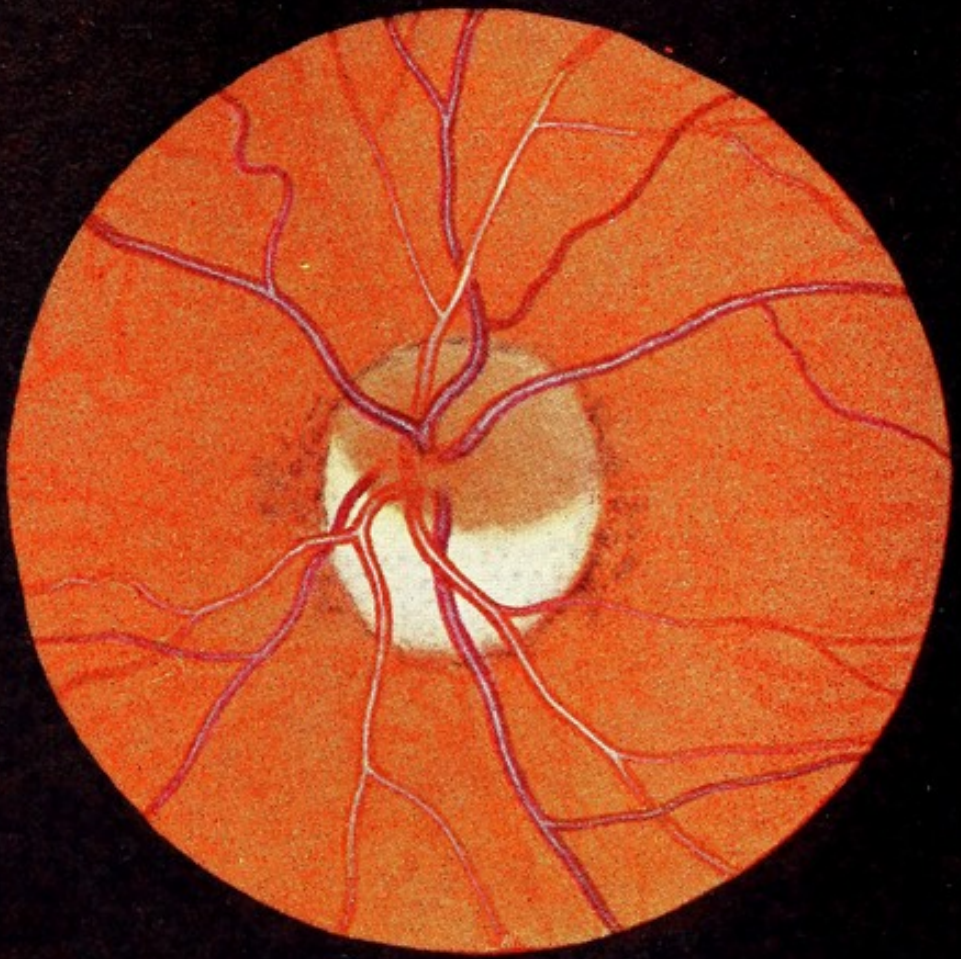

Fig. 10

A. W. Head.

PLATE 6

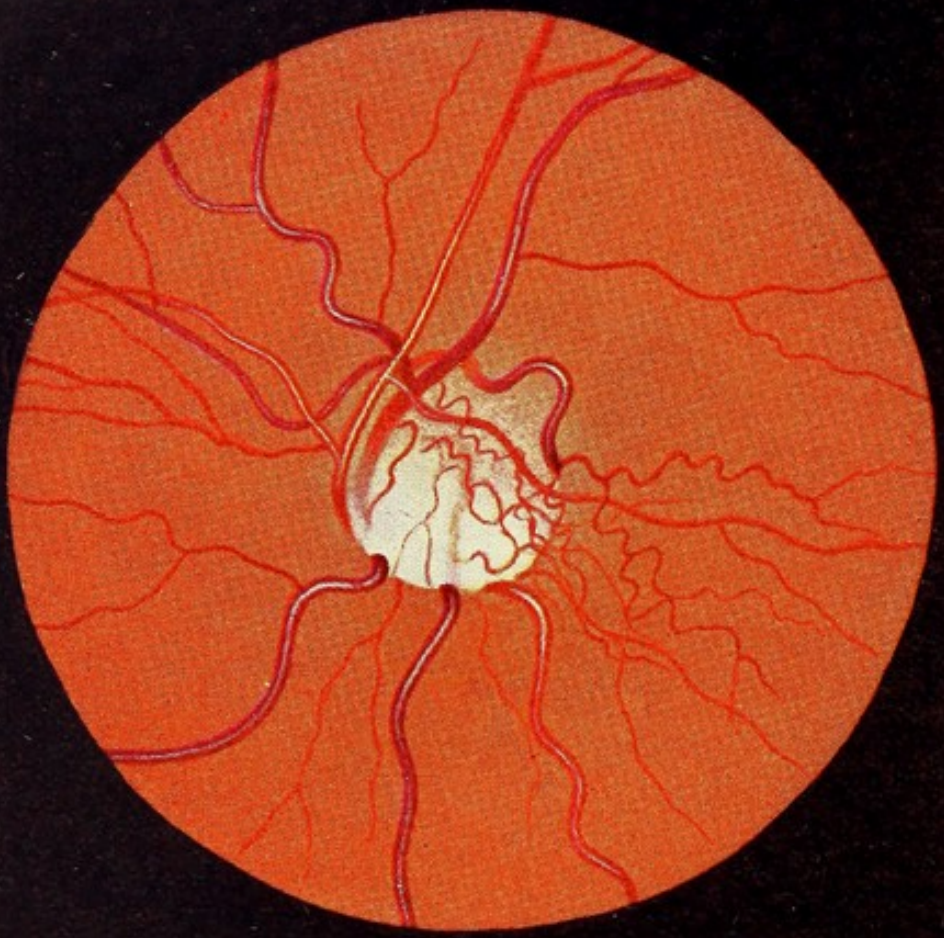

Fig. 11

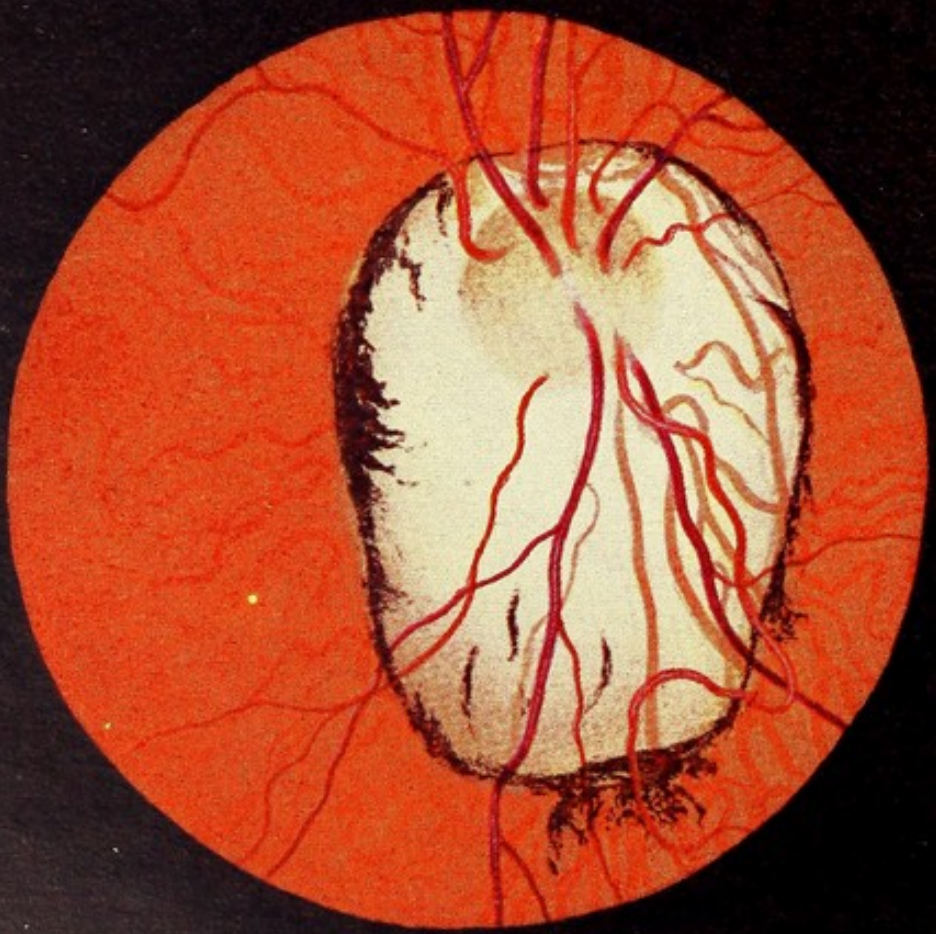

Fig. 12.

A. W. Head.

PLATE 6.

FIG. 11.—COLOBOMA OF THE DISC. LEFT EYE.

These congenital abnormalities are closely related to congenital crescents and physiological cups. They are extremely diverse in appearance, no two being exactly alike. One may trace their origin to some interference with the complete closure of the fœtal ocular cleft, or with the investment of the scleral sheath around the papilla. In this drawing the disc is seen to be excavated at its lower part, and the vessels are seen to curl over the edge, as in a glaucomatous cup. This part is not the margin of the disc, but the edge of the retina and choroid. The lower edge of the disc cannot be identified, as it is evidently tilted down below the general surface of the fundus, which had the appearance of a crater-like hole. The blood-vessels emerge from the disc in a very irregular fashion, and the trunk of the arteria centralis is seen to emerge near the upper edge of the disc. A few cilio-retinal vessels are also seen. (Pp. 112 to 114.)

FIG. 12.—A LARGE COLOBOMA OF THE DISC. RIGHT EYE.

This is a typical form of the unclosed gap in the fœtal cleft, which surrounds the disc on all sides. The free margin of the choroid is well seen, and is very prominent owing to the abundant mossy pigment along its edge. The fundus is covered with choroidal vessels, which encroach on the inner side of the coloboma, almost to the edge of the disc, the lower part of which is buried and lost in the white floor of the coloboma. The macula is seen to the left of the drawing, on a level with the lower border of the disc. V. = $\frac{6}{9}$. (See p. 114.)

PLATE 7.

FIG. 13.—SMALL COLOBOMA OF THE MACULA. LEFT EYE.

A peculiar crater-like hole is seen occupying the whole of the macula area. The base of the pit is covered with a dense layer of black pigment, which is surrounded by a ring of atrophied choroid, and this, again, by a ring of pigment. This latter lies on the general fundus background, which is distinctly seen to be on a higher level than the atrophic ring. Five retinal vessels pass over the edge of the crater and are lost in the central pigment. V. = $\frac{20}{70}$. The lesion appears to be due to a congenital central patch of choroiditis. There was no history of syphilis, but the sight was noticed to have been defective from birth. The disc and other parts of the fundus appeared quite normal. (P. 114.)

FIG. 14.—MACULA COLOBOMA IN THE RIGHT EYE. VERY LARGE.

Extending downwards and about one disc's diameter to the outer side is a large oval area of choroidal atrophy of scleral whiteness, in the centre can be seen a circular patch about the size of the optic disc, of a buff chamois-leather colour, surrounded with a ring of black pigment. Several choroidal vessels cross the exposed sclerotic, and dense black pigment of a mossy nature fringes its margin. Between the pigment are large white oval areas of exposed sclerotic. This, from its size and the presence of a downward congenital crescent, is probably due to a defect in the closure of the fœtal cleft. The direction of this choroidal cleft is always either downwards or obliquely outwards and slightly downwards. In the latter case it usually involves the macula, in which there is sure to be some indication of a coloboma of the disc, or at any rate of a congenital crescent. (Pp. 112–114.)

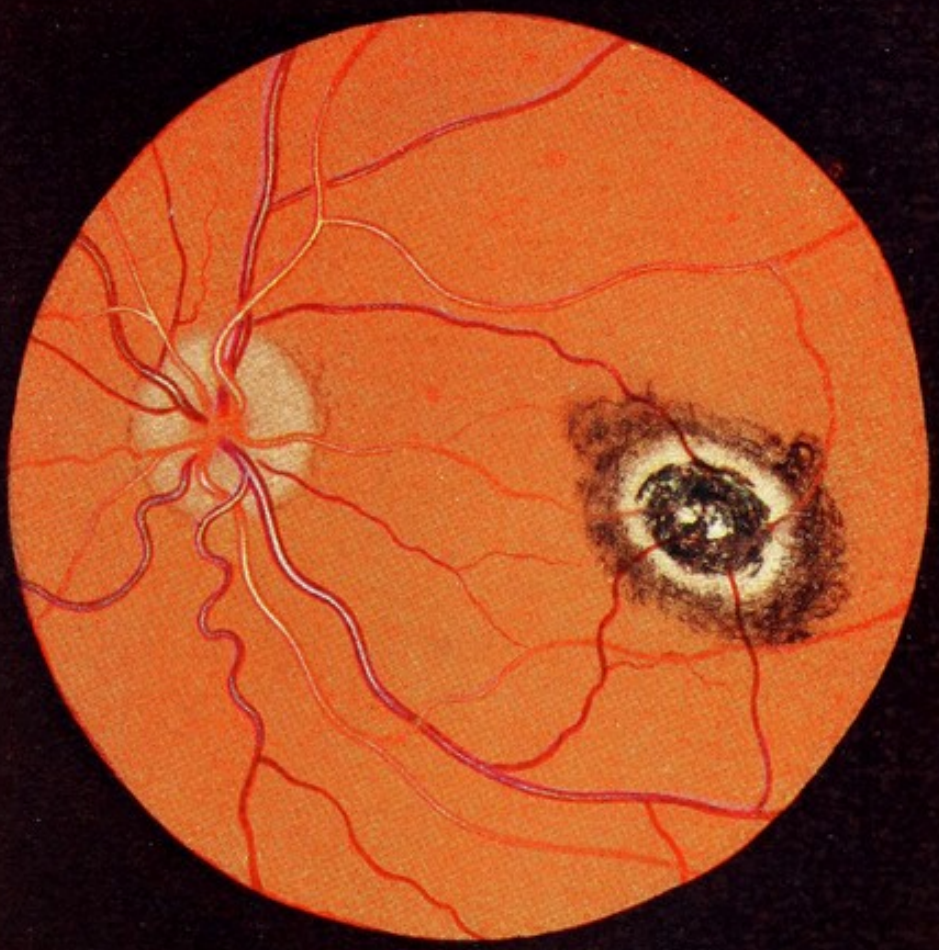

Fig. 13.

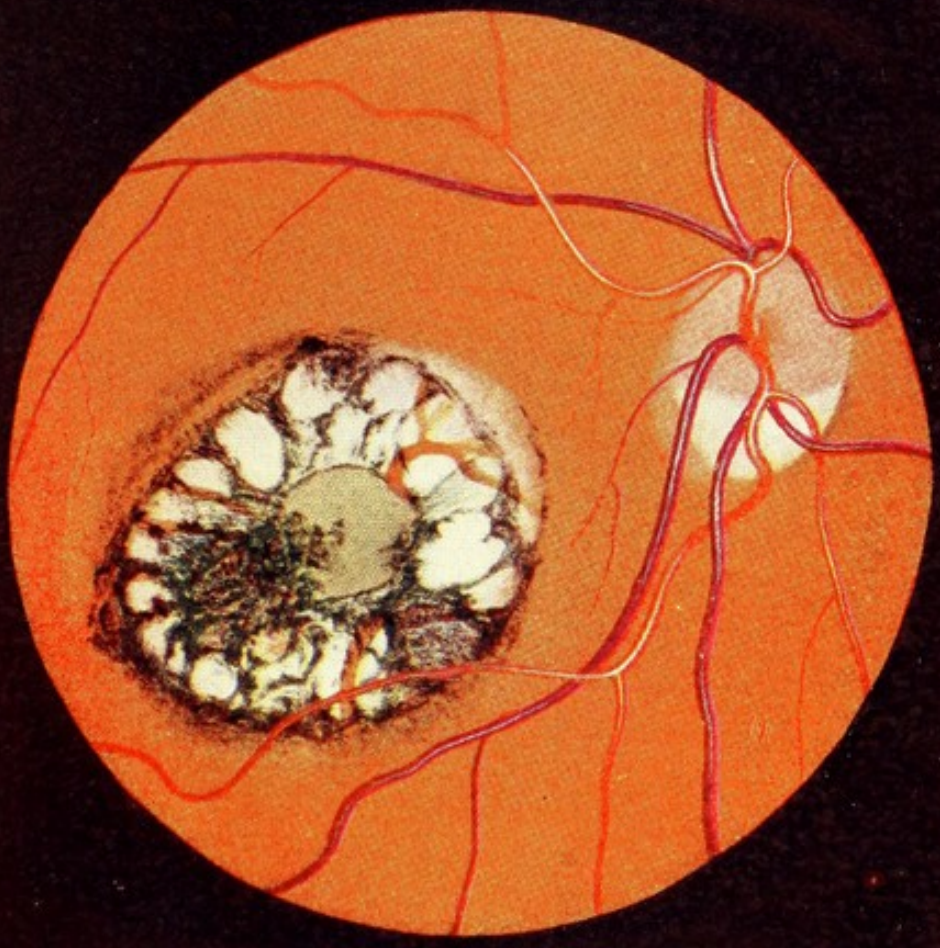

Fig. 14.

A. W. Head.

PLATE 9.

FIG. 17.—SMALL PHYSIOLOGICAL CUP IN THE CENTRE OF THE DISC. LEFT EYE.

This is a very common type of physiological cupping of the optic nerve. In the centre of the disc, which is a little lighter in colour than the surrounding fundus, is shown a typical white central depression, oval in shape, into which the retinal vessels dip. The disc is surrounded with an irregular ring of black pigment. The veins are one third greater in calibre than the retinal arteries, which is their typical normal proportion. V. = $\frac{6}{5}$. Slight degree of hypermetropia.

FIG. 18.—PHYSIOLOGICAL CUP INVOLVING THE OUTER HALF OF THE DISC.

The ordinary type of a shallow physiological cup, involving the outer half or macula side of the optic disc. This is the right eye of a person with medium complexion, light brown hair, grey irides, and fair skin. V. = $\frac{6}{5}$. (Pp. 106 and 141.)

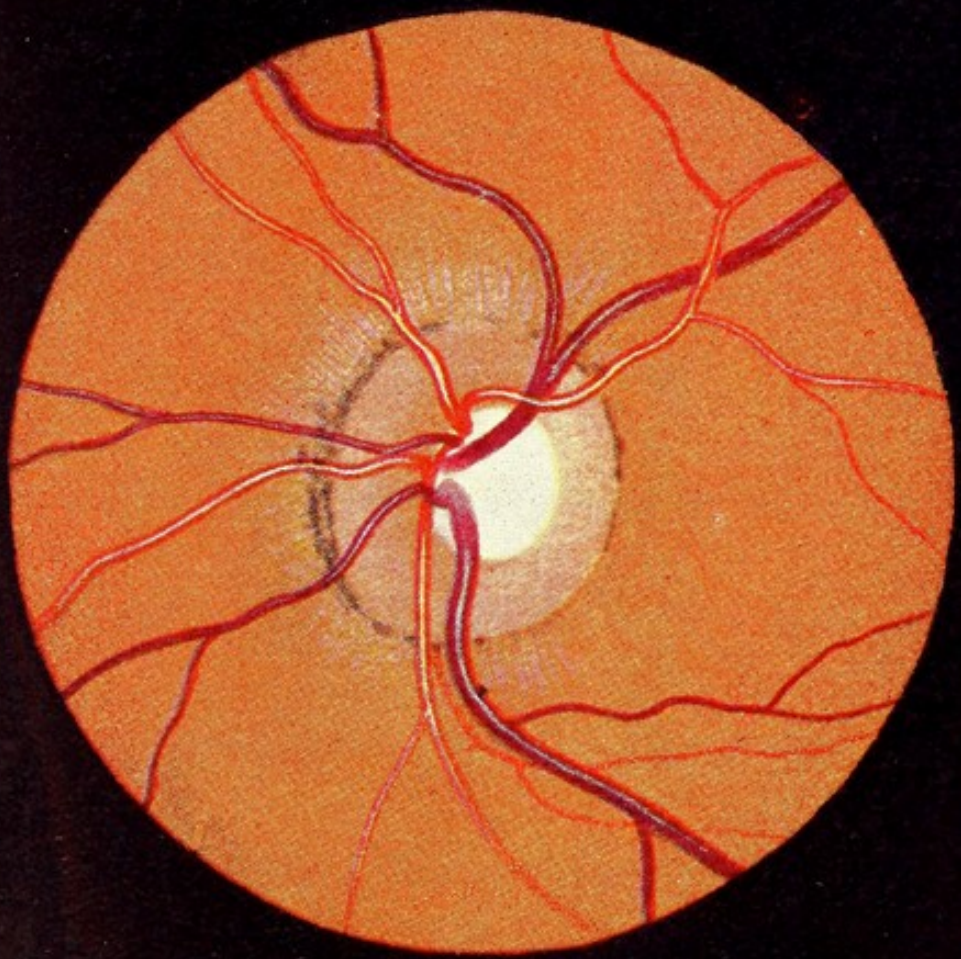

Fig. 17.

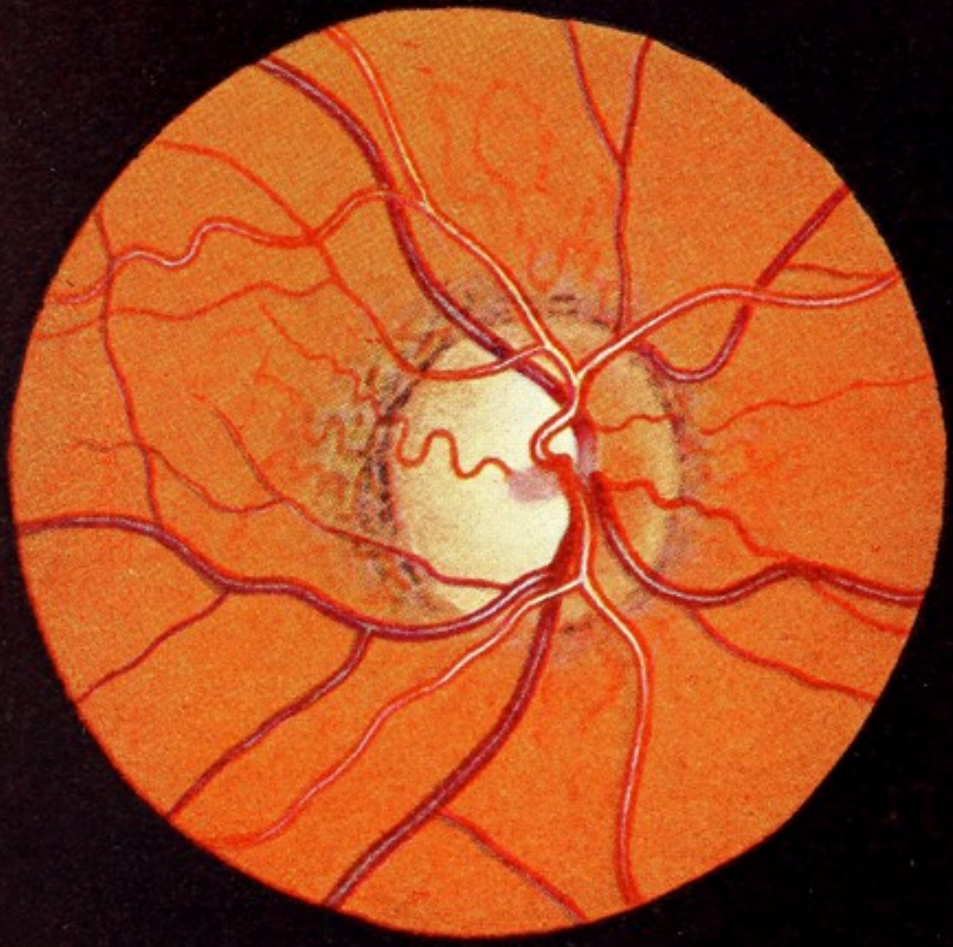

Fig. 18.

A.W. Head.

PLATE. 10.

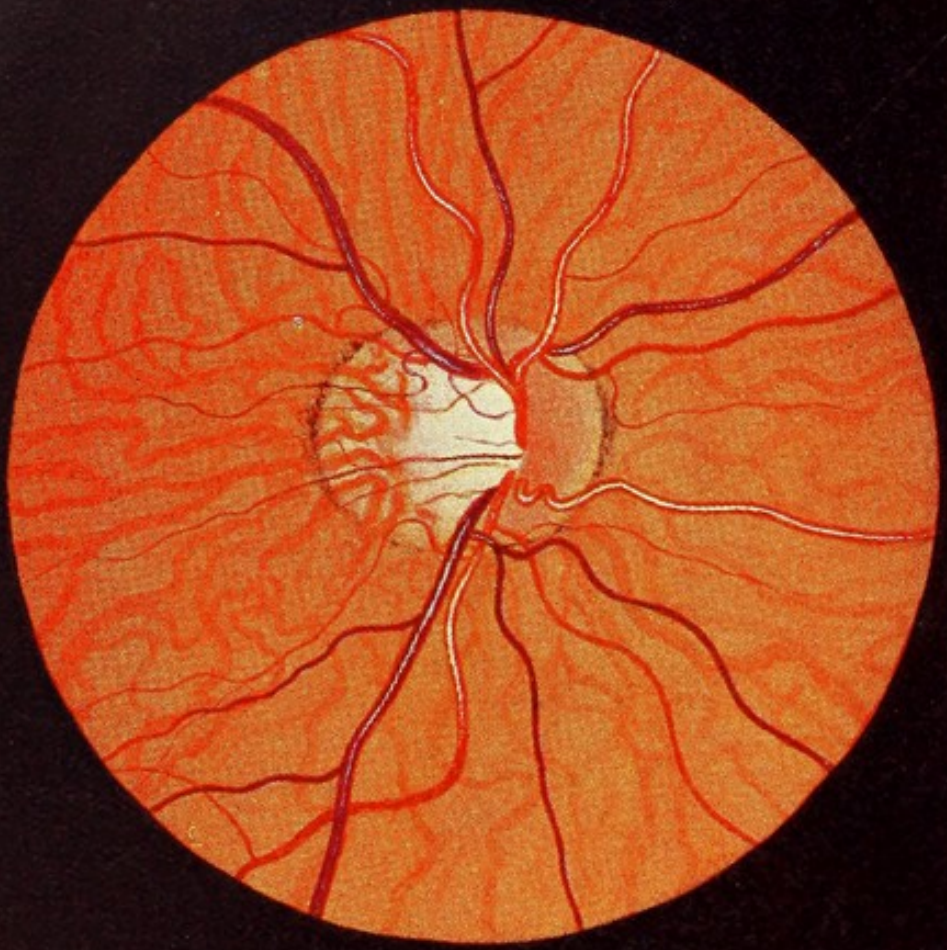

Fig. 19.

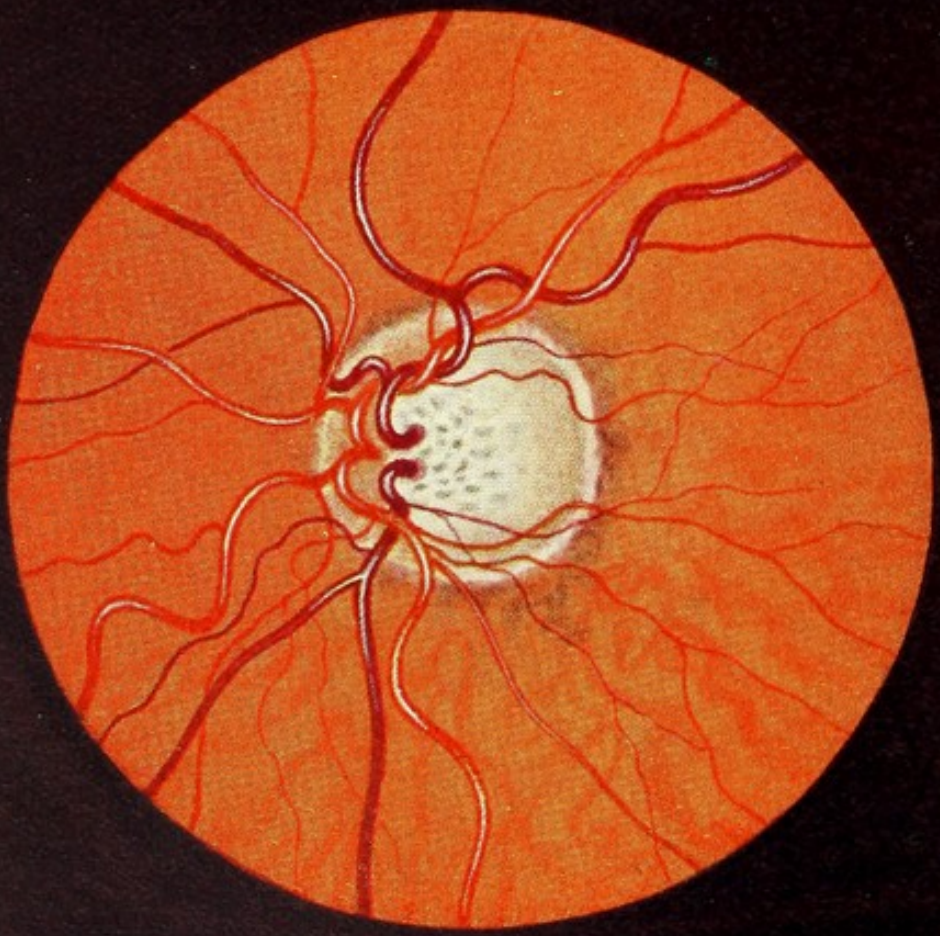

Fig. 20.

A. W. Head.

PLATE 10.

FIG. 19.—PHYSIOLOGICAL CUP IN THE RIGHT EYE. CHOROIDAL VESSELS ENCROACHING ON THE DISC.

A similar physiological cup to Fig. 18, with opening towards the macula or temporal border. Only the choroidal vessels show up distinctly on the edge of the cup, which is stippled with very fine red dots. Some of these choroidal vessels appear to encroach on the disc. It is difficult, though, to determine whether these vessels really lie on the disc or only outside it. Frequently choroidal vessels are found in similar positions in myopic eyes; what their connection with myopia may be is not known. (Pp. 106 and 141.)

FIG. 20.—VERY LARGE PHYSIOLOGICAL CUP, WITH THE LAMINA CRIBROSA SHOWING THROUGH.

This is a left eye, and shows a very marked degree of physiological cupping, and might easily be mistaken for the early stage of a glaucomatous cup, which it very much resembles. Note how the vessels curl over the edge of the central chalky-white pit. Also the grey dots on its floor, indicating the lamina cribrosa. The vessels in this case and in the last one do not curl over the outer side of the disc as in glaucoma, but only over the edge of the pit, which is well inside the edge of the disc. Observe the healthy pink colour of the disc. V. = $\frac{6}{5}$ J. 1.

PLATE II.

FIG. 21.—SIMPLE ATROPHY OF THE OPTIC NERVE. LEFT EYE.

This case occurred in a man, aged 32 years. History of syphilis ten years before. Ataxic symptoms; Argyll-Robertson pupil reaction; absence of knee- and ankle-jerks. The fundus is abnormally red, and the margin of the disc, which is a dead white, is very sharply defined. The shallow cupping of the disc gives it rather a pale grey appearance. A thin ring of coal-black pigment surrounds its outer border; the vessels are slightly smaller than normal, and devoid of all their fine branches; the macula is stippled with very minute red dots. V. = P.L. only. (Pp. 137–138.)

FIG. 22.—ADVANCED GREY ATROPHY OF THE OPTIC NERVE. THE ARTERIES GREATLY REDUCED IN CALIBRE.

A similiar case to above, but more advanced. The disc has a bluish-grey mottled appearance (grey atrophy); the lamina cribrosa is clearly exposed. The arteries have become very small, appearing as mere scarlet threads. The pupils are widely dilated and the macula is a deep red colour. V. = *nil.* (P. 137.)

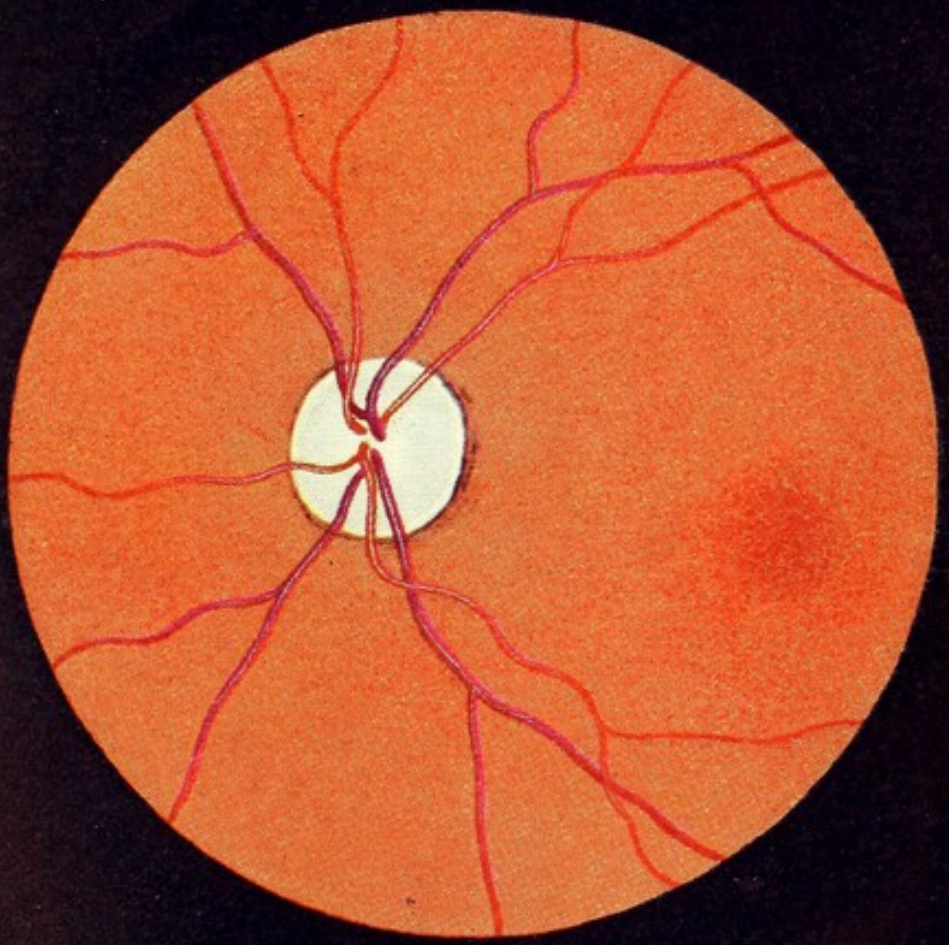

Fig. 21.

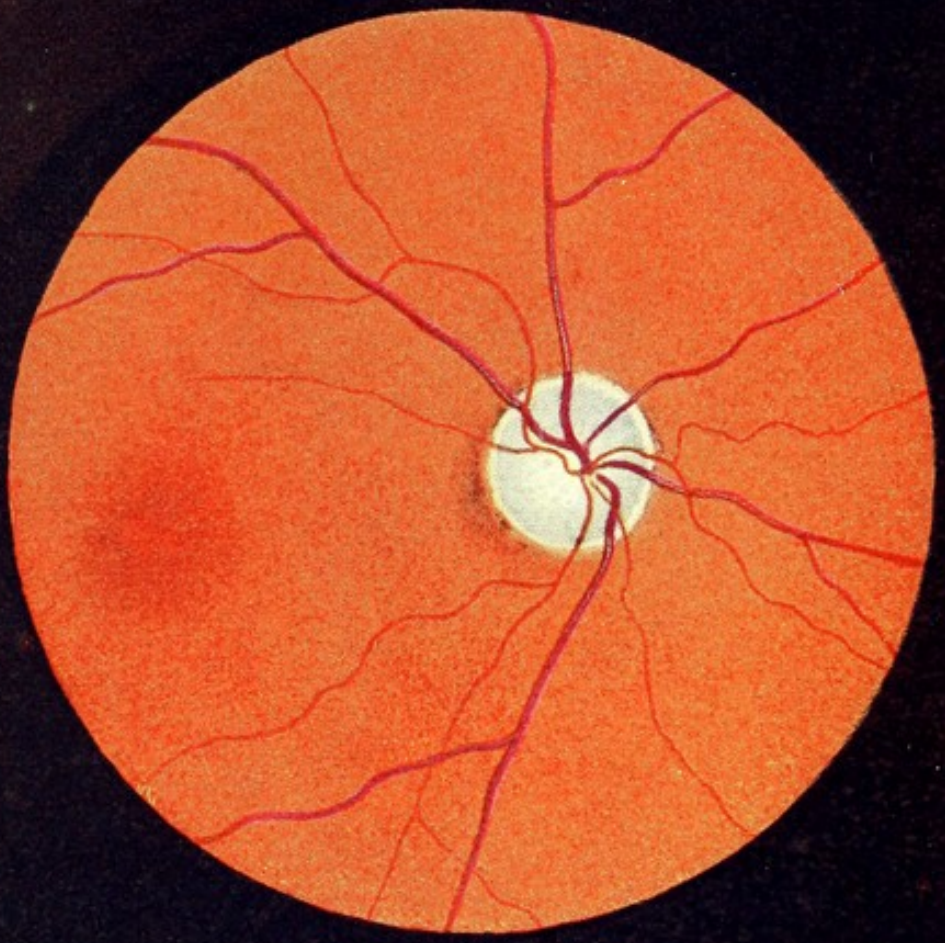

Fig. 22.

A. W. Head

PLATE 13.

FIG. 25.—OPTIC NEURITIS. THE EARLY STAGE.

History of failing vision during the last four months. No trace of syphilis. Had a blow on the head five months ago with a stick; was unconscious for some hours. The disc is swollen and the margin quite invisible, fusing, as it were, into the general red background. The level of the centre of the disc is distinctly above that of its edge. The vessels are not tortuous, but fairly normal. The macula is dark red and stippled. V. = $\frac{6}{12}$. (Pp. 132, 133.)

FIG. 26.—DO. MORE ADVANCED. RETINAL VESSELS VERY TORTUOUS.

The appearance is much the same as in the last case, but the vessels, especially the veins, are very tortuous. The optic disc is greatly swollen and stands forward like the top of a champagne cork. The retinal veins curve over the sides, and are now engorged with blood, while the arteries are contracted and like mere threads. The nerve-fibres appear more distinct and radiate a short distance from the disc all round. (Pp. 132, 133.)

PLATE 13.

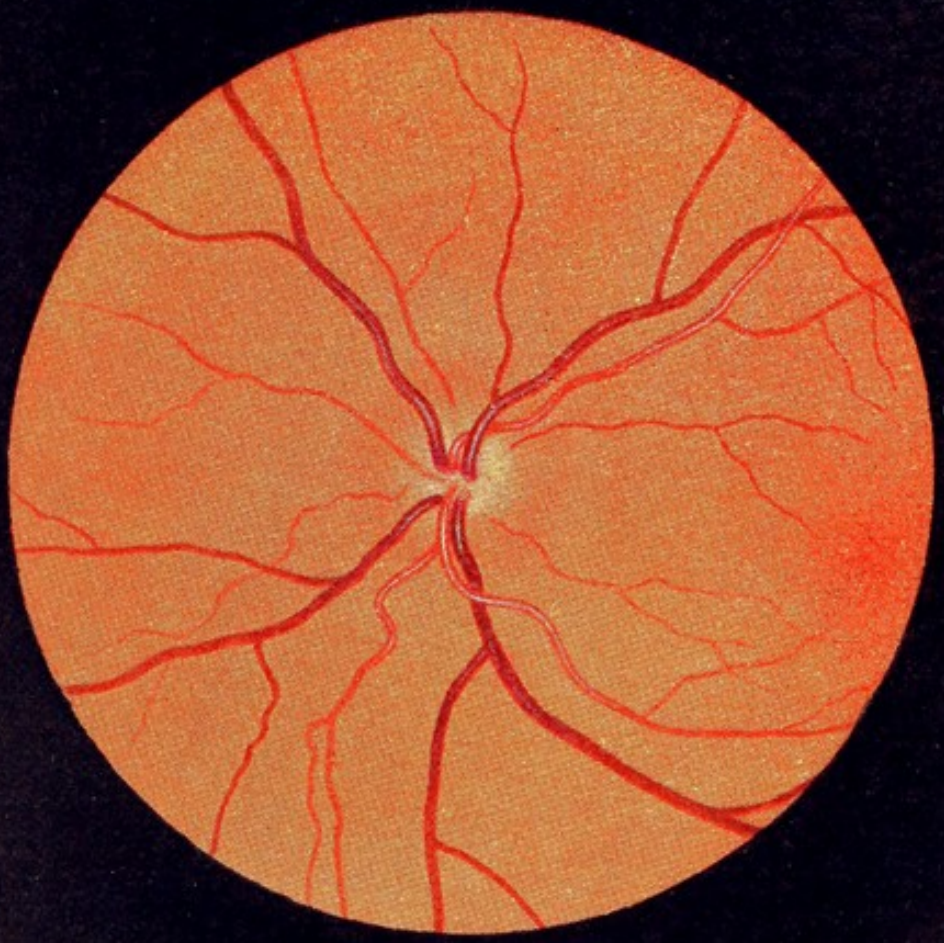

Fig. 25.

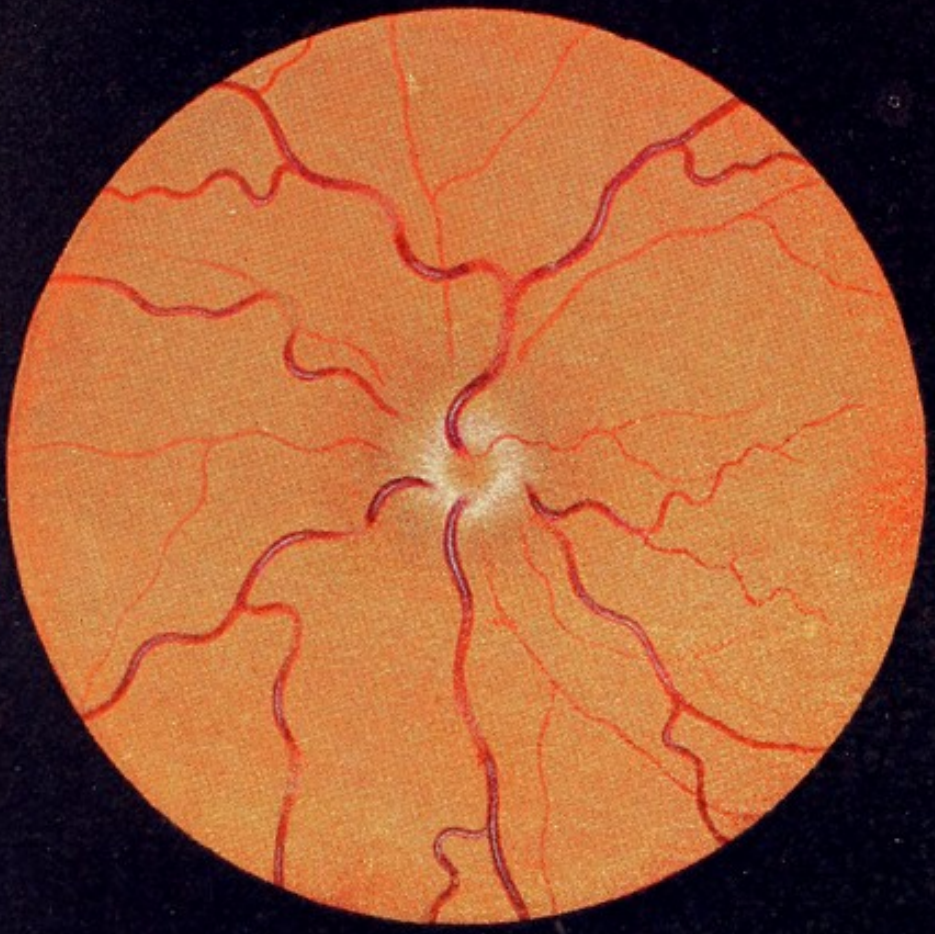

Fig. 26.

A. W. Head

PLATE 14.

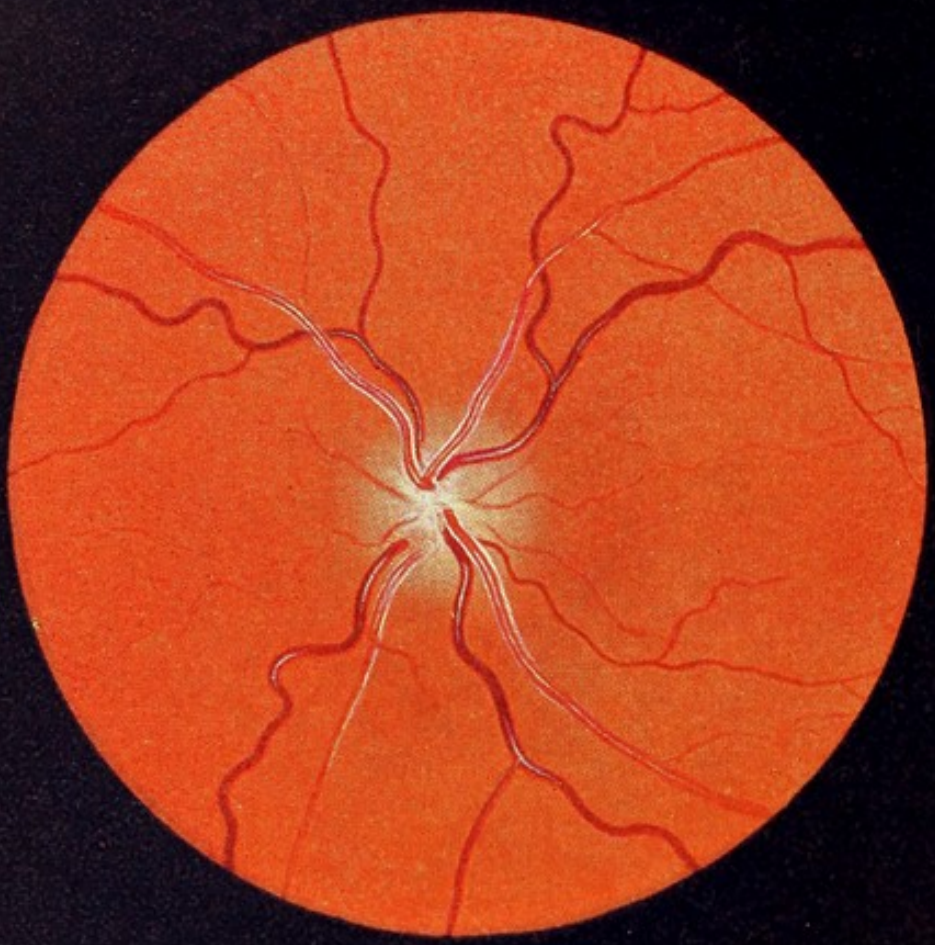

Fig. 27.

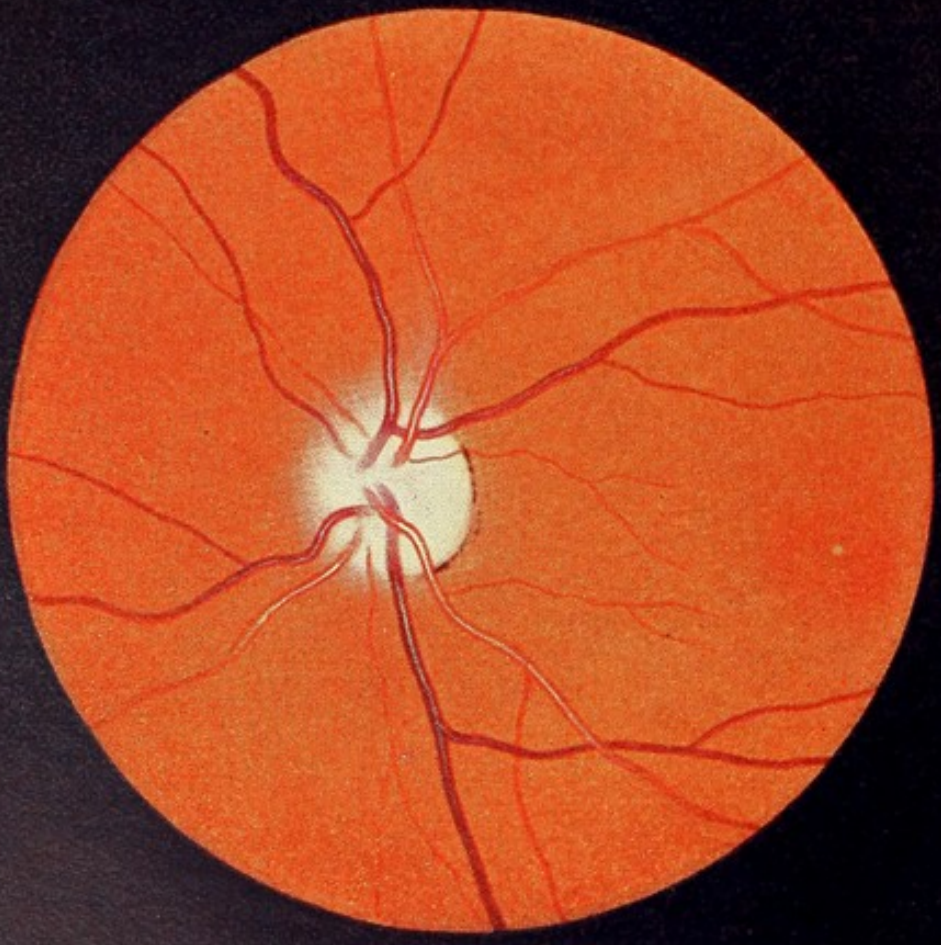

Fig. 28.

A. W. Head.

PLATE 14.

FIG. 27.—OPTIC NEURITIS. SUBSIDING.

The patient came a month before with acute papillitis. Vessels congested and slightly tortuous. The margin of the disc was quite buried in the effusion of the surrounding retina, and the nerve-fibres showed signs of radiation. V. = $\frac{6}{18}$. There was a history of syphilis. Chancre two years ago. Under mercurial treatment pushed to salivation the disc cleared up and the margin became much better defined, although not clear. The vision improved to $\frac{6}{9}$. The disease subsequently cleared up and the vision returned to normal. Such a fortunate termination is quite the exception. (P. 133.)

FIG. 28.—DO. ATROPHIC STAGE. LEFT EYE.

The disc, especially on the macula side, has become quite white, and the margin well defined. On the nasal side the edge of the disc is still blurred and imperfectly defined. The macula is dark red and finely stippled with pigment. The disc will inevitably become uniformly white and clean cut, and the vision will sink to *nil*. (Pp. 137, 138.)

PLATE 15.

FIG. 29.—ALBUMINURIC RETINITIS. EARLY STAGE.

The papilla is somewhat swollen, reddish, and ill-defined. and surrounded by flame-shaped hæmorrhages. Small hæmorrhages are scattered over the retina. The macula is of a dark dull red, and surrounded by numerous chalky-white dots. Several small white woolly patches are to be seen scattered over the fundus; all the retinal vessels are normal in size and colour. The patient's urine was loaded with albumen.

FIG. 30.—DO. VERY ADVANCED. STAR FIGURE AT THE MACULA.

The disc has become almost invisible. White lines appear each side of the retinal veins. A large number of small, chalky white dots of irregular outline are grouped in patches around the disc and macula, also several dull white flocculent or woolly patches are scattered over the fundus. Several flame-shaped hæmorrhages can be observed near the retinal vessels. The macula is a dull dark red, from which a number of irregular glistening white streaks radiate, like the spokes of a wheel. An immense number of fine discrete and brilliantly white dots are scattered round and close to the macula. This patient, who was aged 36 years, only lived four months after this drawing was made. It is extremely rare for a patient suffering from such a marked degree of albuminuric retinitis to remain alive for more than a year afterwards. (See p. 150.)

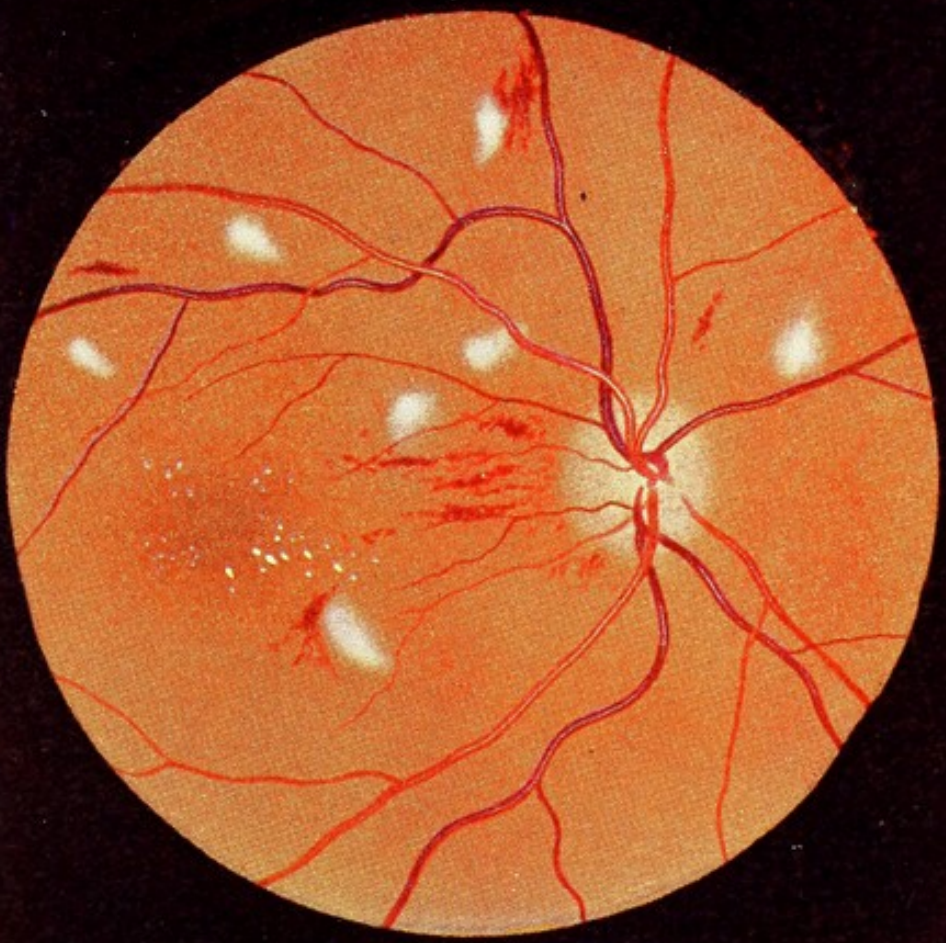

Fig. 29.

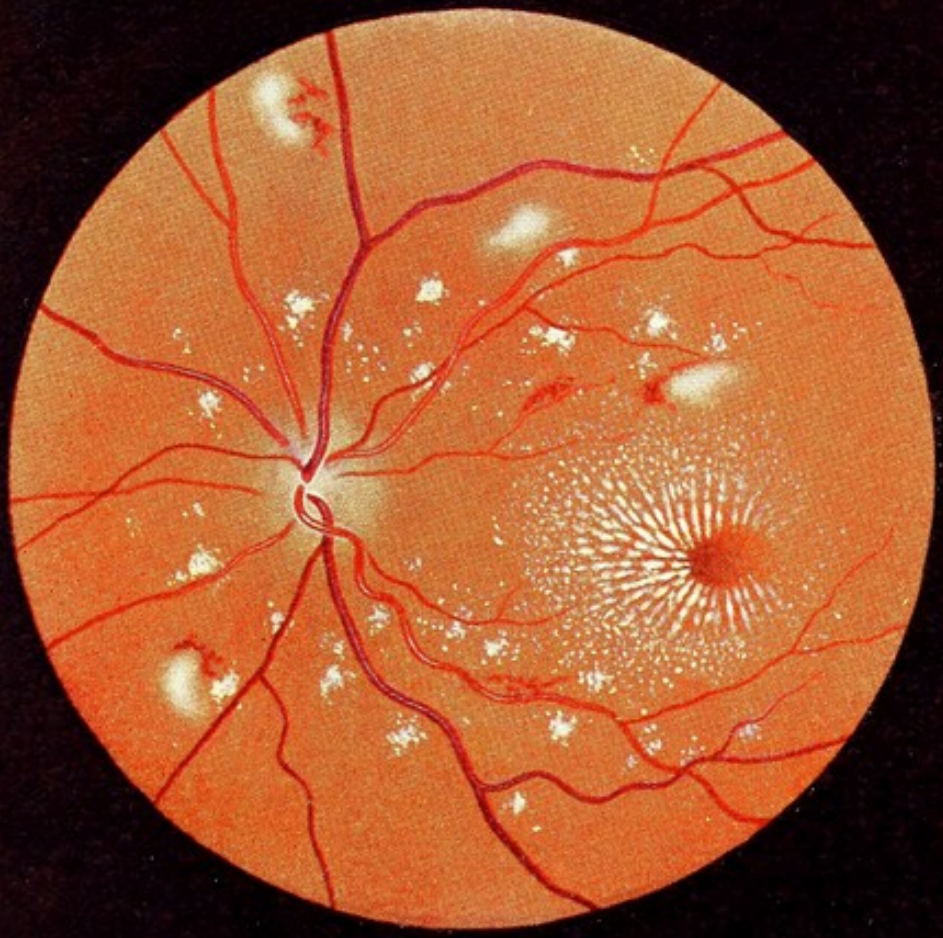

Fig. 30.

A. W. Head.

PLATE 16

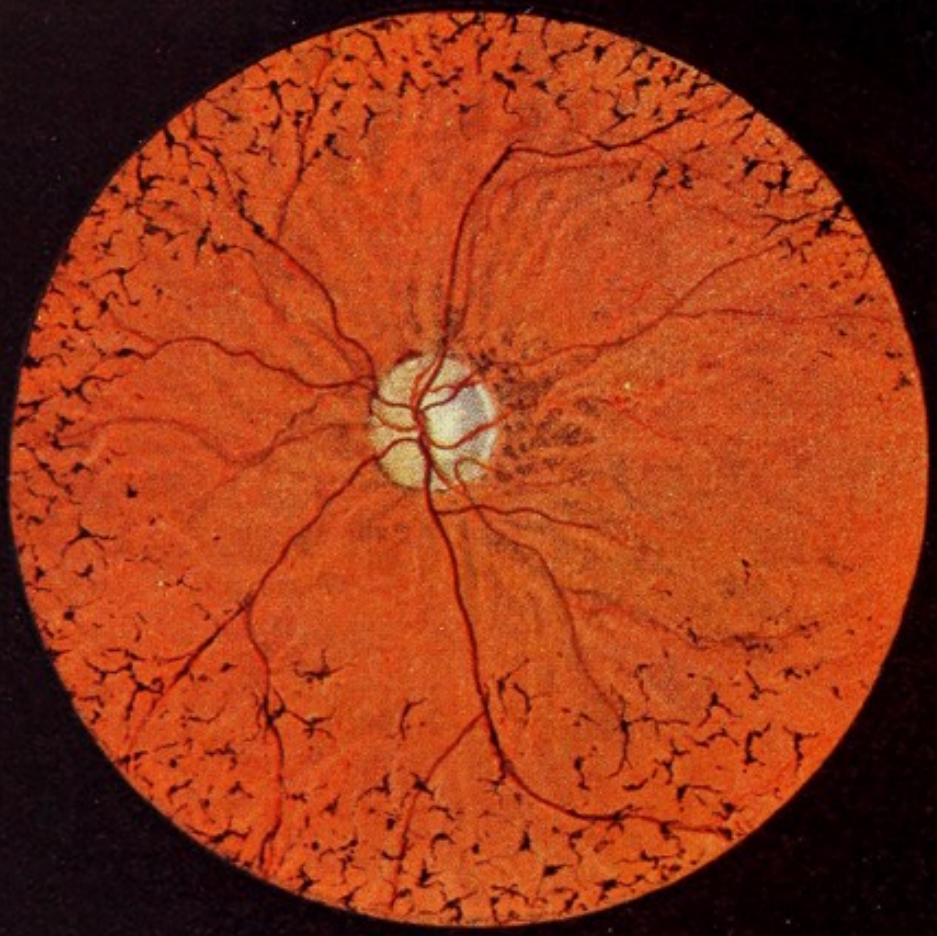

Fig. 31.

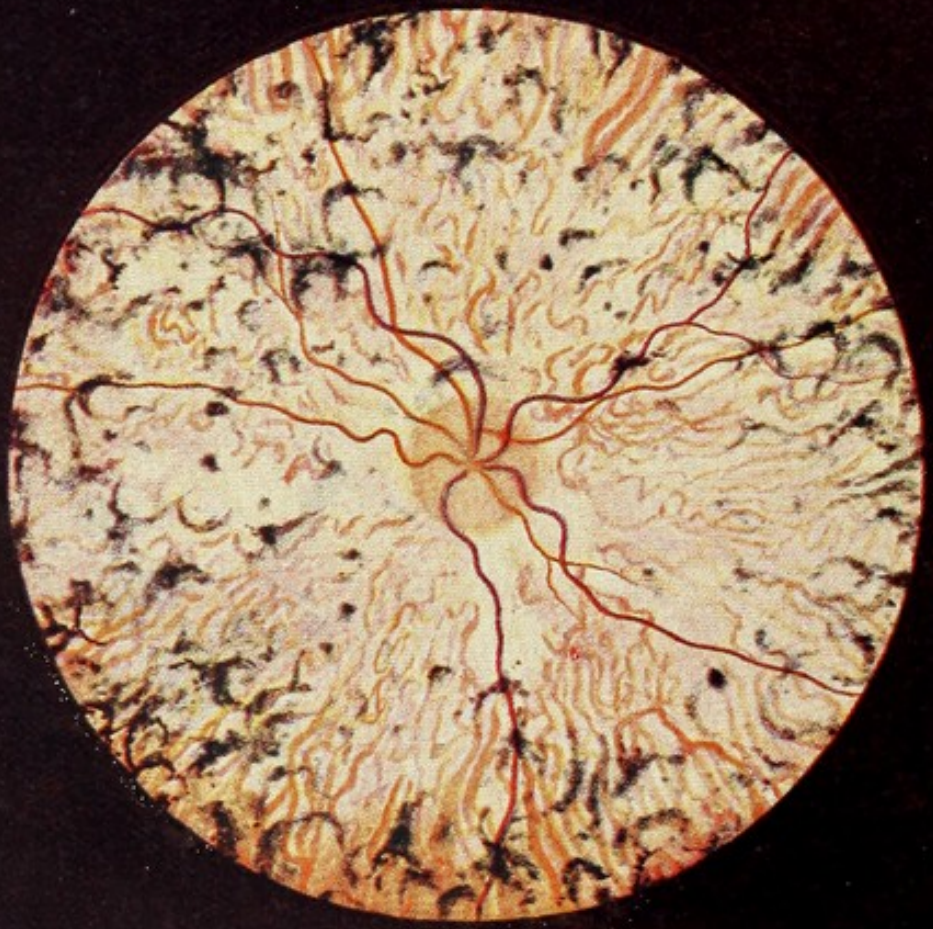

Fig. 32.

A. W. Head

PLATE 16.

FIG. 31.—RETINITIS PIGMENTOSA.

The patient, a young man aged 23 years, had found his vision slowly deteriorating for a long time. His brother and sister were suffering from the same disease. The disc had a pale yellow waxy look on the temporal side, beyond the connective-tissue ring, a mottled crescent fringed with dense black pigment, and the retinal vessels were very thready. The choroidal vessels can be clearly seen all over the retina excepting near the macula area. The central part of the fundus is free from pigment, but towards the periphery the pigment resembling bone-corpuscles increases rapidly in quantity. (Pp. 155–158.) The visual area corresponding to the pigmented part of the fundus, and for some distance internal to it, was quite insensitive to light. The vision varied greatly according to the intensity and character of the illumination, varying from $\frac{6}{24}$ in bright sunlight to $\frac{6}{60}$ or even bare perception of large objects after sunset, or in candle-light.

FIG. 32.—DO. PARTIAL ATROPHY OF THE CHOROID.

The disc has become darker and more waxy looking. The pigment has extended its formation as far as the disc, and is seen to have assumed much larger mossy-looking patches. The natural orange-red of the fundus has nearly all gone, and the choroidal vessels are seen everywhere, but for the most part thin, hidden, and tangled (like a skein). The retinal vessels are mere scarlet threads. V. = bare P.L. only. (Pp. 155–158.)

PLATE 17.

FIG. 33.—RETINITIS PROLIFERANS. RIGHT EYE.

The patient, aged 57 years, had been suffering for a long time with Bright's disease. Small hæmorrhages had occurred in the fundus from time to time. The characteristic features of this disease are well shown here; they consist in several thick fibrous bands of connective tissue, which arise at or near the disc, and spread out into curved leashes, enclosing small round or oval areas of the fundus. There are good reasons for believing that this connective tissue arises from the abundant proliferation of the endothelial cells of the torn blood-vessels, and not from the leucocytes as is often thought to be the case. Hæmorrhages, or the pigmented residua of hæmorrhages, are nearly always present, and local detachment of the retina is very common. In this case there is no detachment, but several oval areas—"islands"—are present or in process of formation. One of these (near the disc) is filled by a partly re-absorbed hæmorrhage. The scar-tissue lies for the most part *behind* the retinal vessels, but one or two of the smaller bands pass in front of them. In some cases the bands run entirely *in front of* the vessels, and even project forwards into the vitreous. As Bright's disease is a very common feature, the urine should be tested in every case for albumen and casts. (P. 154.)

FIG. 34.—RETINITIS CIRCINATA. LEFT EYE.

Patient was a woman, aged 40 years, who complained of a large cloud obscuring the field of view of the left eye. The ophthalmoscope showed a characteristic belt or zone of white spots of various sizes surrounding the macula region and extending over a considerable portion of the fundus. These spots are partly discrete and partly fused together, forming white and perfectly flat patches. Sometimes the zone (as in this case) is circular; more often it is crescentic or horse-shoe-shaped (see Fig. 15). A variable amount of pigment may be seen scattered around the patches. The perimeter showed a large, well-defined, central scotoma. This is very characteristic, and usually embraces 10° to 20° or more. The disease is one of advanced life, and is more commonly confined to one eye, although it has often been observed in both. J. Hutchinson originally described it as symmetrical central choroido-retinitis. It is evidently identical with the "degenerescence circinée" of Dufour and Gonin. In one of the writer's cases the disease occurred during the later stages of syphilis, and most of the patients show signs of arterio-sclerosis. The spots and patches have been variously ascribed to œdema (Siegrist), to hyaline degeneration of escaped blood-corpuscles (Ammann), to fatty degeneration (Landolt), and to albuminous exudates (Fuchs). (P. 153.)

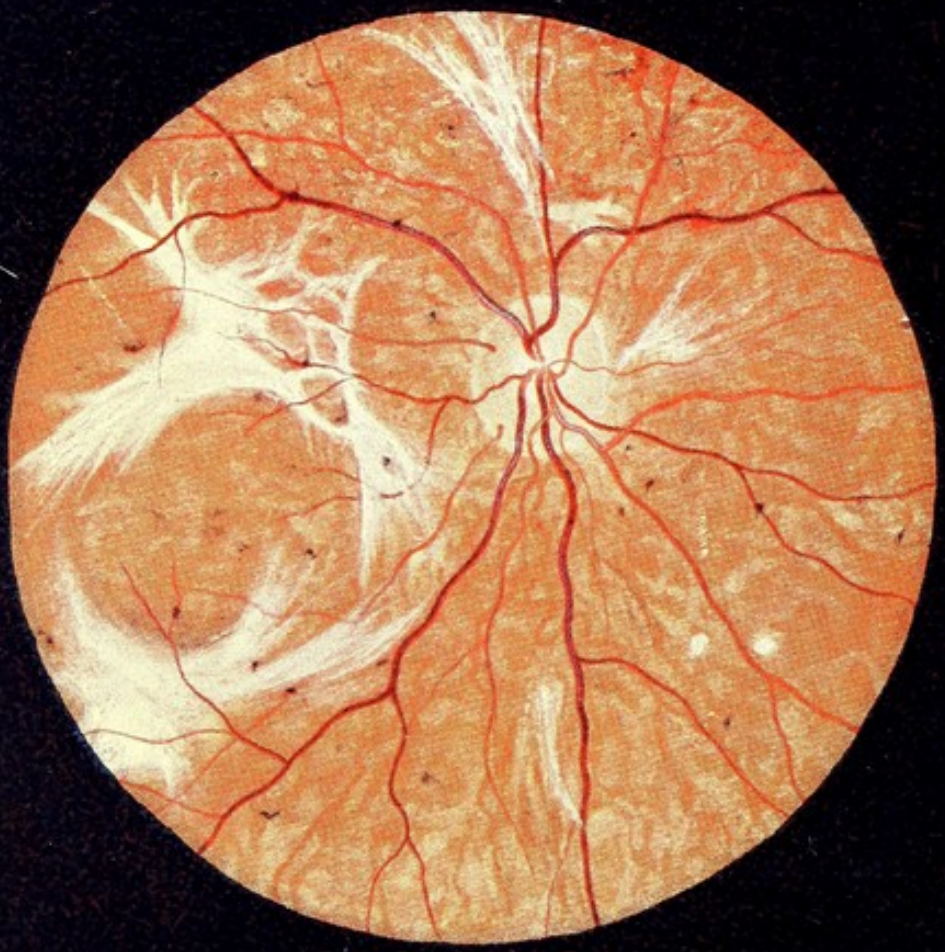

Fig. 33.

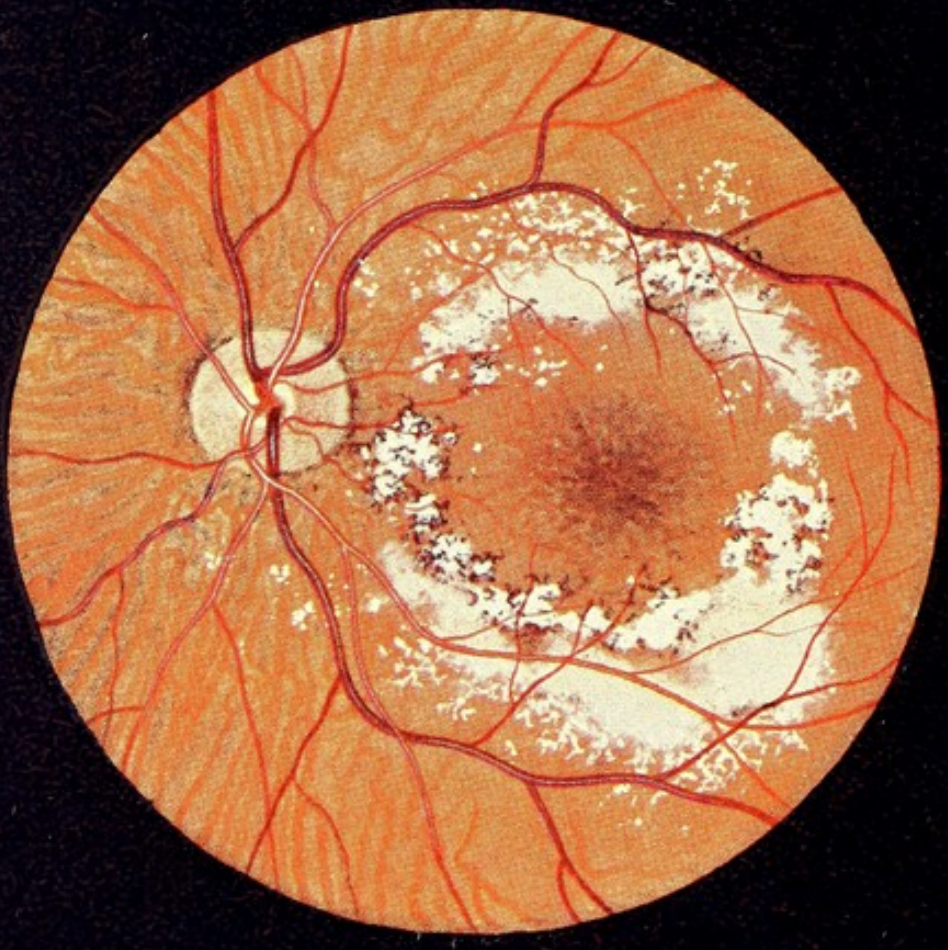

Fig. 34

A.W. Head

PLATE 18.

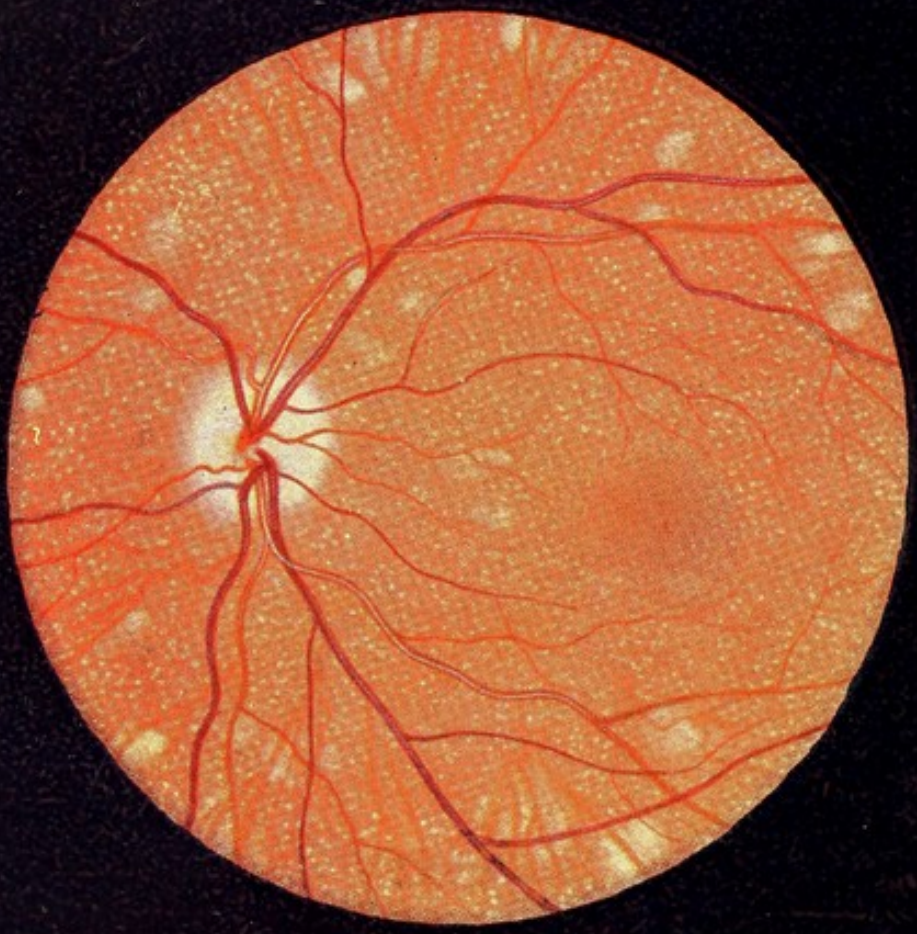

Fig. 35.

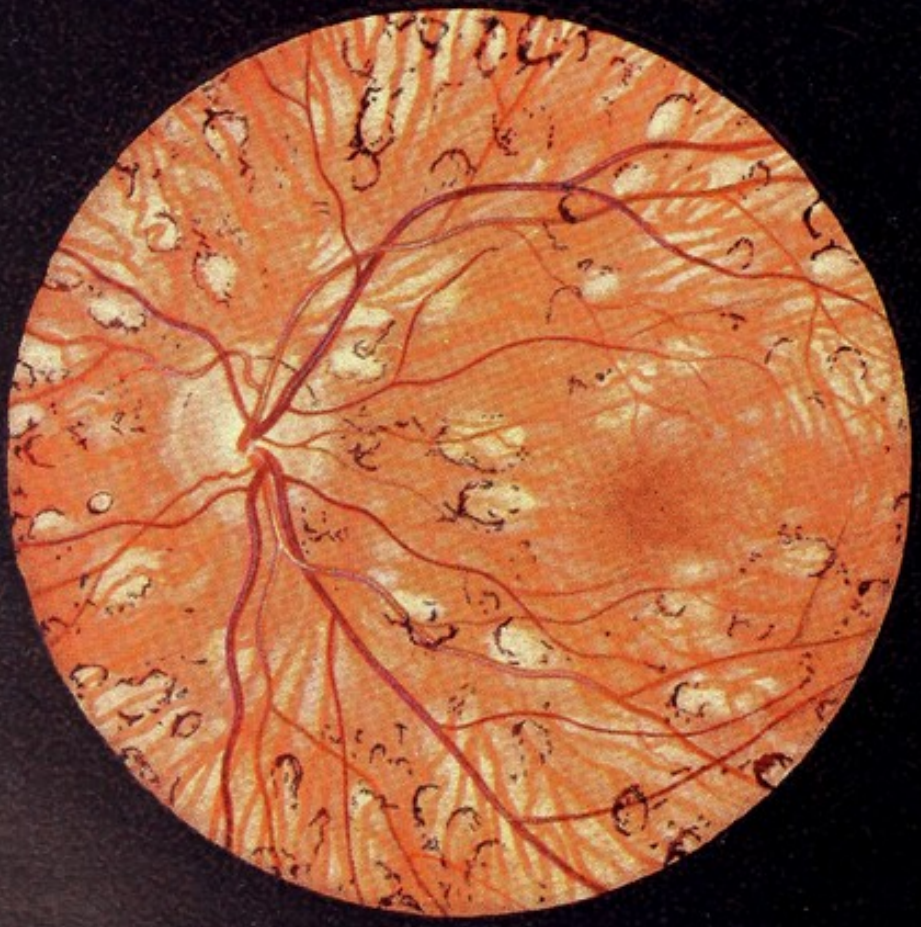

Fig. 36.

A. W. Head

PLATE 18.

FIG. 35.—CHOROIDITIS DISSEMINATA; STAGE OF EXUDATION (EARLY STAGE). LEFT EYE.

Man, aged 35 years. Patient came for advice complaining of difficulty of seeing objects after dusk, otherwise his field and vision was fairly good ($V = \frac{6}{12}$). The ophthalmoscope shows the disc to be hazy and ill-defined round the margin, which passes insensibly into the surrounding fundus. The retinal arteries and veins are normal and sharply defined. The whole background is covered over with minute, pale yellowish-white dots, the size of a small pin's head, when examined by the direct method. In addition to these are a number of pale yellowish-pink or whitish, circular spots or patches, each having about four or five times the diameter of the smaller dots The presence of these larger spots or patches will enable the student to distinguish this disease from a very similar, but entirely harmless, appearance known as *diffuse punctate condition of the retina*. The dots are due to a circumscribed, perivascular, cellular infiltration in the innermost layers of the choroid (chorio-capillaris). This produces an immense number of minute protuberances of inflammatory products (exudates), thus giving rise to the pale yellow spots or areas. These become specially prominent and conspicuous owing to the destruction of the membrane of Bruch and the hexagonal pigment layer which lies over them from defective nutrition. The outer layers of the choroid may remain unaffected for a long time, but the innermost layer invariably becomes attached to the retina by means of cicatricial adhesions over the affected or atrophied spots (Parsons). (P. 176.)

FIG. 36.—CHOROIDITIS DISSEMINATA; SAME EYE. LATER OR ATROPHIC STAGE.

The same eye examined eight years afterwards. The pin-head dots have all disappeared by absorption, and are replaced by a number of round or oval atrophic patches, each bordered by a crescent or circle of pigment. The hexagonal layer has become greatly thinned and has largely disappeared, thus allowing the choroidal vessels to become visible in every part of the field. The choroid is also becoming thinned and destroyed, so that the patches are lighter owing to the sclerotic shining through. This atrophy of the choroid may become complete over the patches, giving rise to the so-called "craters." The macula area stands out prominently as a deep red patch on the atrophic background. The disc margin is now better defined and is surrounded by a yellow buff-coloured zone. A still further atrophic stage will be found depicted in Fig. 38, and when the disease reaches the ciliary region a variety of complications may arise, leading to changes in the lens and lens capsule, and the formation of adhesions and degeneration of the vitreous, with consequent detachment of the retina, etc. (P. 176.)

PLATE 19.

FIG. 37.—CHOROIDITIS DISSEMINATA; ANOTHER CASE. INTERMEDIATE STAGE.

This drawing is typical of syphilitic disseminated choroiditis. The patient, aged 16, showed the usual signs of hereditary syphilis, viz. Hutchinson's teeth, syphilitic cachexia, remains of parenchymatous keratitis, etc. It will be noticed that there is a complete absence of pin-head spots, but numerous atrophic patches and crater spots can be seen. The floor of all the patches shows traces of pigment and scar-tissue. The macula area is slightly pigmented, but as yet not attacked by disease, although one patch is seen encroaching it. The outer (macula) quadrant of the disc is pale, and of a greyish colour. The media were clear, but the sight, $\frac{6}{24}$, was more defective than the patches would have led one to suppose, seeing that the macula was apparently healthy. Clinical experience shows, however, that the disease extends far beyond the boundaries of the patches; in fact, no part of the retina and internal layer of the choroid can be shown under the microscope to be really normal. In course of time the base of each of these patches may possibly undergo atrophy, becoming either covered up with pigment, or of a brilliant white colour, which is due to the sclerotic being exposed. It frequently happens that the disease has run its course at this stage, and beyond the spots becoming whiter, no further change will take place. This is commonly the case in hereditary syphilis. (Pp. 176, 177.)

FIG. 38.—CHOROIDITIS DISSEMINATA. ADVANCED STAGE OF ATROPHY.

This drawing shows a very advanced stage of choroiditis. The patient had contracted syphilis in early life. The entire fundus is honeycombed with atrophic patches, which allowed the white sclerotic to shine through unimpeded, save for the naked choroidal vessels. All the patches are bordered by abundant moss-like pigment, which is loosely scattered round about as well. The retinal arteries are small, almost thready. The retinal veins are slightly reduced in size. The vision, $\frac{6}{24}$, was surprisingly good considering the extent of the atrophy. Many of these cases remain quiescent for years, but occasionally the destructive process will suddenly be re-kindled, leading to a variety of complications, such as hæmorrhage, glaucoma, detachment of the retina, and opacities in the vitreous and lens, or adhesions between the capsule and the iris. (Pp. 176, 177.)

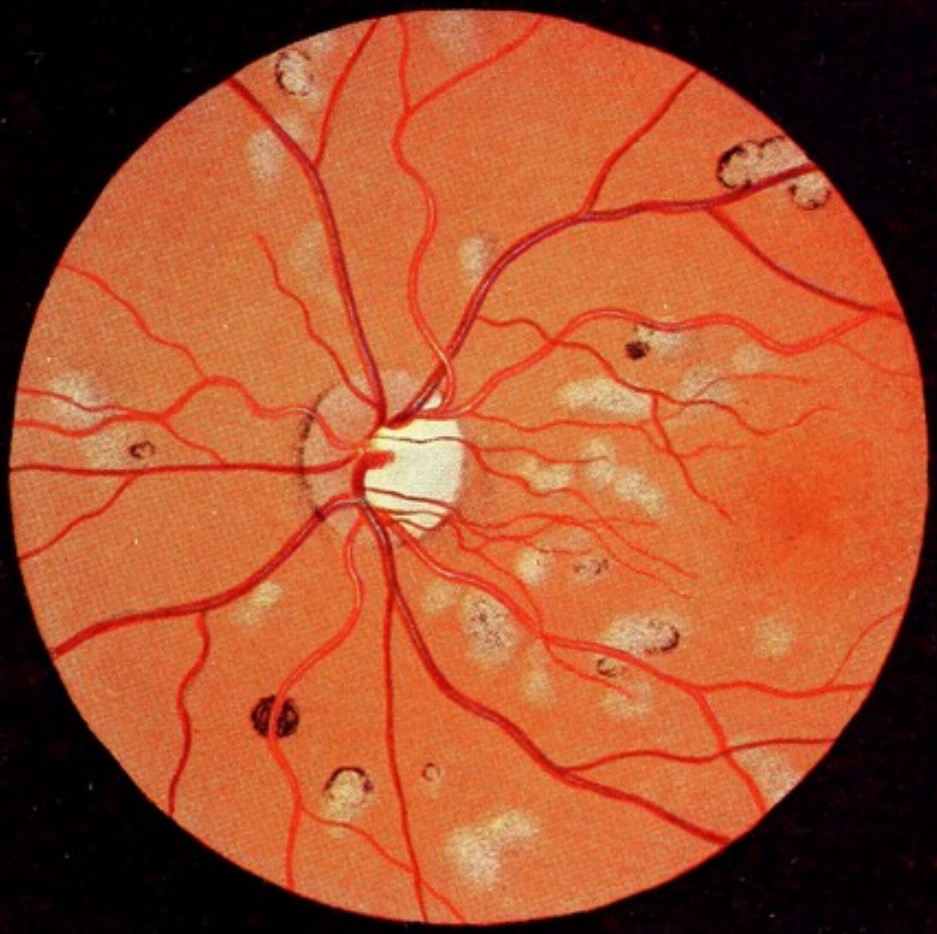

Fig. 37.

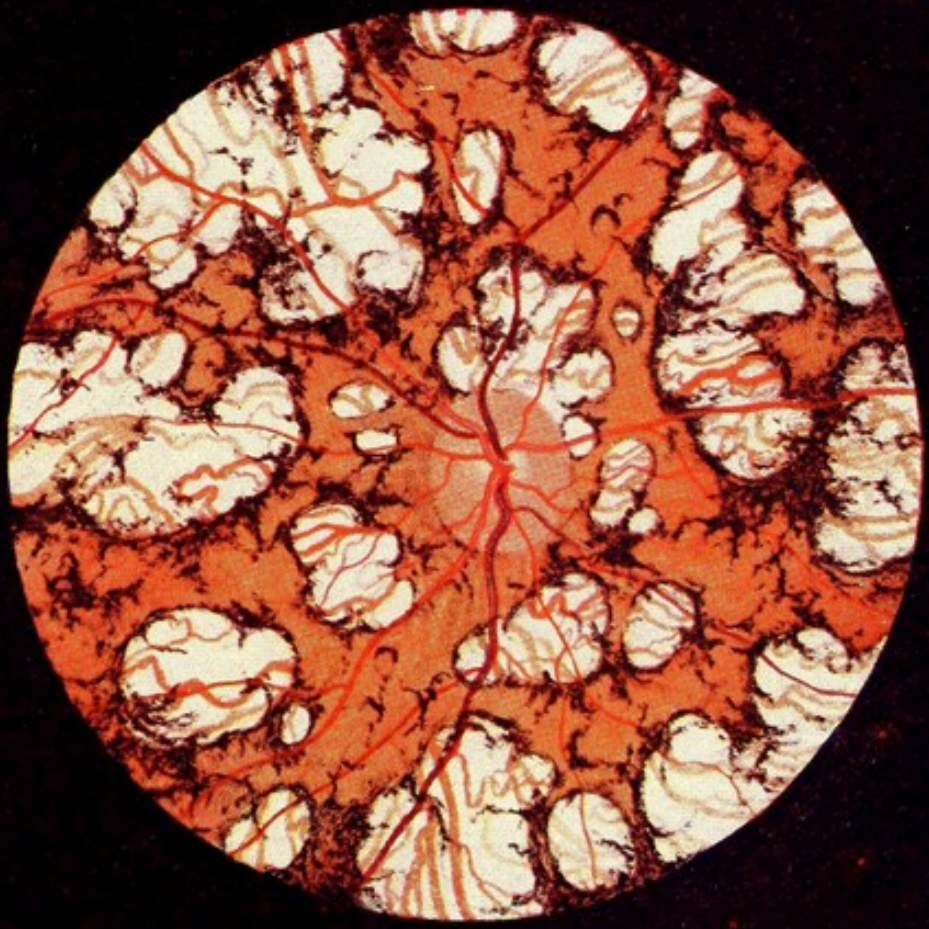

Fig. 38.

A. W. Head.

PLATE 20.

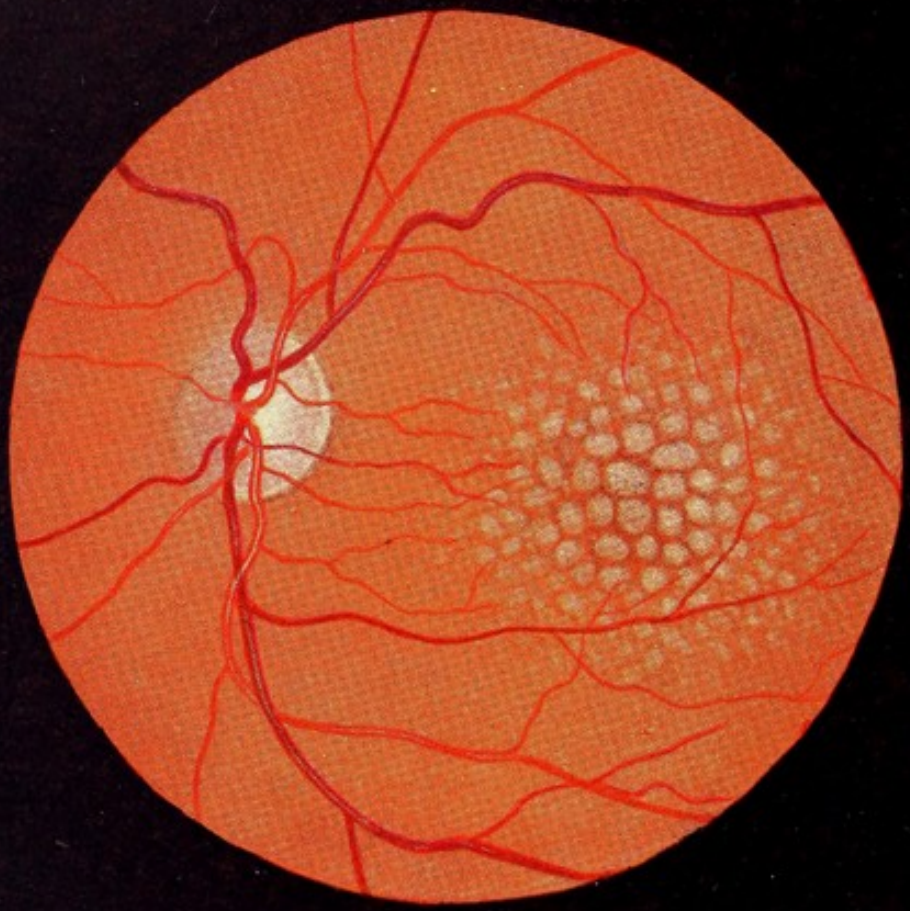

Fig. 39.

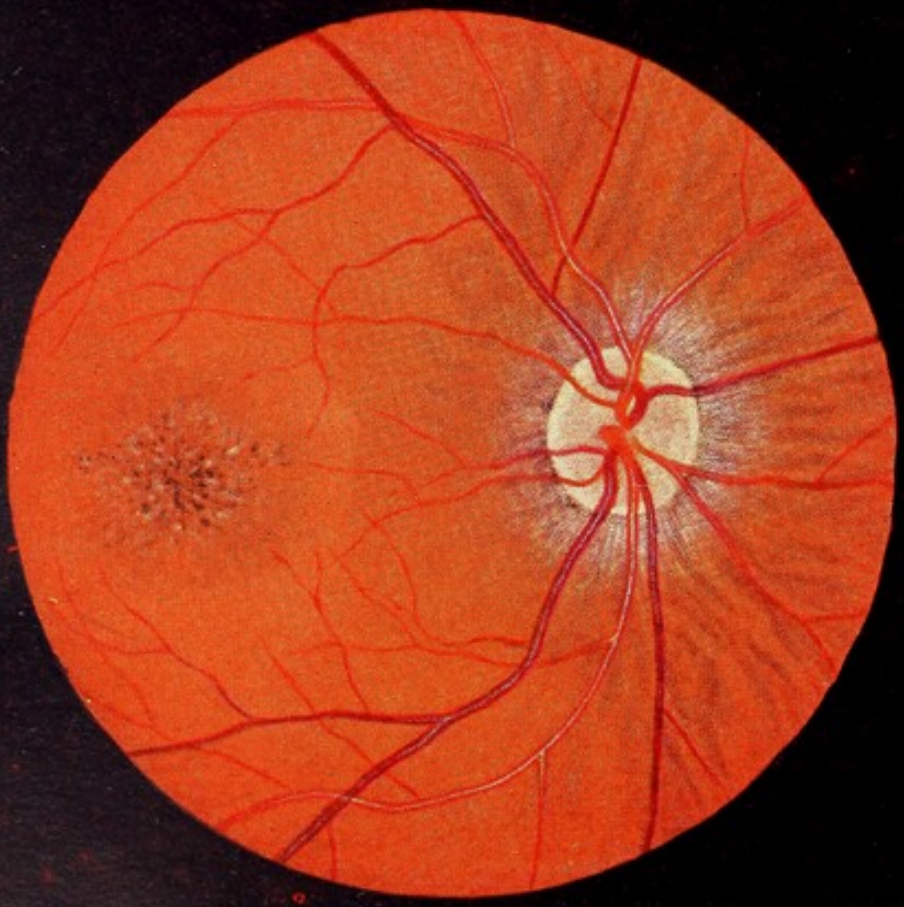

Fig. 40.

A. W. Head.

PLATE 20.

FIG. 39.—CHOROIDITIS OF THE MACULA AREA.

A. K—, aged 34 years, came for advice, complaining of a cloud in the field of view. V. = $\frac{6}{24}$. Field normal in extent. Partial central scotoma. Good colour vision. There was a history of acquired syphilis. Ophthalmoscopic examination revealed an agglomeration of oval spots, resembling cobblestones laid side by side. They appear to be from a fourth to an eighth the diameter of the disc, and are entirely confined to the macula area and a little distance all round it. The rest of the fundus is quite normal, as are the retinal vessels and disc.

The student must be careful not to confuse this disease with *Tay's choroiditis* (punctate retinitis), which consists of an agglomeration of pale, yellowish dots. The latter bodies may be recognised (1) by their softness of outline, (2) the fact that they are much smaller and more uniform in size, (3) the disease does not seriously affect the sight, and (4) the disease occurs in persons past middle life. These bodies are due to a colloid degeneration of the lamina vitrea, and have no connection with syphilis like macula choroiditis. (P. 180.)

FIG 40.—CENTRAL SENILE CHOROIDITIS (CHOROIDITIS GUTTATA).

This disease should be called central senile retinitis rather than choroiditis, for, as Harms has pointed out, the choroid is hardly affected at all, the changes being confined to the retina. This drawing was made from an old man, who came for advice owing to inability to obtain spectacles to enable him to read. There were signs of commencing cataract present, but insufficient to account for the falling off of his vision ($\frac{6}{36}$). The ophthalmoscope showed the whole macula area filled with small yellowish bodies, whose outlines were entirely hidden by pigment and local œdema and congestion. A faint striation radiating from the fovea could be seen on careful focussing. Otherwise the fundus was healthy. (P. 181.) A pathological change at the macula is extremely common in old people, and some alterations in appearance at this spot will almost invariably be found in those cases in which visual acuity cannot be brought up to $\frac{20}{30}$ by means of glasses, or in which a cataract present is insufficient to account for the falling off of acuity. The drawing (Fig. 40) is quite typical of a certain number of cases, but others assume different appearances.

PLATE 21.

FIG. 41.—TAY'S SYMMETRICAL DISEASE OF THE MACULA.

The patient, a child aged about 1 year, was lethargic and nearly, or quite, blind when examined. On ophthalmoscopic examination the retinal arteries appeared slightly thinner than normal, and the disc greyish-white. It is bordered by a thin layer of pigment. The choroidal vessels can be seen over every part of the fundus.

The macula is a very striking feature, appearing as a cherry-red spot about a fourth of the diameter of the disc and surrounded by a brilliant cream-coloured area, somewhat larger than the disc. Both eyes showed a similar appearance. The child died a few months afterwards from an obscure cerebro-spinal affection. The appearance of the macula strikingly resembles that produced by an embolism of the central artery except that the affected area is much less. (P. 167.)

FIG. 42.—DIABETIC LIPÆMIA.

The ophthalmoscopic appearance of this rare disease is very striking and characteristic. The entire fundus has a peculiar salmon-pink colour which is evidently due to the fatty-like granules in the blood. The retinal veins and arteries are both larger than normal and almost indistinguishable from each other. They are not tortuous or varicose as one might suspect. They have a peculiar buff-straw colour, becoming pink near and at the disc, where the central streak is in evidence. The disc has a deep grey colour quite different from that seen in any other disease. The macula region is mottled and of a deep shade of red pink. Traces of the choroidal vessels can be traced over the entire fundus. (P. 151.)

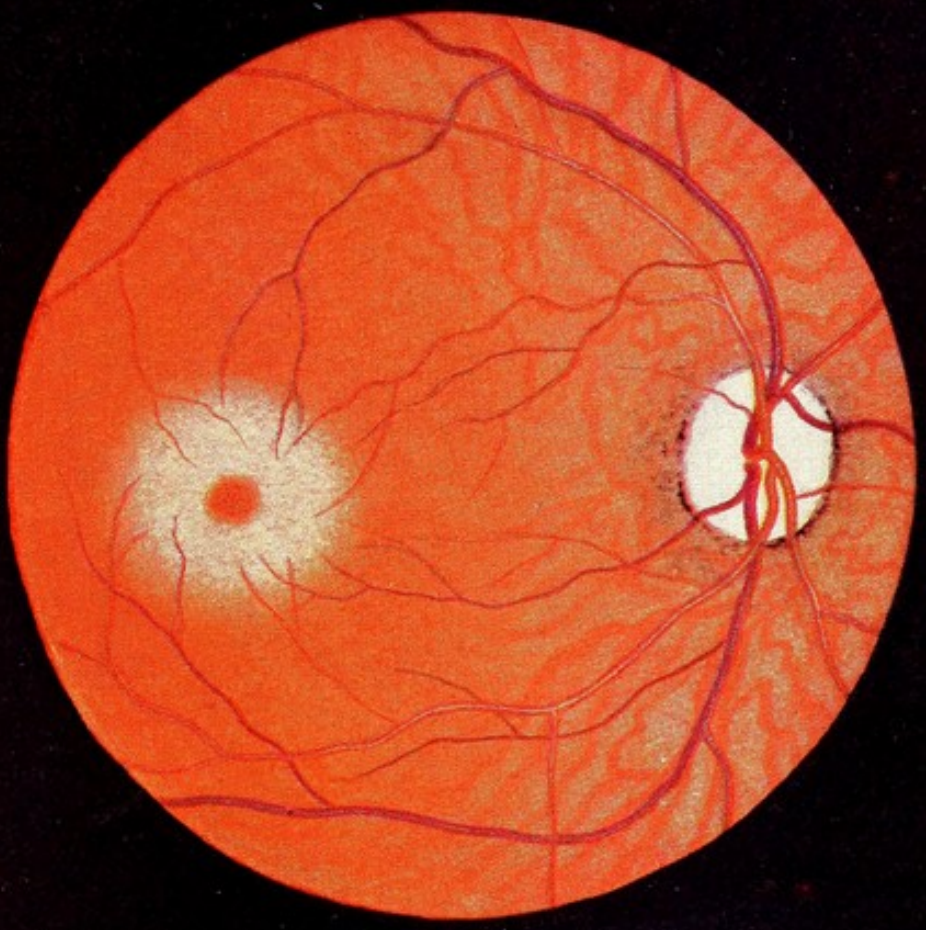

Fig. 41.

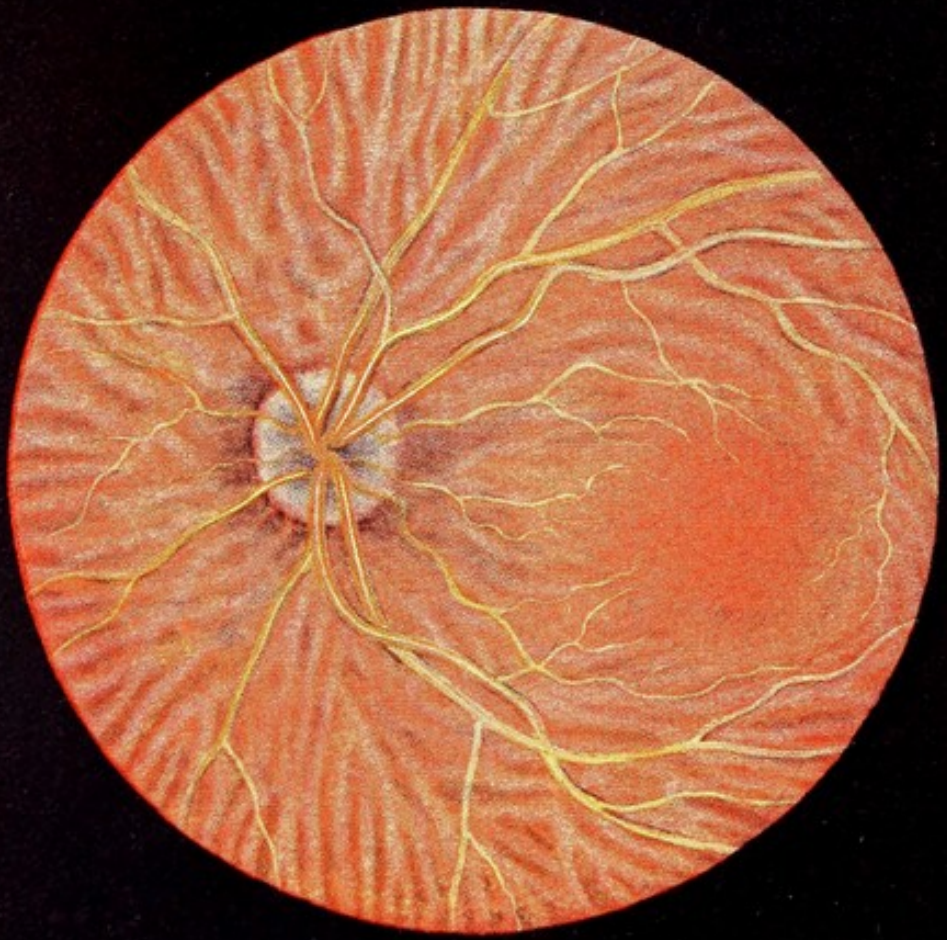

Fig. 42.

A. W. Head.

PLATE 22.

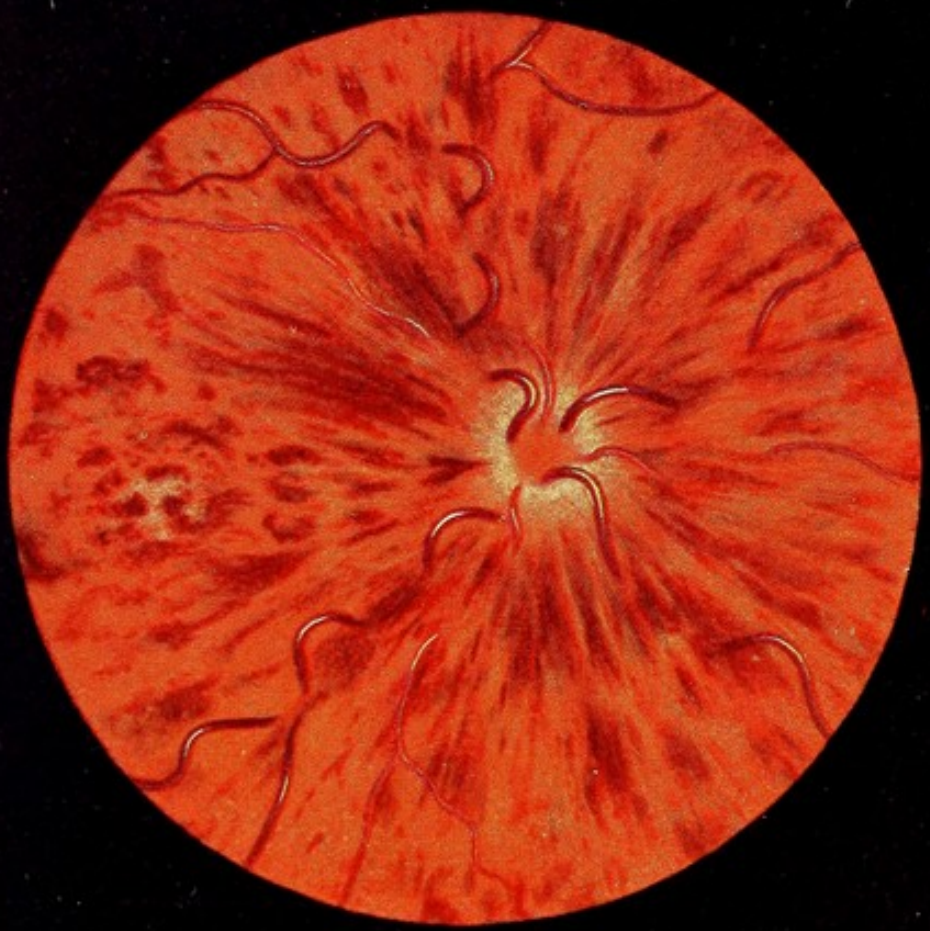

Fig. 43.

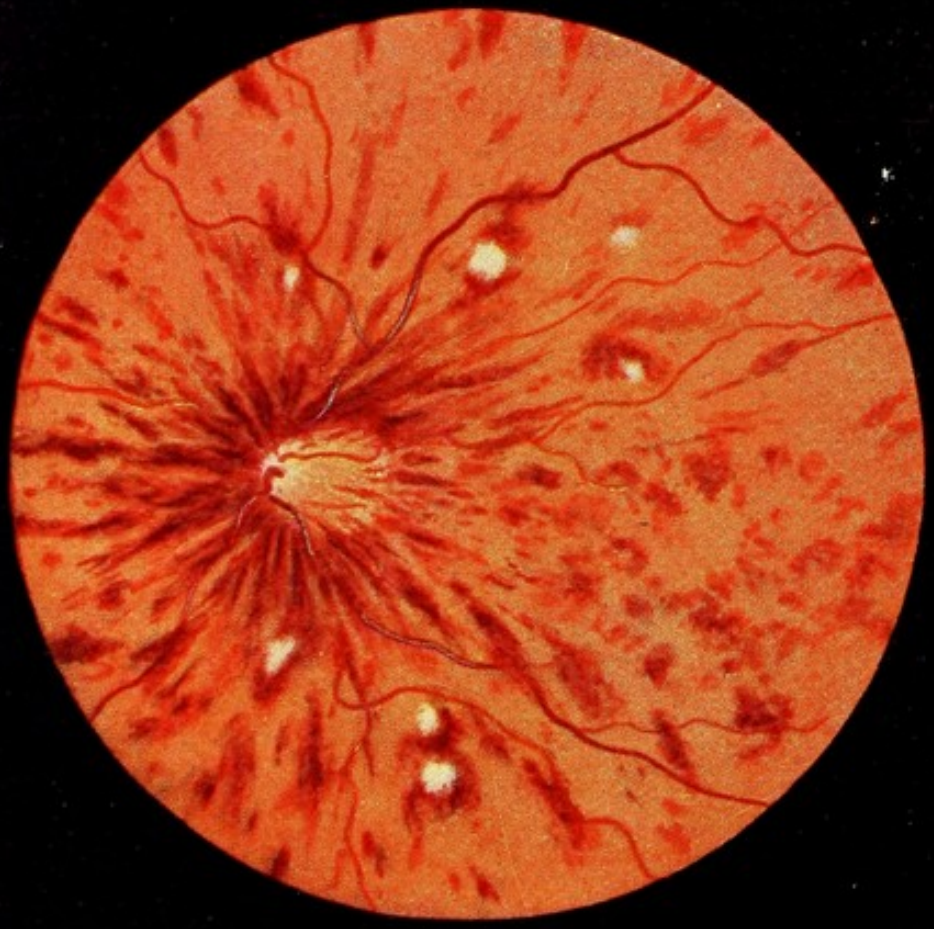

Fig. 44.

A. W. Head.

PLATE 22.

FIG. 43.—THROMBOSIS OF CENTRAL VEIN.

This drawing was made from a man suffering from advanced kidney disease. V. = P.L. only. The other eye showed some hæmorrhage but was not otherwise affected and the sight was good. The ophthalmoscope shows a swollen, highly inflamed disc in which all details and outlines are lost. The veins are swollen and greatly increased in calibre and extremely tortuous, dipping in and out of the red background in bold curves. The arteries form mere threads and are only slightly tortuous. The entire fundus appears to be splashed with blood, especially along the nerve-fibres, which radiate in every direction from the papilla. The macula can be recognised by the peculiar arrangement of the blood-clots, which form a series of broken and imperfectly formed circles round the fovea. Occasionally only one of the veins gets plugged, in which case the retinal changes are limited to its area of distribution. (P. 167.)

FIG. 44.—THROMBOSIS OF CENTRAL VEIN. LATER STAGE.

This shows a partial recovery of a similar case. The veins have resumed their normal calibre and direction, the tortuosity having almost entirely disappeared. Many of the hæmorrhages have disappeared, in a few places leaving white spots of atrophic choroid due to the sclerotic shining through. (P. 167.)

Diagnosis between Thrombosis of the Central Vein and Embolism of the Central Artery.

Thrombosis of central vein.	*Embolism of central artery.*
Veins engorged, often enormously distended.	Veins normal in size.
Veins excessively tortuous, dipping in and out of the retinal tissue.	Veins not tortuous and never lost to view.
Veins pulsate on pressure.	No pulsation.
Hæmorrhages very extensive.	No hæmorrhages.
Disc swollen, œdematous.	Disc not swollen or œdematous.
Fundus vivid red and splashed with hæmorrhages.	Fundus creamy white over central parts.
Macula buried in œdema and hæmorrhages.	Macula forms a cherry-red spot on cream-coloured ground.

PLATE. 24.

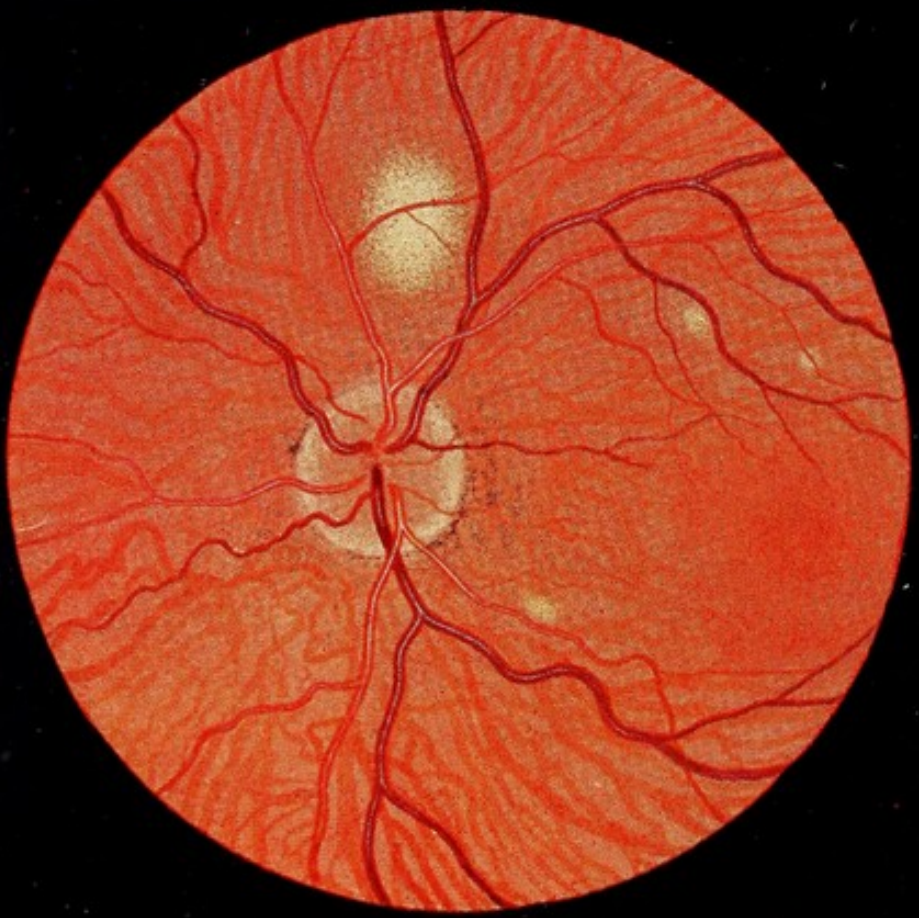

Fig 47

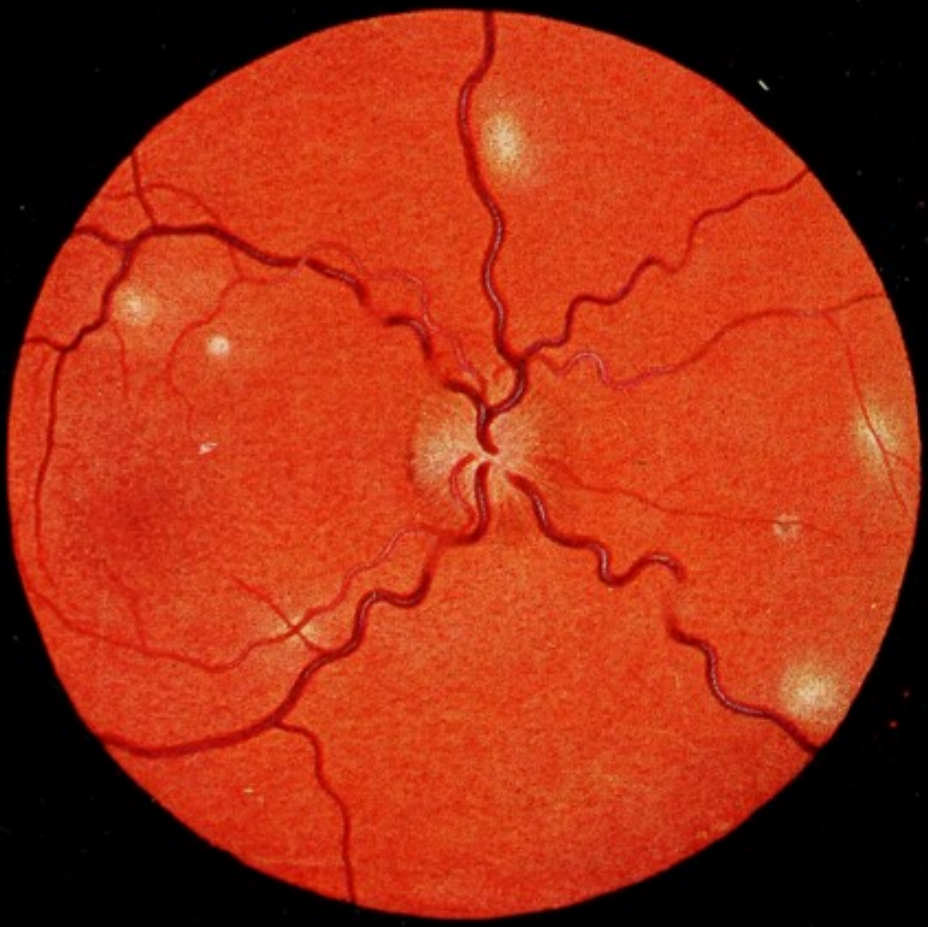

Fig 48

A W Head

PLATE 24.

FIG. 47.—MILIARY TUBERCLES OF THE CHOROID.

From a child, aged 2 years. The disc is clearly defined, but somewhat congested and œdematous. The retinal vessels, especially the veins, appear slightly tortuous in places. The macula is of a darker red than normal. Around the disc four oval yellowish-white nodules, one or two having a faint pinkish margin, can be seen. These nodules or patches are slightly raised above the surface. This is shown by the retinal vessels being distinctly curved where they pass over the nodule. The margins of the nodules are hazy, and fuse, as it were, into the general background. In this particular case the choroidal vessels are everywhere distinctly seen. The child only survived a few weeks. (P. 178.)

FIG. 48.—MILIARY TUBERCLES OF THE CHOROID. ADVANCED CASE.

In this drawing, which was made only a few hours before the death of the child, the papilla is seen to be congested, œdematous, and striated; in fact, a typical case of acute papillitis. The macula is also œdematous and congested, and the retinal vessels, the veins more especially, very tortuous and swollen. A number of tubercular nodules can be seen scattered over the fundus. The eye was quite blind and the pupil widely dilated.

Diagnosis.—The only other disease which it is likely to be mistaken for is an early stage of choroiditis disseminata. As a rule, however, in this latter disease one or more of the spots towards the periphery of the field will be found partly encircled by a border of pigment, giving rise to the so-called "craters," which are highly characteristic of it, whereas the tubercles have generally a pinkish border. (See Fig. 36 and pp. 176, 177.) Furthermore, the child's general condition, the pasty expression of the face, the absence of fever and the family history will generally suffice to establish the diagnosis. In cases of doubt both v. Pirquet's inoculation test and Wassermann's hæmolytic reaction should be made. (See Appendix.)

PLATE 25.

FIG. 49.—EXTENSIVE COLOBOMA OF THE CHOROID.

This drawing is typical of the appearance of an unclosed choroidal cleft which extends either directly downwards or obliquely downwards and outwards. In some cases the cleft can be traced nearly as far as the iris, in which also there is a gap, indistinguishable from a perfectly made artificial iridectomy. Observe that both the choroid and retina are completely absent over the whole of the cleft, the background consisting of sclerotic only. The margins of the cleft are bordered by abundant mossy pigment, and the retinal vessels pass over the cleft in a perfectly normal manner. If the coloboma can be traced with the ophthalmoscope sufficiently far forwards it will be generally found to terminate abruptly in a rounded extremity, the tissue beyond (as far as the ora serrata) consisting of all three coats, and identical in colour and structure with the rest of the normal fundus. Many of these cases can be traced to some intra-uterine disturbance, and the coloboma itself is due to a failure of completion in the development of the mesoblast and the closure of the fœtal cleft. Choroidal colobomata are very commonly to be found in microphthalmic (undeveloped) eyes. (Pp. 112–114.)

FIG. 50.—RUPTURE OF THE CHOROID.

The patient was firing a gun, when the cap blew off and struck his eye. He was quite blind for some hours afterwards, and on examination the next day the vitreous was seen to contain blood. As this cleared up during the ensuing week a clot of blood was observed on the outer side of the disc. This ultimately absorbed and left the eye permanently in the condition depicted. A whitish fork-like crescent is seen partly encircling the outer part of the disc. The rest of the fundus is normal; the white of the crescent is due to the rupture and shrinkage or atrophy of the choroid over the part, allowing the sclerotic to shine through. In the above case the sight returned to normal and remained so. In some cases, however, the eye does not heal perfectly, but after a longer or shorter time macula changes supervene accompanied by serious diminution of visual acuity. (P. 184.)

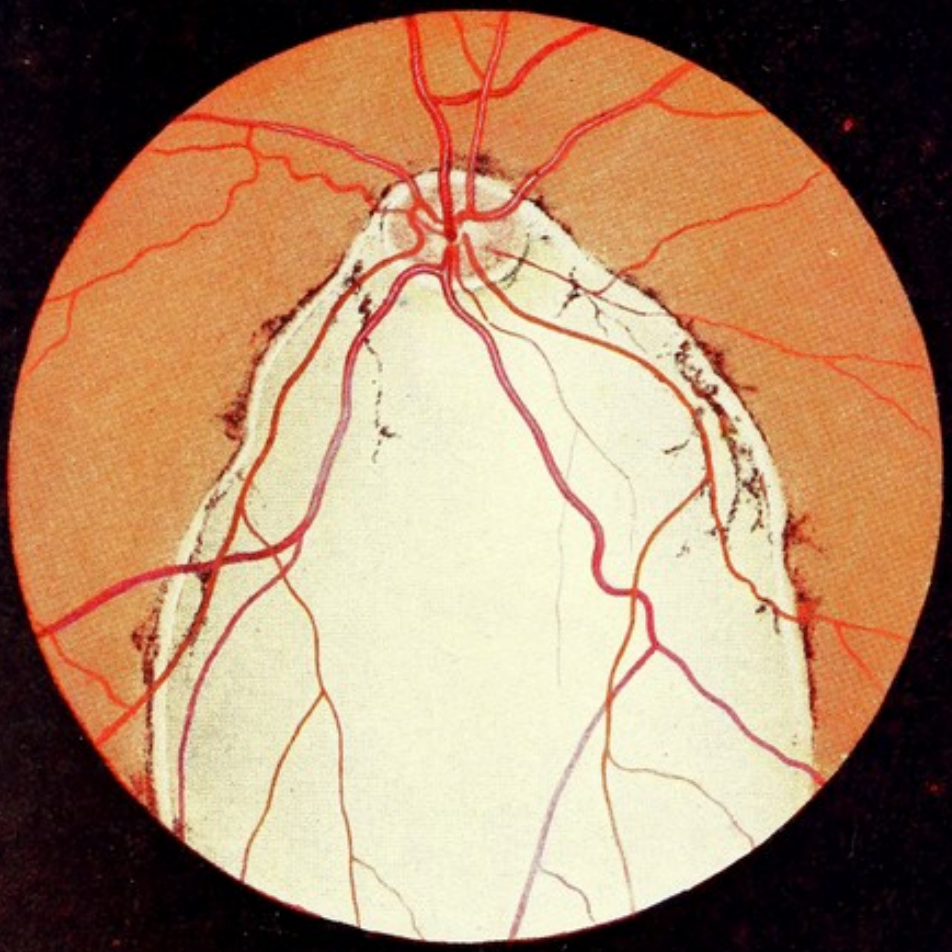

Fig. 49

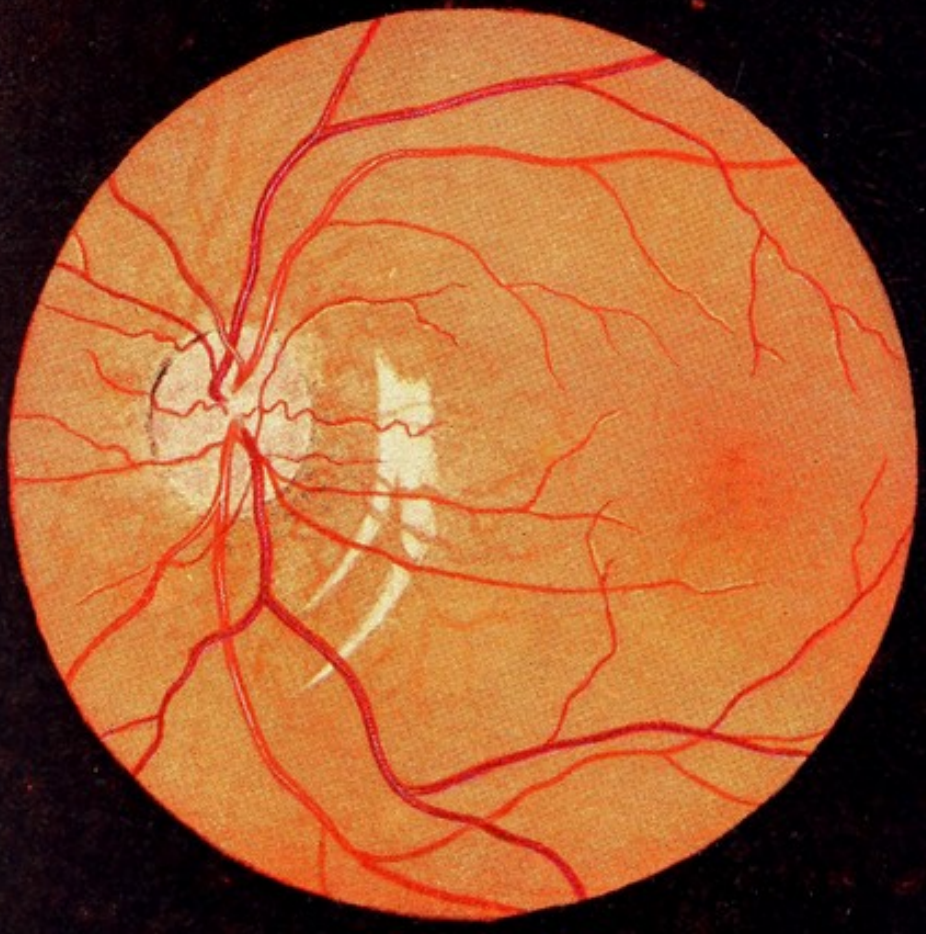

Fig. 50.

A. W. Head

PLATE 26.

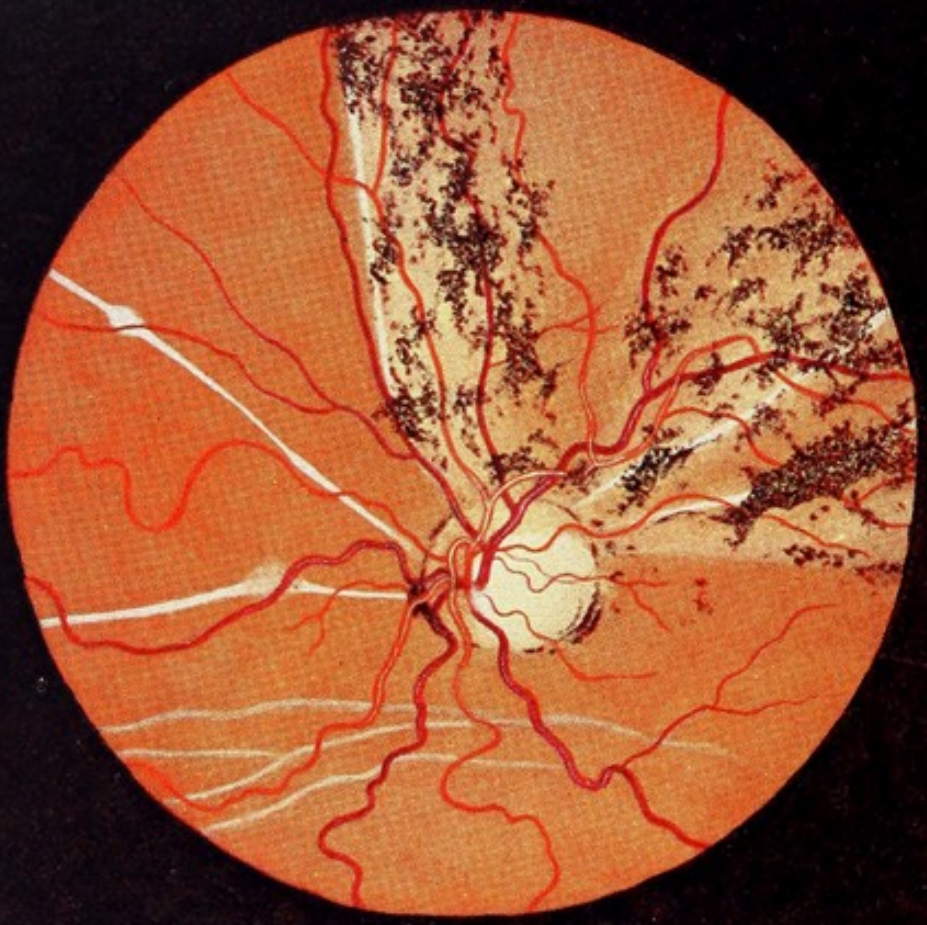

Fig. 51

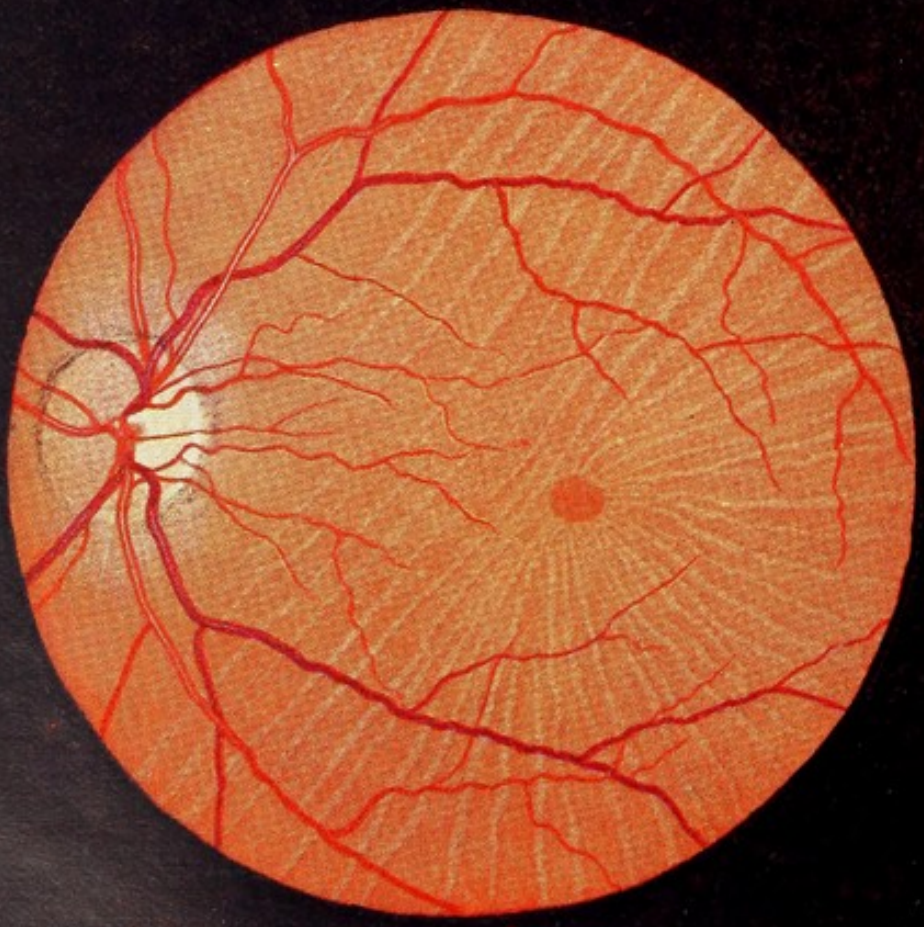

Fig. 52.

A. W. Head.

PLATE 26.

FIG. 51.—DETACHMENT OF THE RETINA. CICATRICIAL BANDS ON THE FUNDUS. LEFT EYE.

The patient was a man, aged 54 years. His eye had long been defective, but he came for advice as he noticed the upper part of the visual field was becoming more defective. The ophthalmoscope showed four horizontal linear folds of retina, which formed grey-looking lines of detachment. Above and to the right and left of the disc a number of white, thick bands of scar-tissue could be seen stretching from the disc in straight lines to the periphery. A large quantity of pigment could be traced over and around the retinal vessels and several of the cicatricial bands in the upper and outer quadrant of the eye. These bands, by their contraction, were the probable cause of the detachment. (See pp. 159–163, where the whole question is discussed.)

FIG. 52.—EXCAVATION OF THE MACULA AND COMMENCING DETACHMENT OF THE RETINA.

The patient, a girl, aged 19 years, was struck on the eyeball with the brim of a straw hat. A few hours afterwards she noticed everything appeared as if seen through a thick mist. On examination with the ophthalmoscope the next day the central part of the fundus appeared thrown into faint ripples. The retina between the macula and the disc is raised in fine parallel lines, that part which lies below and to the outside being thrown into similar folds, which radiate from the macula. The latter appears as if the retina had been punched out at that spot, leaving the inflamed choroid to form the base. There can be no doubt that the retinal layers are wanting, and that there is an actual hole merely lined or covered over by a layer of delicate connective tissue. Both the folds of detached retina, which radiate from the macula and the macula gap itself, appear to be due to the œdema set up by the inflamed choroid behind them. The effusion and exudation provoked by a blow, or foreign body in the eye, or by chlorosis, tuberculosis, and various acute specific fevers, push forward the delicate retina at the fovea (which here only consists of two layers, viz. cones and bi-polar cells) and easily rupture it. Once ruptured it retracts on all sides by virtue of its elastic fibres, and the rent becomes a large hole, while the effusion, seeking lines of least resistance, forms the radiating folds of detached retina shown in our illustration. (See p. 159, *et seq.*, also pp. 168, 169.)

PLATE 27.

FIG. 53.—DETACHMENT OF THE RETINA ABOVE THE DISC (EARLY STAGE).

A typical case of slight detachment. Numerous lines or flame-shaped folds of pale, greenish white, partially bleached retina may be seen forcing their way downwards towards the disc. The fundus behind the area of detachment is paler and more of an orange pink than at the healthy area. (Pp. 154–157.)

FIG. 54.—A MORE ADVANCED AND OLDER CASE OF DETACHMENT.

The case was one which began spontaneously. The detachment crept down to the lower part of the eye by gravity acting on the subjacent fluid. The retinal folds are very large and conspicuous, and the retinal vessels appear wavy and of a dark red or chocolate colour. In this figure folds of retina can be seen at three different levels in front of the unaffected retina, which can be seen (out of focus) in the upper part of the drawing. The chocolate colour of the vessels is a very characteristic sign of detachment. When the retina is completely detached over a given area it becomes, after a time, bleached by the light, and assumes a white or greenish-white colour. Where this has taken place its functions are destroyed and they can never be renewed. If, however, the retina be only partially detached, provided that bleaching has not taken place, its functions can be completely restored by causing it to come in contact with the hexagonal pigment layer and through it with the subjacent choroid. (Pp. 154–157; see also pp. 75, 76, *et seq.*)

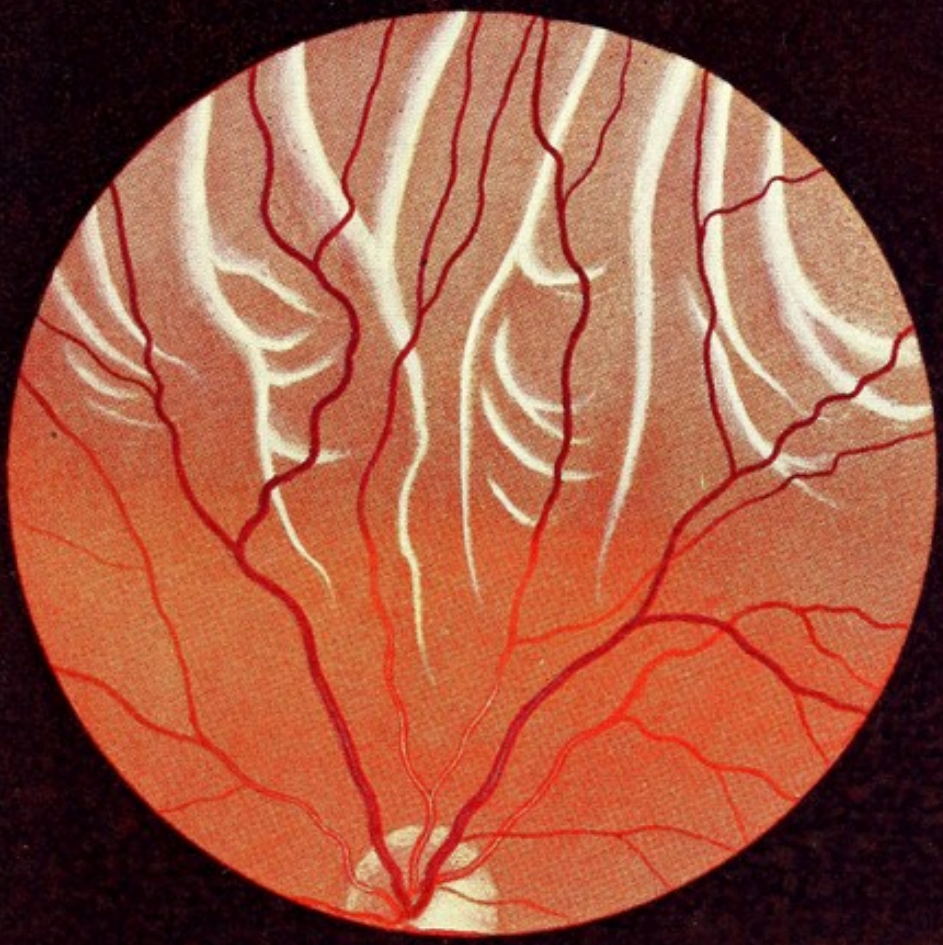

Fig. 53.

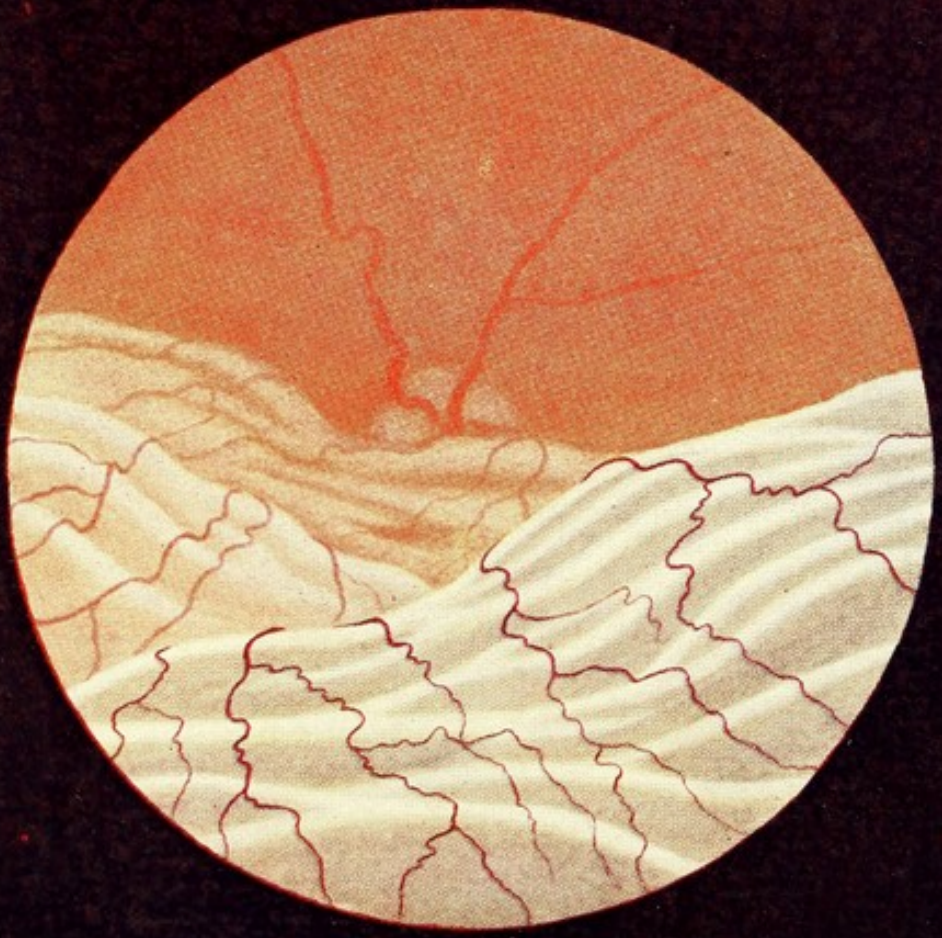

Fig. 54.

A. W. Head