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A MANUAL AND ATLAS

OF

MEDICAL OPHTHALMOSCOPY

By the same Author.

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A

MANUAL AND ATLAS

OF

MEDICAL OPHTHALMOSCOPY

BY

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FOURTH EDITION

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LONDON
J. & A. CHURCHILL
7 GREAT MARLBOROUGH STREET
—
1904

Pardon & Sons, Printers, Wine Office Court, Fleet Street, London.

PREFACE TO THE FOURTH EDITION.

In preparing the Fourth Edition, as with the Third, I have had the advantage of the help of Mr. Marcus Gunn. When this book was written, twenty-five years ago, the subject with which it deals was more familiar to physicians who constantly used the ophthalmoscope, than to ophthalmic surgeons. But the position of the two has been to some extent reversed during the years which have elapsed, in consequence of the sedulous use made of the opportunities for the investigation of medical cases by those who devote themselves to the study of the eye. Hence it is, I believe, a fact that such a book as this, at the time it appeared, could not have been produced by any other than a physician, but cannot now be kept abreast of general and special knowledge except by the conjoint efforts of a physician and an ophthalmic surgeon. I consider myself and the reader fortunate in the collaboration of Mr. Marcus Gunn, whose experience has been wide in range and wisely used.

In the present edition such additions have been made as are needful to represent adequately current knowledge. The autotype plates, reduced facsimiles of sepia drawings, have been felt to be so far more true to nature than the chromolithographic illustrations, that the latter have been omitted. The absence of colour is more than compensated for by the attention, which its absence compels, to the more important elements of form. One or two illustrations from the omitted plates have been reproduced as figures in the text. The reader is advised to study the plates with the aid of the descriptions prefixed to them.

The ophthalmoscopic illustrations, both in the plates and in the text, and the microscopical figures, of which the source is not stated, are reproductions of my own drawings.

WILLIAM R. GOWERS.

50, Queen Anne Street,
London.

CONTENTS.

DESCRIPTION OF THE PLATES.

MEDICAL OPHTHALMOSCOPY.

INTRODUCTION.

THE ophthalmoscope is of use to the physician because it gives information, often not otherwise obtainable, regarding the existence or nature of disease elsewhere than in the eye. This information depends upon the circumstance that we have under observation—1. The termination of an artery and the commencement of a vein, with the blood circulating in each. 2. The termination of a nerve, which, from its close proximity to the brain, and from other circumstances, undergoes significant changes in various diseases of the brain, and in affections of other parts of the nervous system. 3. A nervous structure—the retina, and a vascular structure—the choroid—which also suffer in a peculiar way in many general diseases.

For the efficient use of the ophthalmoscope in medical practice, the student must be familiar with the use of the instrument; he must also be familiar with the normal fundus oculi, with the changes in its appearance (congenital and other) that are of no clinical significance, and also with those that are ocular in origin, such as posterior staphyloma, glaucomatous excavation, and the like. An acquaintance with these must be gained from the ophthalmic surgeon before inferences can safely be drawn regarding the significance of other alterations met with in various diseases. The following pages assume the possession of a general knowledge of the use of the instrument, but a few words regarding some points which are of special importance may be of service.

A first requisite in medical ophthalmoscopy is familiarity with the direct method of examination. The disc is then

seen magnified many times; and this method may show minute changes of the highest significance, which cannot otherwise be detected, or the true nature of appearances which, seen by the indirect method, are obscure. But both methods should always be employed. Not only has each its special advantage, but the two together often give information which neither alone affords.

Another requisite is skill in the examination without dilatation of the pupil. In most eyes much can be seen with the pupil undilated—often all that is necessary, and almost always enough to determine whether or not there is more to be learned by dilatation. The coincident paralysis of accommodation is a source of annoyance, especially when there is no disease of the eye itself. If the sight has not been previously affected, a subsequent failure of sight, due to neuritis, atrophy, &c., is often ascribed by the patient, not unnaturally, to the effect of the mydriatic.[1] For the same reasons one pupil only should be dilated at a time, unless the sight of both eyes is already impaired. If it is a matter of indifference which is chosen, an eye the sight of which is impaired should be preferred. These disadvantages have been lessened by the use of homatropine, since the paralysis of accommodation passes off in a few

[1] "If we use the ophthalmoscope, or if we use atropine, or if we apply a blister to the head, or adopt any new kind of treatment, the patient may blame us for his blindness, if he saw well before such procedures. A patient who reads the smallest print and supposes his sight to be good, may have double optic neuritis. The use of atropine affects his sight for near objects gravely, and if, from the advance of the neuritic process, what I may call retinal sight fails before the effect of the atropine has passed off, he very naturally blames us for the subsequent permanent affection of his sight. A patient, when asked how long his sight had been bad, replied, 'Only since the drops had been put in.' We must, then, when we discover neuritis, sight being good, tell the patient that his eyes are not really good, and that we are anxious about his sight. Whether we give this warning or not, we shall be blamed by an unintelligent patient for 'tampering with his eyes.' We must, however, act for our patient's good, regardless of selfish considerations. In very many cases we can see enough for diagnostic purposes without using atropine"—Hughlings-Jackson Lectures on Optic Neuritis, "Med. Times and Gaz.," September 16, 1871.

hours, and the dilatation of the pupil seldom persists more than a day. Cocaine is likewise a useful mydriatic, on account of the short duration of its effects, and from the facility with which they yield to eserine. Its use is particularly indicated where there is any danger of exciting increased tension in the eyeball by ordinary mydriatics.

In making an examination with the ophthalmoscope, it is best to look at the eye first from a distance, without an intervening lens, in order to ascertain whether the red reflection from the fundus is clear. This at once gives information regarding the presence or absence of opacity of the lens or vitreous, or may reveal iritic adhesions—conditions which convey important information, and explain what would otherwise be a puzzling obscurity of detail. Next, the refraction of the eye should be roughly estimated by observing if the vessels of the retina can be distinctly seen from a distance, and, if so, whether they move in the same direction as the observer's head (hypermetropia), or in the opposite direction (myopia). The knowledge thus gained is important, since in myopia the details of the fundus, when seen with a lens by the indirect method, seem small, and in hypermetropia large. If necessary, the refraction may be more accurately ascertained by the direct examination: the lens needed to correct it, if the observer's refraction is normal, indicates the degree of error.

It is frequently necessary to examine patients in bed. The indirect method of examination can be applied as readily to a patient in bed as to one sitting on a chair, the most convenient place for the light being above the patient's head. Even in daylight little difficulty is experienced unless the pupil is small, but the examination is facilitated by a screen of some kind, even by the shade of an umbrella. The direct method presents more difficulty; a convenient position is at right angles to a patient in bed, with the lamp near the top of the patient's head.

All who have employed the ophthalmoscope in medical practice will agree with Hughlings-Jackson in urging the routine use of the instrument in all diseases in which

ophthalmoscopic changes are, even occasionally, met with. It often happens that unexpected information is gained regarding the nature of the disease, or its probable consequences.

It has been remarked that the medical ophthalmoscopist should possess familiarity with those changes in the eye which are of purely ocular significance. It is of equal importance that he should be familiar with those congenital changes in the eye which are of no clinical importance. Many of these will be alluded to in describing the morbid appearances with which they are most liable to be confounded. One or two, which give rise to special trouble to the beginner, may be here briefly mentioned. One of these is the variation in the colour of the optic disc. It has been well remarked that the tint of the optic disc may vary as much as the tint of the cheek. It is always redder in the young than in the old, except in early infancy. In the old the redness has often a grey tint mingled with it. In the young the tint may even be scarcely, or not at all, paler than that of the adjacent choroid. When the choroid is bright in tint, the apparent redness of the disc is increased by indirect examination with a wide pupil and a bright light, and is a very frequent source not only of error in diagnosis but of scientific mistakes. It is the sharpness of the edge of the disc to which attention should be especially directed, as evidence of morbid change.

When the physiological cup is very large, the vascular portion of the disc is confined to a narrow rim at the side, often much narrower than that shown in Pl. I. 1, which represents a large, but not very large, cup. When the part of the disc occupied by the nerve fibres is reduced to, say, one-half of that shown in the figure, the fibres are so crowded together that the choroidal limit is often less distinct than normal, and the central white cup may be mistaken for the disc, the edge being regarded as part of the fundus. Knowledge of this danger, however, will be sufficient to prevent an attentive observer from falling into this error; there is no confusion on direct examination.

White patches near the disc, due to choroidal atrophy and to opaque nerve fibres, sometimes present puzzling appearances (Fig. 1). The recognition of choroidal atrophy by the greyish-white tint of the sclerotic, by the pigmentary disturbance, and by the comparative absence of change in the retinal vessels, is usually one of the first points learned. Now and then a narrow posterior staphyloma may surround, or almost surround, the disc, and its edge may be mistaken for the edge of the disc, which seems white with a red centre. (It is well to remember that posterior staphyloma is common in myopic eyes, and also that a somewhat similar appearance may be seen occasionally in hypermetropic eyes.)

The white patches of opaque nerve fibres (such as are shown in Fig. 1) are characterised by their position, adjacent to the disc; by the peculiar shape of the spot, which, if large, follows the course of the nerve fibres; by the partial concealment of the vessels; the feathery edge; and by the centre of the disc being commonly unconcealed. When a small patch lies near, but separated from the disc,

FIG. 1.—OPAQUE NERVE FIBRES.
Surrounding optic disc, and concealing the vessels in that neighbourhood.

the resemblance to an inflammatory exudation may be very close; the characters of its edge, the obscuration of the vessels, and the absence of other changes, will usually enable its nature to be recognized.

Peculiar white films sometimes lie in front of the vessels on the disc, looking like fragments of tissue paper or white gauze, and allowing the vessels behind to be dimly seen. These are usually congenital, the relic of tissue at the back of the vitreous.

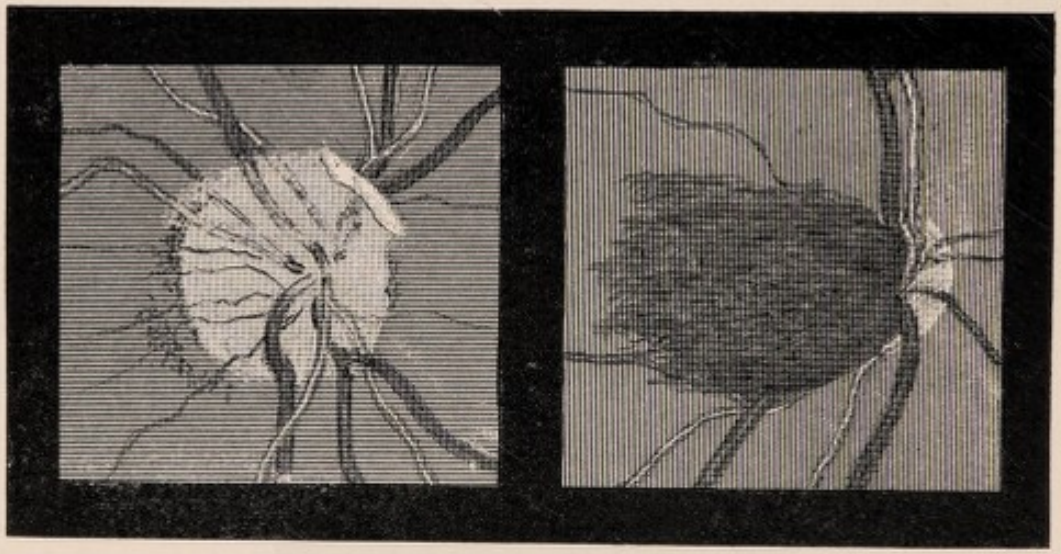


FIG. 1A. FIG. 1B.

Fig. 1A.—A congenital film of connective tissue at the back of the vitreous, turned over at the right edge, veiling the upper half of the optic disc.

Fig. 1B.—A pigmented mass of tissue over the left half of the optic disc and adjacent retina. It presented no change under observation, and in the opinion of Liebreich and others was probably congenital.

In considering what may be learned regarding the conditions of the general system by observation of the fundus oculi, it will be convenient to consider, in the first place, those intra-ocular changes which are of general medical significance, viz., the changes in the vessels and the circulation; the changes, inflammatory and atrophic, in the optic nerve; and, more briefly, the alterations in the retina and choroid; and secondly, the changes which are met with in special diseases of the nervous and general systems.

PART I.

CHANGES IN THE RETINAL VESSELS AND OPTIC NERVE OF GENERAL MEDICAL SIGNIFICANCE.

THE RETINAL VESSELS.

In no other structure of the body are the termination of an artery and the commencement of a vein presented to view, and some information regarding the general state of the vascular system is often to be gained from their inspection. It must be remembered, however, that the vessels there seen are of very small size. One of the primary divisions of the retinal artery, large as it appears to direct ophthalmoscopic examination, is in reality so small as to be scarcely visible to the unassisted eye, being less than the $\frac{1}{100}$th of an inch in diameter. The retinal capillaries are always invisible, and, away from the optic disc, they are never sufficient to occasion any reddish tint. The red colour of the fundus oculi is due to the choroidal vessels.

A second point to be remembered is that the red lines, spoken of as the retinal arteries or veins, are not the vessels themselves, but the columns of blood within them. The walls of the vessels are, as a rule, invisible; they are always invisible to the indirect method of examination, but by the direct method the walls of the larger branches may be sometimes seen, as fine white translucent lines along the sides of the red column of blood, most distinct where one vessel passes over another. They are best seen by feeble illumination, and especially by so moving the mirror as to render the illumination slight and oblique. Sometimes, as will be described immediately, the outer coat of the vessel is so thick as to be very conspicuous.

The paler line which runs down the centre of each vessel is probably a reflection of the light from the middle of the anterior surface of the column of blood. It is distinct only when the vessel lies in a plane at right angles to the line of observation. If the vessel ceases to be in a plane at right angles to the line of observation, this central reflection is no longer visible, and the whole width of the vessel is of the same dark colour as the edge. In the case of veins this change is very striking, and the greater amount of colour makes these portions appear darker in tint than the rest.[1] Many examples of this will be found in the appended plates and figures.

Size.—In estimating variations in size of the retinal vessels, allowance must be made for the refraction, *i.e.*, magnifying power of the eyeball, remembering that, by the indirect method of examination, in myopic eyes the details appear small, while in hypermetropic eyes the objects appear large. In the direct method there is less variation, because, for distinct vision, the myopic refraction requires correction by a lens. The apparent size of the disc may be taken as the guide to the amount of magnification. There is no very exact method of estimating the absolute size of the vessels.[2] Sometimes, however, the alteration is such as to be at once evident and unquestionable. A little familiarity with ophthalmoscopic observation will enable a distinct deviation from the normal to be readily recognized. Special attention must be given to the number of primary branches of the

[1] It is probable that such portions of the veins are especially dark, since by their obliquity to the line of vision, this passes through a greater amount of blood; the light reflection from behind is thus lessened, the choroid being much paler than the blood in the veins. Hence the change in tint is far greater in the veins than in the arteries, which are nearly of the colour of the choroid.

[2] If a wire grating is fixed in front of the light used for the direct examination, the lines of the wires are seen on the fundus, and can be used for measurement. An instrument for use with any light, with wires a definite distance apart, has been made by Hawksley and described in previous editions of this book.

vessel. It often happens that veins are thought to be pathologically large, merely because they are few.

The relative size of the arteries and veins can be observed with more exactness than their absolute size. In comparing the two it is usually desirable to have the pupil dilated, since the vessels have often to be traced for a considerable distance from the disc. A difficulty arises from the fact that the distribution of the arteries and veins corresponds approximately, but not exactly. Sometimes two arterial branches accompany one venous trunk: sometimes two veins accompany one artery. But in each eye there is usually at least one set of vessels which closely correspond, and are available for comparison. When this is the case it will be found that, as a rule, the width of the artery is about two-thirds or three-quarters that of the vein. An alteration in this relation may arise from a change in the size of the artery or of the vein. The change may be so considerable that its nature is at once evident: *e.g.*, the veins may be obviously wider than normal, or the artery unquestionably narrower, perhaps visible as a mere line even by the direct method of examination (Pl. X. 2, 3). When the difference is slighter, we have to form an opinion as to the change on which it depends (whether enlargement of vein or diminution of artery) by our knowledge of the normal size of the vessels—an approximate absolute estimation. A little familiarity with the appearance of the vessels under normal conditions will commonly enable an opinion to be formed as to the direction in which the change exists.

Equality in size of the artery and vein is usually due to dilatation of the artery. When the relative size of the artery is smaller than that given ($\frac{3}{4}$ or $\frac{2}{3}$) it is generally due to one of three causes: (1) Venous distension, general or local; (2) Imperfect filling of atonic veins, in consequence of which they are flattened at right angles to the line of observation: (3) Contraction of the arteries, which may occur from general anæmia (in which case the veins are large and atonic) or from primary arterial contraction, as some-

times in Bright's disease, or from local obstruction to the entrance of blood.

Veins.—Increased width of the veins, therefore, usually means their dilatation, either from distension or from atony, and this effect is commonly uniform. The central reflection is preserved. A varicose condition has been observed in a few cases, but is of doubtful significance. Moniliform dilatation has been met with, especially in Bright's Disease (*q.v.*)[1]

The distension may be part of a general venous fulness, as in cases of cardiac or pulmonary obstruction; or it may be of local origin. Increased intra-cranial pressure of *rapid* development, probably causes at least a transient increased fulness of the retinal veins. When of *slow* development, this effect is rare, in consequence of the anastomoses of the orbital and facial veins. The same effect is believed to be the consequence of distension of the sheath of the nerve, and pressure within the sclerotic ring. The former will be considered in connection with neuritis; its precise influence is difficult to estimate. The influence of the rigid sclerotic ring has not been demonstrated beyond question, but is said to intensify the effect of an obstruction. A very efficient cause of distension of the veins is their compression by inflammatory products in the optic papilla. Extreme distension occurs in cases of thrombosis in the central vein of the retina behind the globe. Occasionally, some of the retinal venous branches are found to be distended in consequence of local pressure, from an overlying artery whose walls have undergone degenerative changes.

Increased width of vein, however, does not necessarily imply over-distension. A vein which is underfilled may present an increased width. Usually, if the quantity of blood within a vein is less than normal, its contractile power enables it to adapt itself to the diminished bulk of the

[1] *See* also "System of Diseases of the Eye," by Norris and Oliver, vol. iii. p. 428 (with drawing).

contents; it retains its cylindrical form, and both appears and is narrower. But in states of anæmia, the atony of the vein may prevent it from following the contents in calibre, and retaining the cylindrical form. It may then have a more or less elliptical lumen (the same circumference enclosing a smaller area as an ellipse than as a circle), and in the retina, in consequence of the intra-ocular pressure, the flattening always takes place in the plane of the retina, at right angles to the line of vision, and the vein appears of undue width. At the same time the central reflection is altered, becoming less distinct, and sometimes unduly broad.

This condition of the veins is seen especially in extreme anæmia, and in leucocythæmia, as in the accompanying figure (*see* also Pl. VII.). In these cases the arteries are usually smaller than normal, and so the contrast between the veins and arteries is enhanced.

Diminution in the size of the veins is probably always the result of diminished supply of blood.

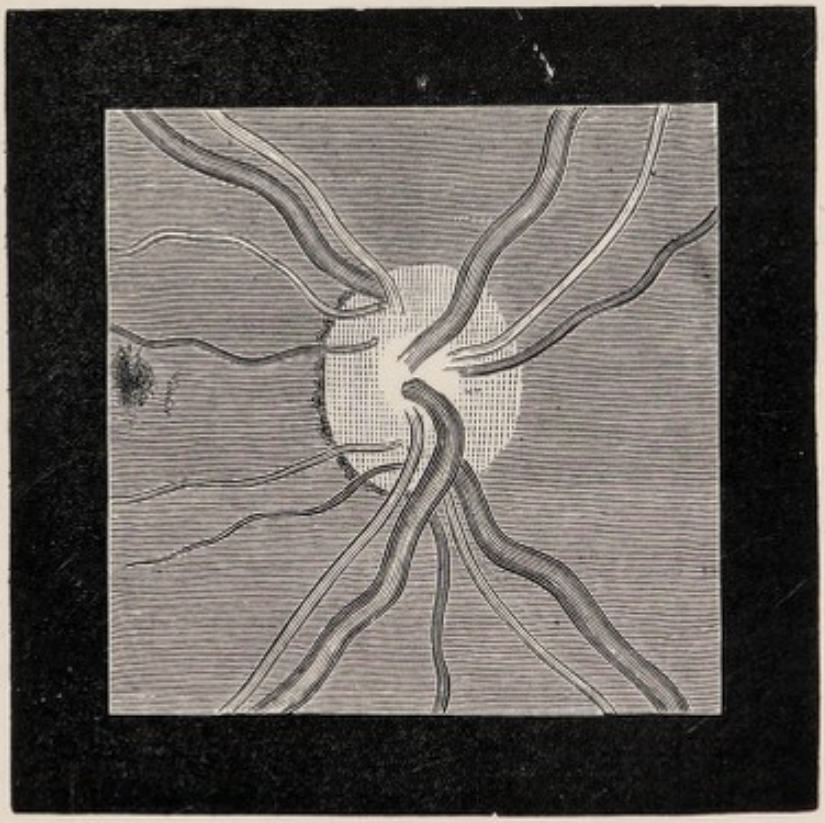


FIG. 2.—BROAD RETINAL VEINS AND NARROW ARTERIES.
From a case of leucocythæmia.

The *arteries* may be diminished in size by causes similar to those which lead to increased width of the veins, such as local obstruction to the entrance of blood. The latter does not appear to result from general intra-cranial pressure, probably because of the resistance afforded by the strong walls of the arteries. It is doubtful whether inflammatory effusion into the sheath of the nerve is capable of diminishing the blood supply, but this effect may be produced by a large hæmorrhage into the sheath-space. It is certain, however, that the pressure of inflammatory products within the papilla, and especially their cicatricial contraction, may cause sufficient constriction of the artery to lead to a great diminution in the size of its branches. Hæmorrhage around the vessel, or the pressure of growths, may have the same effect. In no condition, however, does the diminution in the size of the vessel reach such a degree as in obstruction by embolism (Pl. X. 2 and 3). General underfilling of the arterial system, as in cholera, may lead to a great diminution in the size of the arteries, their strong muscular coat maintaining their adaptation to the blood within them. Mere atony does not cause the increase in width in the arteries which is observed in the veins. Persistent spasm causes a marked diminution in their size, which may be sometimes seen in advanced Bright's disease. If papillitis is superadded, slight compression from this may cause a diminution in the size of the arteries rarely observed from other causes except embolism (*see* Pl. VII. 3 and 4). Spasm of the retinal arteries has been thought to be a consequence of malarial poisoning (*see* Part II., "Malarial Fevers").

Dilatation of the arteries elsewhere is due to the vasomotor nerves, but this effect seldom extends to the retinal vessels, which are preserved from such influences by the closed cavity of the eye. It is doubtful whether this cause alone ever produces visible dilatation of the retinal vessels.

Arrangement.—The anatomical arrangement of the vessels varies considerably in different individuals, and is, in itself,

of little medical significance. The number of branches into which the primary trunk divides, and the number of tributary veins, should be noted in connection with the apparent size of the vessels. The general arrangement of the vessels in the two eyes is usually similar. Moreover, similarity in vascular arrangement may be inherited—a striking proof of the transmission of vascular arrangement in general, upon which depends vascular strain and its effects.

Course.—The course of the retinal vessels usually presents few tortuosities, and those which exist are lateral, in the plane of the retina. A considerable increase in tortuosity may be associated with a nævus of the adjacent part of the skin.[1] The arteries are rather more tortuous in hypermetropic eyes than in others; they are also often markedly tortuous when they have undergone degeneration. When the vessels are elongated by their distension or atony, these lateral curves are exaggerated. Antero-posterior curves, at right angles to the plane of the retina, are indicated by the change in the central reflection already mentioned, by the relative displacement of parts at different levels on movement of the observer's head, and sometimes by slight obscuration of the vessel at the lowest point of the curve. They always indicate irregularities in the retina in which the vessels lie, commonly swelling, as in retinitis and retinal œdema.

Structural Changes.—Most changes in the tissue of the retinal vessels are visible only to the direct method of examination. The commonest change is an increase in the amount of tissue of the wall, especially of the outer coat, so that the red column of blood is bounded by distinct white

[1] *See* Allen Sturge in "Clin. Soc. Trans.," vol. xii. 1879, p. 162; Horrocks, "Tr. Ophth. Soc., iii. p. 106. For cases of idiopathic tortuosity of retinal vessels, chiefly affecting the veins, *see* Benson, "Trans. Ophth. Soc.," vol. ii. p. 55; Nettleship, *ibid.* p. 57; Stephen Mackenzie, *ibid.* vol. iii. p. 101, and vol. iv. p. 152; all with accompanying drawings.

lines. Such an appearance may often be seen near the centre of the disc, where the amount of such tissue varies much, apart from any disease. When a vessel curves over the edge of a hollow central cup, and is seen foreshortened, the white tissue of the wall often appears as a ring around the blood-column. When a disc is very full coloured, whether normally or from pathological causes, this white tissue is rendered, by contrast, very conspicuous, and may easily be mistaken for a pathological condition. It may indeed be left by preceding inflammation, but then it is usually accompanied by distinct constriction of the vessels. It has been thought that this tissue is sometimes a result of chronic congestion of the disc, but the condition is so common that its presence alone has little significance. An undue visibility of the wall of the vessel is said to be sometimes caused by a "sclerosis" of the middle coat, a condition of thickening of the coat which, under the microscope, bears considerable resemblance to the appearance presented by lardaceous degeneration.

Occasionally, the arteries, while retaining their normal red colour, are unusually bright, with a glistening central light-streak ("silver-wire arteries"). Such arteries generally have an unusually tortuous course, show local inequalities of breadth, and, where a branch crosses over a vein, the venous flow is often markedly obstructed. Such changes are highly significant of similar changes in the vessels of the pia mater of the brain, and in small arteries elsewhere in the body; they are often associated with granular kidney. Microscopically, these arteries show great thickening of their coats, with hyaline degeneration. (*See* "Bright's Disease.")

In very rare cases, there is such a thickening of the outer coat of the vessel, or an increase in its perivascular sheath, that the tissue is visible, not merely at the sides of the vessel, but in front of it, concealing the red reflection from the column of blood within it, and broad white bands then indicate the position and course of the vessel. These bands may cease suddenly, so that lengths of red blood may alter-

nate with the white bands (*see* Pl. X. Fig. 1). If the artery is occluded the band may end gradually. This condition is seen in Bright's disease (*q.v.*). Sometimes a vessel may be narrowed at the affected area; more commonly its calibre is unaffected. In some cases an old clot exists within the white-sheathed vessel, closing it, and preventing any circulation in the artery beyond.

In most inflammatory conditions, leucocytes accumulate in the perivascular sheaths, and in the retina they may give rise to an appearance similar to that just described; this has been termed "perivasculitis."

Fatty degeneration of the vessels has been met with as a senile change, or after inflammation. It has only been recognized by microscopical examination, and there is doubt whether it can be detected during life.

In senile fatty degeneration of the outer coat of the retinal vessels, calcification of the degenerated portion has been found after death. Actual atheroma—*i.e.*, endarteritis deformans—has not, so far as I am aware, been found in the retinal vessels after death; and in cases in which it is well marked elsewhere I have often looked for appearances in the retina suggesting its existence, but without success. The retinal arteries are far below the size in which atheromatous changes are common. They have been said to present undue tortuosity in this condition.[1]

Aneurism.—The retinal arteries are occasionally the seat of aneurismal dilatation. Instances of it are, however, rare, probably on account of the support which is afforded to the vessels by the vitreous humour. When aneurism does occur, its significance is important, because in no other way can the existence of aneurisms on vessels so small as those of the retina be ascertained. Dilatations of such small vessels are commonly not associated with aneurisms on large arteries, but when minute aneurisms exist in the retina

[1] Concerning so-called "Arteritis obliterata" *see* Fürstner, "Centralbl. f. Nervenkr.," 1882, and "Centralbl. f. Augenheilk.," 1882, p. 509.

they almost always exist also in the small arteries of other organs.

Two forms of aneurisms have been observed: (1) aneurisms of some size on the primary branches of the central artery on or near the disc; (2) smaller aneurisms of the arterial branches in the retina, and even of the small capillary vessels.

(1). Very few instances of the larger aneurismal dilatations are on record. One, which was described by Sous,[1] occupied the upper two-thirds of the disc, was oval in form, and presented distinct pulsation, synchronous with the radial pulse. The arterial branches in the retina were very narrow. The patient was a woman, aged sixty-four.

(2). Miliary aneurisms were found post mortem by Liouville,[2] in cases in which cerebral hæmorrhage resulted from the rupture of similar aneurisms in the brain. The largest was about the size of a pin's head; they were chiefly situated at the branchings of the vessels. In one case they were widely distributed through the body. They have been found in glaucomatous eyes. I have seen them during life on small arteries in a case of Bright's disease, in which there was extensive cardiac and vascular disease (Pl. X. 1). The lower branch of the artery is seen to present three globular dilatations in its course, the third being just in front of a narrowed segment. The general characters of these aneurisms are there seen. The central reflection of the artery is widened at the dilatation in accordance with the altered surface of the blood within the vessel. The wall of the aneurism is, of course, invisible, just as is the wall of the vessel elsewhere; its existence is declared by the change in the form of the column of blood. Bouchut[3] has figured two examples of a series of fusiform dilatations of the retinal arteries in general paralysis of the insane. His figures, however, suggest considerable exaggeration.

The recognition of these minute arterial aneurisms presents little difficulty. The contours of the arteries must be

[1] "Ann. d'Ocul.," 1865, liii. p. 241. [2] "Comptes Rend.," 1870, lxx. p. 498.
[3] "Atlas d'Ophthalmoscopie Médicale et Cérébroscopie."

followed from the disc as far outwards as possible by the direct method of examination. A twist in a vessel may cause the appearance of a local bulging which may look like an aneurism, but a careful examination will prevent error. Minute hæmorrhages in the course of the vessels can be readily distinguished from aneurisms by the irregularity of the outline of the clot. Aneurisms, as a rule, contain fluid blood, and present a bright central reflection, which is absent in the extravasation. It must be remembered, however, that a miliary aneurism has been found surrounded by a halo of extravasation. The centre of any hæmorrhage situated at the bifurcation of a vessel should therefore be carefully scrutinised.[1]

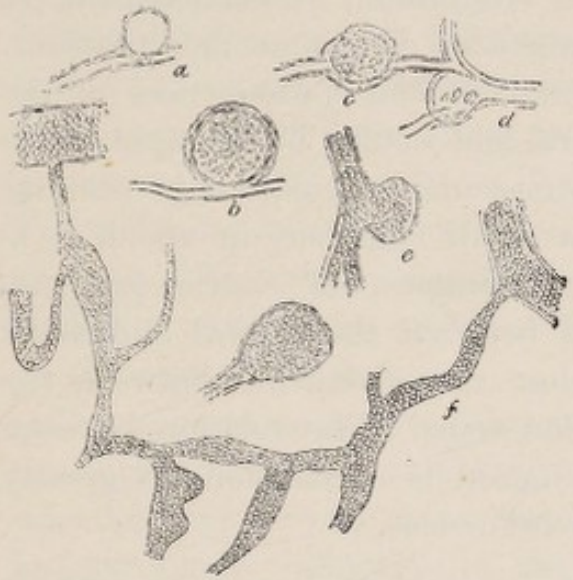


FIG. 3.—CAPILLARY ANEURISMS, AND VARICOSE CAPILLARIES.

a—*e* From a case of diabetes with retinal hæmorrhages (from preparations by Mr. Nettleship) At *a*, *b*, and *c*, the aneurisms are situated laterally, at *c* in the course of a capillary, and at *d* at the bifurcation of a vessel (× 150). *f*, Varicose capillaries from a case of Bright's disease (× 150).

On the retinal capillaries the microscope may show aneurismal dilatations, sacculated in form, and also varicose dilatation. They are met with chiefly in constitutional diseases. Examples of these are represented in Fig. 3, from a case of glycosuria described by Dr. Stephen Mackenzie.[2] Hæmorrhages into the retina and vitreous were observed during life. Capillary

[1] In the "Trans. Ophth. Soc.," vols. iii. p. 108, and vi. p. 336, a striking instance of aneurismal dilatations of retinal arteries and veins is recorded by Story and Benson. The case affords a valuable illustration of the manner in which aneurisms may result from damage done to the walls of small vessels by an inflammation of the walls as part of a general inflammation of the structures in which the vessels lie. The history of the case is unfortunately defective.

[2] "Ophth. Hosp. Rep.," December 1877.

aneurisms, from a case of Bright's disease, are also shown in the same figure.

Changes in the Circulation.

The central artery of the retina brings blood to the eye from within the cranial cavity; the blood comes from an artery which also supplies part of the cerebrum and meninges: the retinal vein returns the blood chiefly to a cranial sinus. Hence the intra-ocular circulation has been regarded as a portion of the cerebral circulation, as participating in the same influences, and presenting the same modifications. This is true to some extent, but the consequences of the common origin of the cerebral and ocular blood-supply have been exaggerated. The intra-ocular circulation is peculiar in its rigid enclosure in a small chamber, in which it is always exposed to a certain amount of elastic pressure. Moreover, the anastomoses between the orbital and facial veins tend to prevent a close correspondence between the intra-cranial and intra-ocular veins. The relation between the cerebral and ocular circulation is unquestionably greatly modified by these and other influences.

Pulsation.—*Arterial.*—As a rule, before reaching arteries so small as those of the retina, the pulse-wave has become so feeble, the current so equable, that visible pulsation can no longer be perceived. The pulsation is also diminished by the normal pressure within the eye; this, in giving support to the retinal vessels, necessarily lessens their distension. If, however, the current be rendered less equable, arterial pulsation may sometimes be perceived. A change in the intra-ocular tension will suffice: thus distinct arterial pulsation is produced by pressure on the globe with the finger. Again, a diminution of intra-ocular tension may, perhaps, sometimes occasion visible arterial pulsation.[1]

In conditions of acute anæmia from hæmorrhage, the

[1] Such a diminution is said sometimes to occur in the course of typhoid fever, and pulsation has been observed in the retinal artery in this disease by Schmall. *See* "Retinal Circulation and Arterial Pulse in General Disease," "v. Graefe's Archiv.," xxxiv. 1, p. 37, and "Oph. Rev.," 1888, p. 268.

continuous flow of blood into the small vessels may be feeble, and the pulse-wave then becomes distinctly visible.[1] But it is especially when the pulse-wave is increased in strength and suddenness that it becomes visible in the retinal arteries. In aortic regurgitation, spontaneous pulsation of the retinal arteries is especially frequent.[2] It is more distinct, the greater is the hypertrophy of the left ventricle, and is absent when the heart is greatly weakened, when much aortic constriction co-exists, or the amount of regurgitation is small. It may be seen best on the disc, but can often be recognized far towards the periphery of the retina, and in this latter respect is distinguished from the pulsation due to mere increase of intra-ocular tension. It consists, like the pulsation of other vessels, in a widening and an elongation. The widening is best seen behind a division at a considerable angle, and by noting the central reflection. The elongation is most evident where an artery lies in an S curve, especially on the edge of the disc.

Pulsation in an extreme degree would appear to be sometimes physiological. It was present in a man under my care who had also a very faint diastolic basic murmur but no hypertrophy or dilatation of the left ventricle, so that there was certainly not enough aortic regurgitation to account for the pulsation. A capillary pulse could readily be obtained in the forehead. The increased pulsation seemed to be in the small arteries only, since at the wrist, even when the arm was raised, the artery had the normal pulse-characters. In the eye, pulsation was conspicuous in both arteries and veins, and slight pressure on the globe increased it to such an extent

[1] An arterial pulse has also frequently been observed by Schmall in cases of chlorosis. Here, as in anæmia generally, Rählmann ascribes the pulsation to hydræmia, but Schmall considers it due to "a certain amount of cardiac contraction, combined with sudden relaxation of the heart muscles, occurring in certain states of low arterial tension." (*Op. cit.*) Arterial pulsation was also noted by Wordsworth during syncope (R.L.O.H. Rep. vol. iv. p. 8.).

[2] Quincke, "Berlin. Klin. Wochenschr.," 1868, No. 34, and 1870, No. 21. Becker, "Arch. f. Ophth.," xviii. 206—296. Fitzgerald, "British Med. Journal," Dec. 23, 1871, p. 723. Dr. Stephen Mackenzie has also recorded several cases ("Med. Times and Gaz.," 1875, vol. i.).

that some veins on the disc, of full size in the diastole, actually disappeared at each systole. Moreover, the diastole of the arteries corresponded to the systole of the veins, and the pulsation in the latter must therefore have been due to the mechanism to be presently mentioned.

Capillary pulsation has been described in aortic regurgitation—a pulsatile redness of the disc—due to the intermitting distension of the capillaries in consequence of the great fall of pressure between the successive pulses. Such an appearance is, however, very rare, and can seldom be detected, even when a pulsatile blush is visible on the forehead.

Venous.—Pulsation in the retinal veins may frequently be observed as a normal condition, chiefly in the large branches upon the optic disc,[1] especially where the veins curve down the sides of the cup. It is almost constant in aortic regurgitation.

Several explanations have been given of the venous pulse. The most plausible assumes that where the artery and vein are near together, in the sclerotic ring or optic nerve, the arterial distension compresses the vein and causes a temporary obstruction to the return of the blood. The nearer the two are, the more readily will this effect be produced. This is probably the true explanation. But others may be mentioned.

Coccius suggested that the venous pulse depends directly on the intra-ocular tension, being such as occurs in glaucoma, or may be produced by pressure on the eyeball. Every time the pulse-wave reaches the intra-ocular arteries, their distension causes a sudden increase in the intra-ocular tension, which compresses the thinner walled veins, and lessens the amount of blood in them. Hence the contraction of the veins should correspond to the arterial diastole, to the pulse-wave, and the dilatation of the veins to the arterial systole, to the interval between the pulse-waves. As a rule, however, this is not the case: the distension of the

[1] Messrs. Lang and Barrett found a venous pulse on the disc in 73·8 per cent. of the eyes examined by them at Moorfields. "Ophth. Hosp. Rep.," vol. xii. p. 60.

veins nearly corresponds in time with the arterial distension. Hence, Stellwag von Carion imagined that the extension of the sclerotic by the increased intra-ocular pressure at each pulse, stretches the lamina cribrosa, and narrows its meshes so as to compress the vein.

According to Donders, the increased intra-ocular pressure acts directly on the venous trunks in the optic disc, hindering the return of blood. Similarly, Jacobi, on the grounds of the common limitation of pulsation to the papilla, suggests that the increased intra-ocular pressure depressing the papilla, augments the curve of the veins, and so causes a sudden obstruction to the circulation through them.

Helfreich,[1] on the other hand, considers that the venous pulse is due to a pulse in the cerebral veins, grounding his opinion on experiments that show the tension in these to be high, and that they pulsate. But the conditions of the two are different; if he were right, the pulsation should be constant.

Anæmia of the Retinal Vessels may be part of general anæmia, or may be due to local pressure upon the artery, and transient anæmia is probably sometimes due to the vasomotor nerves.

When due to local causes ("retinal ischæmia" of the Germans) there is usually simultaneous pressure on the retinal vein, which runs side by side with the artery. The arteries are then unduly narrowed; whether the veins are distended or not depends on the rapidity or slowness with which the obstruction is developed. This condition is constantly seen during the contraction of inflammatory tissue in the papilla. In rare cases, in which the pressure is on the artery immediately after its entrance into the optic nerve, and behind the vein, which enters a little in front of the artery, the arteries may be narrowed without any distension of the veins.

[1] Ophth. Congress, Heidelberg, 1882, and "Ophth. Review," 1882, p. 408.

Spasm of the retinal vessels has been supposed to occur in epilepsy, and also to be the cause of "retinal epilepsy," *i.e.*, epileptiform amaurosis. But in epilepsy no change can be seen, even during an attack, at least of minor nature. In a severe fit, the veins are distended during the cyanotic stage.

Benson[1] has recorded a case where temporary obscurations of vision occurred, lasting about two minutes. Observations made during three of the attacks showed a local emptiness of a large arterial branch, the empty portion being gradually pushed towards the periphery; when it reached the first bifurcation of the vessel, the normal condition was immediately restored and vision returned.

General defective blood-supply is much less evident in the vessels of the eye than elsewhere: probably because the intra-ocular tension effects a regulation of the size of the vessels (Donders). Loss of blood, for instance, causes but a slight change in the retinal vessels, except an increased disproportion between the arteries and the veins, due in part to contraction of the arteries, and in part to atony and flattening of the underfilled veins. The effect soon passes off, because the volume of the blood is quickly reproduced by the passage into it of liquid from the tissues, &c.[2] A similar condition was observed by v. Graefe in cholera. During the stage of collapse the arteries became narrow, the veins dark, but of normal width. Spontaneous pulsation appeared in the arteries, and was attributed to cardiac weakness, but may, perhaps, have been due to the diminution of the volume of the blood, rendering the amount ejected from the left ventricle at each systole so small that the shock (pulse-wave) exceeded the movement of the blood.

[1] Trans. Eighth Int. Ophth. Congress (Edin., 1894), p. 81.

[2] In some observations on the effect of venesection in the corpuscular richness of the blood, made for me by the late Mr. W. S. Tuke, on some patients of Mr. Wharton Jones (who employed bleeding long after it was generally discontinued), it was found that the fall in the number of blood-corpuscles indicating the dilution of the circulating blood to reproduce its volume, took place in the course of an hour. It was found also that the fall was greater than the amount of blood lost could account for —*i.e.*, that the hydræmia became for a time excessive, a fact which may account for the reputed influence of slight, quick loss of blood.

The acute cerebral anæmia of syncope is probably attended by a similar condition of the retina, and to it may be due the transient blindness which sometimes succeeds syncope.

Conditions of general defective blood-supply render the disc paler, but the variations in the normal tint of the disc are so great that it is only by comparison of the state of the disc with its appearance in the same patient at another time, that any information can be gained. The other eye is usually affected in the same degree, and is not, therefore, available for comparison.

Hyperæmia of the Retinal Vessels.—(A.) *Active Congestion.*—Apart from the active congestion of commencing inflammation and of purely ocular conditions, such as refractive asthenopia, and exposure to excessive light, &c. (which are not considered here), an increased supply of blood to the retina may be due to whatever causes an overfilling of the whole or part of the arterial system of which the retinal artery forms part. Of these, excited action of the heart is the most potent. The retinal arteries may be seen to be large, and sometimes, though rarely, to pulsate, and the communicated pulsation in the retinal veins may also, commonly, be observed. When the overaction is long-continued, hæmorrhages may occur. A similar overfilling may occur from obstruction in another region of the internal carotid. Dilatation of the arteries, as in exophthalmic goitre, may also cause increased arterial flow.

(B.) *Passive Congestion.*—Passive congestion of the retinal vessels may occur from local or general causes. The causes of local obstruction to the return of blood from the eye are, for the most part, the same as those of local arterial anæmia. The most intense passive congestion ever seen is met with in thrombosis of the retinal vein. Pressure on the cavernous sinus only causes transient effects, on account of the free connection of the orbital and facial branches. Local venous congestion in the retina may be caused by the pressure of an overlying thickened artery (*see* p. 14). Passive congestion may result from whatever hinders the return of the

blood from the head, or obstructs the circulation through the chest. It is then part of a cephalic congestion, or of a general venous stasis. The former commonly results from pressure on the jugular or innominate veins, the latter from some pulmonary or cardiac obstruction, acute or chronic. The common acute causes are—cough, effort, and an epileptic fit. Unless, however, there is also disease of vessels, hæmorrhages rarely occur, no doubt in consequence of the support afforded to the vessels by the intra-ocular tension, which is probably augmented during these conditions of increased strain, in consequence of the fulness of the capillary vessels. Intense passive congestion, such as that of suffocation, sufficient to cause death, does usually lead to retinal hæmorrhages, but minor degrees of congestion rarely do so. It is very common, for instance, for a violent cough, or an intense asphyxial stage of an epileptic fit, to cause rupture of a subconjunctival vessel, but it is extremely rare for any retinal vessel to give way. I have often, in such cases of epilepsy, searched the retina for extravasation, but the search has always been unsuccessful. In whooping-cough, retinal extravasations have been seen only in extremely rare cases.

Chronic general causes of passive congestion are chiefly heart disease (especially mitral) and emphysema of the lungs. In the venous distension of congenital heart disease, the retinal vessels participate, often conspicuously. The venous distension may be extreme, and may be accompanied by normal arteries, or the arteries may be also large. The blood in the arteries and veins may appear abnormally dark. Sometimes the retinal tissues are thickened. The congestion from emphysema of the lungs, and from dilatation of the right heart, is also often very marked. The retinal veins become much distended and tortuous, and the smaller branches, ordinarily invisible, may become conspicuous.

Hæmorrhage.—Rupture of retinal vessels and consequent extravasations of blood are very common in many morbid

states, and are frequently of important general significance. They may occur as part of inflammation of the retina, and such cases will be considered subsequently. More frequently they are dependent directly on general conditions, or on retinal disease consequent on general conditions.

They vary much in size, number, position and aspect. They may be so small as to be visible only as a spot or line on direct examination, or they may be three or four times the diameter of the optic disc. There may be only one or two, or innumerable extravasations may exist over the whole fundus. When few, they are commonly seated near the disc, or in the neighbourhood of the macula lutea; when numerous, the largest are often situated near the macula. They often follow the course of vessels, especially the veins, but not infrequently the arteries. Their shape and aspect depend very much on their position in the substance of the retina. The commonest seat is in the layer of nerve fibres. The fibres are separated, not torn, by the extravasation, and the blood lies between them, extending along their course in the direction of least resistance. Hence the smaller hæmorrhages are linear, the larger striated in part or altogether, and they often radiate from the disc. Such hæmorrhages are shown in Pl. III. 4, IV. 1, VII. 1, 2, VIII. 1, IX. 1. The next most frequent seat is in the inner nuclear layer. Here there is no tendency to striation; the extravasations are round or irregular (as in Pl. IV. 4, X. 1). If the extravasation in this position is large, it may separate the retina from the choroid, while a hæmorrhage in the nerve-fibre layer may break through into the vitreous. This sometimes happens in Bright's disease, as in one case which came under my observation.[1] The patient, a girl of seventeen, was suffering from chronic Bright's disease and hemiplegia, and had well-marked albuminuric retinitis of the usual type. A fortnight later, a hæmorrhage occurred, partly obscuring the fundus. It did not become diffused, but remained attached to the retina by a pedicle.

[1] *See* also "Ophth. Review," vol. vii. p. 132.

Now and then, especially in the neighbourhood of the macula lutea, the blood may be extravasated in a thin film between the retina and the vitreous. Such an extravasation is sometimes very irregular in shape, and processes of blood occasionally extend into the vitreous. Occasionally a large hemispherical hæmorrhage is found at the macula, bounded superiorly by a straight horizontal line. Here the blood seems to be effused between the internal limiting membrane of the retina and the hyaloid membrane, which are more loosely attached to each other in this situation than elsewhere. The blood quickly gravitates to the lower part of this space, where it is confined by the comparatively close connection between these membranes there existing, and we thus get a hæmorrhage of the characteristic hemispherical form. The more recent a hæmorrhage, the brighter is its colour; when old, it may be almost black. Extravasations may cause permanent white spots; sometimes they are produced rapidly. There may be a hæmorrhage one day, and the next a white spot in its centre.

It is doubtful whether extravasations into the retina occur, however small, except from actual rupture of vessels;[1] probably they are often due to the degeneration of minute vessels, such as may cause the capillary aneurisms shown in Fig 3. Brilliant plates of cholesterin may be seen in the retina adjacent to, or left by, extravasations. The white spots (Pl. IX. 1), when small, may be granular in aspect. They are probably due to fatty degeneration of the disturbed retinal elements or of the effused blood.

Small extravasations are readily absorbed; larger ones more slowly. Sometimes pigmentary degeneration results, and an irregular black spot is left. The white spots disappear very slowly, and white granules may remain for a long time.

[1] According to Leber they are frequently due to diapedesis. "Graefe u. Saemisch's Handbuch," vol. v. p. 557. When local venous obstruction, from pressure of diseased arteries, occurs (such as is described on p. 14), the affected retinal area becomes at first œdematous, or later the seat of hæmorrhages. These extravasations are evidently venous in origin, and probably due to an escape of the red blood corpuscles through the altered coats of the engorged venous branches.

Symptoms.—Small hæmorrhages, away from the centre of the retina, may give rise to no symptoms. Larger ones cause local loss of vision, serious in proportion to the proximity to the macula lutea, in which a small extravasation may cause permanent loss of central vision. A ring of hæmorrhage around the macula may cause considerable central amblyopia (Pl. IX. 2). Occasionally the patient is conscious of the red colour of the extravasated blood (*see* under "Leucocythæmia"). At the moment of extravasation there may be no symptoms, or there may be sudden dimness of sight, or there may be ocular spectra.

Causes.—Hæmorrhage into the retina, as elsewhere, depends on one or both of two causes—increased intra-vascular pressure, decreased strength of vascular wall. Both result from many of its causes. Local increased blood pressure is a common cause. In optic neuritis with much constriction of the veins, the whole fundus is occasionally covered with extravasations (Pl. IV. 1). Similar extravasations may attend all forms of retinitis. They may be large and abundant in thrombosis of the retinal vein (*see* p. 29). General increased blood-pressure seems to be an occasional cause, for high arterial tension may exist when no other cause can be discovered; and it is probably most common in gout, inherited or acquired. But it is, on the whole, a rare accident, considering the frequency with which high tension exists. Its rarity may be due to the efficient support of the retinal vessels, as explained in the description of the effects of passive congestion. In the peculiar vascular condition which attends arrested menstruation, hæmorrhages occasionally occur: also, it is said, in suppression of some other habitual discharge.

In some cases of heart disease, especially when conjoined with degenerated vessels, numerous extravasations may occur into the retina, with other signs of retinal disturbance, particularly venous distension and œdema. This condition has been called "hæmorrhagic retinitis." It may occur without recognizable cardiac disease in apparently healthy persons after middle life, and is often unilateral, but most sufferers

have been gouty. In the great majority of such cases, the retinal arteries are manifestly degenerated, and the hæmorrhages seem indirectly due to this cause (*see* footnote, p. 26). It is possible that, in some cases, they are the result of thrombosis in the retinal vein.

Degeneration of the retinal vessels is probably the most potent cause of hæmorrhage, although it cannot always be demonstrated post mortem. We can only thus explain the retinal extravasations so common in certain general blood diseases, especially in kidney disease and diabetes, pernicious anæmia, leucocythæmia, ague, purpura, scurvy and septicæmia, and many exhausting conditions, such as over-lactation. In some of these cases, as septicæmia, the blockade of vessels may assist. Capillary aneurisms from a case of retinal hæmorrhage in diabetes, and diseased capillaries in renal retinitis, are shown in Fig. 3. Jaundice is said to be an occasional cause.

Senile vascular degeneration is the probable predisposing cause in many cases when retinal hæmorrhage follows some violent effort, such as that of coughing. In all conditions of vascular degeneration its occurrence is of importance, on account of its frequent association with cerebral hæmorrhage.

Another occasional cause is a blow upon the eye or skull. Hæmorrhages have been also met with in young persons without discoverable cause. A remarkable series of cases in young men has been recorded by Eales ;[1] the only etiological condition with which it could be associated was habitual constipation.

The prognosis depends on the position of the hæmorrhage, and on the extent to which its causes are under control. It is worse when there are signs of general retinitis.

The chief local treatment is the application of cold and gentle pressure on the eyeball, to give temporary support to the vessels, and promote contraction. Other measures are those suited for the general state, and for hæmorrhage elsewhere.

[1] "Birm. Med. Review," July, 1880, p. 262.

Hæmorrhage from the choroidal vessels is rare, and possesses little medical significance.

THROMBOSIS.—*Veins.*—Thrombosis is occasionally observed in smaller branches of the veins, which then lose their double contour—*i.e.*, their central reflection disappears, and they appear dark and large, their branches being unduly conspicuous. The condition usually depends on local causes, and has little general significance.[1]

Thrombosis may also occur in the central vein of the retina behind the eye. It is met with chiefly in the old, in whom thrombosis elsewhere is common, and has been seen in association with senile gangrene of the foot (Angelucci). But it occasionally occurs also in younger persons, in association with heart disease, aortic and mitral,[2] apparently due to phlebitis. In one case the vein at the spot thrombosed was thickened to three times the normal size, chiefly from changes in the external coat. The new tissue consisted of concretions such as are met with in psammomata, and was ascribed to an inflammatory process in the connective tissue of the central part of the nerve. The thickening of the vein was so great that it must have compressed the artery. In only one case it was supposed to have resulted from a primary inflammation.[3]

The symptoms observed have presented considerable variation. There is always sudden failure of sight, often discovered on waking in the morning. It is usually incomplete, and soon presents slight improvement. In the most severe

[1] Under the title "Primary Retinal Phlebitis," Mules has lately recorded two cases where the thrombosis was confined to branches of the central vein. There was no local disease found to account for the condition, but evidence of choroiditis subsequently appeared in one of the cases. The general relation of the thrombosis is not apparent, though Mules considers that, in one of the patients, the phlebitis was due to gout. In neither was there any optic neuritis. *See* "Trans. Ophth. Soc.," vol. ix. 1889, p. 130.

[2] Angelucci: "Ann. d'Ocul.," 1880, ii. *See* also "Kl. Monatsbl.," August, 1878; Zehender: "Bericht über 11 Versam. Ophth. Gesel.," p. 182.

[3] Fox and Brailey: "Ophth. Hosp. Rep.," vol. x. pt. ii., June, 1881, p. 205.

cases observed by Michel,[1] the ophthalmoscopic appearances were those of an intense hæmorrhagic retinitis. The veins were extremely distended and tortuous; the retina around the papilla was suffused with blood, beyond this were circumscribed hæmorrhages, and around the macula lutea there was a greyish discoloration. The vitreous sometimes became opaque. In other cases, in which it was assumed that the occlusion of the vein was incomplete, there were merely broad striated hæmorrhages around the papilla, and round and oval hæmorrhages towards the periphery, the arteries being indistinct, and the veins dark and tortuous. In still slighter cases, supposed to be of the same nature, there were no hæmorrhages, but merely a disproportion between the arteries and the veins. In most instances the disc was little affected.

That hæmorrhages may be absent even when the occlusion of the vein is complete, is proved by the case recorded by Angelucci,[2] in which thrombosis of the retinal vein, 1 mm. behind the lamina cribrosa, was associated with senile gangrene of the foot. The veins were tortuous, but there were no hæmorrhages.

In thrombosis of the retinal vein the loss of sight is less complete than in embolism of the artery, and the ophthalmoscopic appearances differ in that the arteries, as a rule, although they may be slightly narrowed, are not empty or filiform,—in the enormous distension of the veins,—and in the circumstance that venous pulsation can usually be observed, and that the veins may appear interrupted here and there. But in some cases the appearances simulate those of embolism very closely. There may be a cherry-red spot at the macula, and in severe cases (probably in which the central artery is compressed by the distension of the vein from clot, or by the thickening of the wall which caused the thrombosis) the arteries may be extremely narrow, the veins partly emptied of blood, and the disc pale.

Artery.—Thrombosis has been observed in the retinal

[1] "Archiv. f. Ophth.," vol. xxiv. pt. 2, p. 37.

[2] "Klin. Monatsbl.," October, 1878. *See* also the same, January, 1880.

artery. Its ocular signs were identical with those of embolism, to be described immediately. It has been found associated with foci of softening in the brain, probably from a similar process. Thrombosis in the ophthalmic artery is a very rare event, and probably always the result of thrombosis in the internal carotid. I am not aware that any case has been observed during life, but some years ago I made a necropsy on a case in which this accident had occurred. The patient, an aged man, had suffered from cerebral softening in the region supplied by the left middle cerebral artery, which was much diseased. A fortnight or three weeks before his death there was no ocular or ophthalmoscopic change. He lay in a comatose condition, and his eyes were not again examined. Post mortem, a recent clot was found extending down into the intra-cranial portion of the left internal carotid, fully distending it, and passing also into the commencement of the ophthalmic artery, which, however, near the eyeball, was pervious, being only partially obstructed by clot. The eyeball was quite rotten, the sclerotic of a brownish colour, and giving way before the scissors like brown paper. The retina was greatly atrophied, reduced to two-thirds of its normal thickness. Its several layers were no longer recognizable. The outer half was occupied by a thick layer of nuclei, apparently representing the two nuclear layers. Its inner half consisted of a series of lacunæ, limited by the remains of the thickened vertical fibres. No nerve-fibre layer, ganglion cells, or molecular layers could be discovered. A case of the same character, but in which a freer collateral circulation was established and the retinal changes were slighter, has been recorded by Virchow (*see* "Softening of the Brain"). Parinaud[1] relates a case of thrombosis of the central artery of the retina, followed by symptoms of cerebral softening, in a woman aged seventy-one, who was suddenly seized with dimness of vision in the left eye, accompanied by the appearance of green and yellow spots on a grey ground. A few days later there was a central scotoma with pronounced peripheral limitation of the field of vision, and loss of colour-

[1] "Gaz. Méd. de Paris." 1882. p. 627.

sense. Ophthalmoscopically, the only change observed was a diminution in the calibre of both veins and arteries, followed ten days later by œdema of the retina with hæmorrhages, and capillary congestion around the macula. Three months afterwards there was atrophy of the disc, and several branches of the central artery were filiform and white.

Priestley Smith[1] has urged that arterial thrombosis is the lesion in many cases that are thought to be embolism, and this view is strongly supported by clinical evidence. We are justified in regarding the arterial obstruction as thrombotic when the retinal arteries show evident signs of degeneration, and when there is an absence of any cardiac or vascular lesion likely to cause embolism. A history of previous similar attacks that soon passed off is also in favour of thrombosis.

Embolism.—The central artery of the retina is not infrequently occluded by an embolus, and the occurrence is of much medical interest. Nowhere else can the process be observed during life. The accident is commonly the consequence of heart disease, and is sometimes the first thing which draws attention to the existence of the cardiac affection. It was so in the case of a girl who had sudden loss of sight in one eye, and was found to have mitral constriction. Embolic infarction in other organs coexists in many cases, and the ocular accident may indicate the nature of disease elsewhere. It occasionally concurs with cerebral embolism, and may even furnish a warning of the probability of the latter, as in a case recorded by Landesberg, in which the ocular embolism was followed, a week later, by loss of consciousness and hemiplegia. This patient suffered at different periods from embolism of each retinal and one cerebral artery. The cerebral and ocular accidents may occur simultaneously, as in the case shown in Pl. X. Fig. 2. The diagnosis of cerebral embolism is usually sufficiently clear without it, but its occurrence is an important corroborative, and almost demonstrative, proof of the nature of the cerebral

[1] "Ophth. Rev.," vol. iii.

lesion. Retinal, as cerebral, embolism is rather more frequent on the left than on the right side. Its common cause is, as already stated, cardiac disease, especially mitral stenosis. It has also been observed in atheroma of the aorta and in febrile diseases, pregnancy, and Bright's disease, probably from the formation of a clot and its detachment. It may, therefore, occur at any age.

The position of the obstruction may be in the trunk, or in one of the branches.[1] In each case there is sudden and complete loss of sight, persistent when the obstruction is in the trunk and is lasting.[2] In rare cases the loss of sight is not instantaneous, but comes on in the course of a few minutes, commencing at the periphery. When the obstruction is in a branch, sight soon returns, except from that portion of the retina which is supplied by the occluded vessel.

The arteries beyond the obstruction, deprived of their supply of blood, contract, so that to the ophthalmoscope they appear as fine lines only (Pl. X. 2). They commonly, however, retain their red colour, because the contraction does not obliterate their cavity, although reducing it almost to capillary dimensions, and there is still a narrow column of blood within. Towards the periphery, however, they are so small as to be invisible. The delicate wall of the vessel is unrecognizable, except in the larger vessels, where, on account of its contracted state, it is more distinct than normal, and appears as a white line on each side, bounding the narrow, red column. When the obstruction is complete and no collateral circulation is established, the red column may disappear, and only a white line indicate the position of the empty vessel, which gradually becomes transformed into fibrous tissue (Pl. X. 3). In this drawing the arterial branch which passes upwards and to the left is represented

[1] See account of such a case (with ophthalmoscopic drawing) by Lawford ("Trans. Oph. Soc.," x. p. 153).

[2] Should the retina be nourished in part by a cilio-retinal artery, embolism of the trunk of the central vessel will not cause complete loss of sight, since the retinal area corresponding to the distribution of the abnormal artery will retain its function. Such a case is recorded by Benson ("Ophth. Hosp. Rep.," vol. *x*, pt. iii. 1882, p. 336).

only by a branching white line, while one which passes vertically upwards, and is not quite empty, is bounded on each side by a white line. Not infrequently detached columns or cylinders of blood have been seen in the arteries and in the veins, moving onwards in pulsatile jerks; probably only when the obstruction is incomplete, or when the arteries are becoming refilled.

The veins are narrowed, but less than the arteries. They are sometimes, but not always, broader towards the periphery than near the disc.

The optic disc is paler than normal, and the pallor gradually increases. The retina undergoes very marked changes, consequent on the disturbance of its nutrition. It presents a greyish or white opacity, always most marked around the macula lutea (Pl. X. 2), and commonly also conspicuous around the disc. This opacity may come on in a few hours, but sometimes not for some days. The opacity usually stops short of the fovea centralis, leaving this of a bright red colour, so red that it was once thought to be extravasation, but it is now generally believed that the tint is merely the effect of contrast with the adjacent pallor. The latter is believed to depend on œdema of the inner layers of the retina, and the absence of these layers at the fovea centralis to explain its absence there. But occasionally the fovea may be as opaque as its vicinity, as in Pl. X. 2. Here I found the opacity to depend on much graver structural alterations than are usually supposed to exist. Besides evidences of œdema, there was an infiltration of all the retinal layers with lymphoid cells, similar to those of the nuclear layers, so that the thickened vertical fibres were the only structural elements which could be distinguished. The layer of rods and cones was destroyed in the region of the macula, probably during life, because the pigment-epithelium was in contact with, and adherent to, the outer nuclear layer. In other places the thickened vertical fibres were widely separated.

Hæmorrhages are sometimes seen. The opacity commonly disappears in the course of a few weeks, but may leave

white spots, due to foci of degeneration. The edges of the optic disc are usually hazy. In most cases the pallor persists, and passes into the whiteness of atrophy, which, at last, resembles simple atrophy, except in the extremely small size of the vessels.

The plug has been found after death in several cases, commonly just behind the bifurcation of the artery, in other cases in its course. In the case of simultaneous embolism of the middle cerebral and retinal arteries (figured in Pl. X. 2), the artery in the nerve contained an oval granular embolus (Fig. 4). Other smaller fragments were seen in the narrowed arteries upon the disc.

Very rarely the circulation gets re-established by the

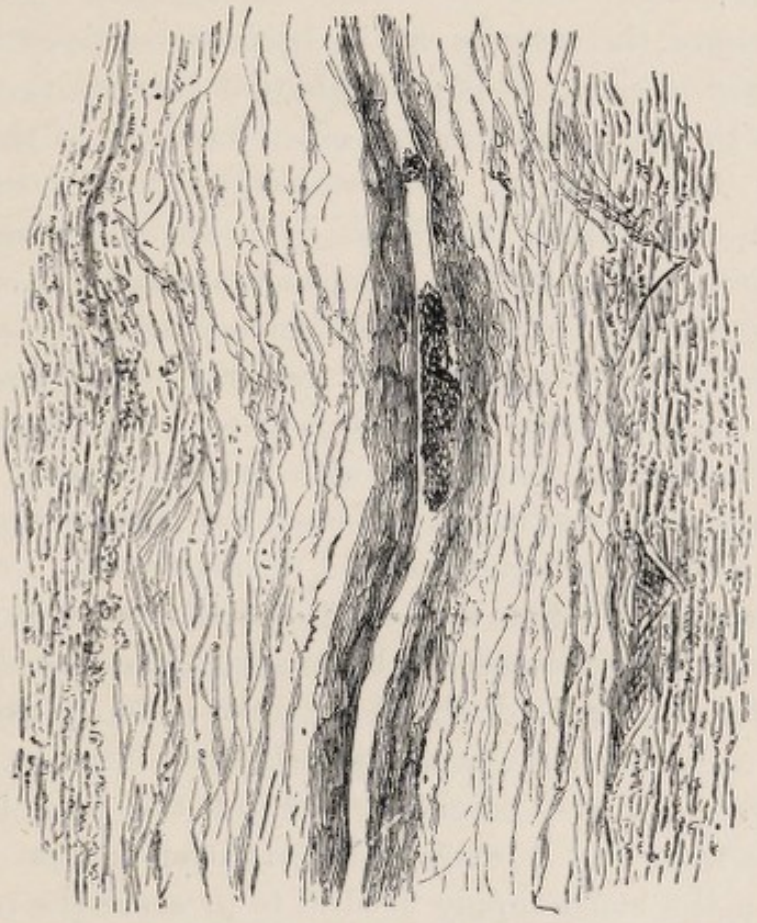


FIG. 4.—EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA (PL. X.). Longitudinal section through the artery, one-eighth of an inch (3 mm.) behind the eyeball. On each side the nerve-fibres are indicated, and between these and the vessel is much loose connective tissue. Within the contracted vessel is an oval granular mass, and in front of this is a small round body (× 300).

normal course. Columns of blood appear in the arteries, in part interrupted, and for a long time easily broken up by pressure. The arteries continue below normal size. Vision may be recovered, especially at the periphery, rarely at the centre. Commonly, however, the obstruction remains complete.

The retinal artery is regarded as a "terminal" artery—*i.e.*, one that has no anastomoses. In most cases very little collateral circulation is set up: the arteries remain narrowed to lines as far as they can be traced. But they are visible in almost all cases as red, not as white, lines. Hence they must contain blood, persistent and therefore circulating, which has come from anastomoses such as exist in the optic disc.

Sometimes the arteries again become pervious although diminished in size. It is probable that this is due, in some cases, to the partial restoration of the channel of the artery, and in other cases to the establishment of considerable collateral circulation. In Pl. X 2, for instance, the arteries are filiform only upon the disc, and as far as they remain unbranched; beyond this, they have nearly their normal size. A similar case has been recorded by Knapp. This points strongly to the establishment of a collateral circulation, probably by connection with the long ciliary arteries, although, in the researches of Leber, such connections could not, in the normal condition, be demonstrated. It is commonly supposed that the chief connection between the retinal and ciliary vessels is by means of the vessels of the optic disc, but it is doubtful whether it is by this means that a collateral circulation takes place. The arteries are never filled in the neighbourhood of the disc, but at a distance from it. A collateral circulation in the disc may maintain the blood-supply needful to preserve the red colour of the filiform arteries, but certainly is insufficient to maintain the peripheral circulation in most of the cases in which this is re-established. Probably, as Mauthner has suggested, there are, in different cases, very variable anastomoses. The re-establishment of the circula-

tion a few hours after the obstruction, has been observed by Wood White and by Eales.[1] In each case recovery of sight occurred. It is probable that the clot either became broken up or so moved as to allow the blood to pass. In Wood White's case the event was apparently produced by pressure on the globe by the finger. The fact is of interest in connection with the occasional transient duration of the symptoms of cerebral embolism.

In Pl. X. 3 the vessel, which is still pervious, though narrowed, is bordered for a distance by a fine white line, indicating the wall thickened by contraction. The blood column within it, narrow as it is, still presents a central reflection, and towards the periphery the vessel again widens out exactly as in the other case, shown in Fig. 2 of the same plate. This broadening of the peripheral portion of the vessel, nearly to its normal calibre, indicates that blood enters it beyond the narrowed portion, by some junction with other arteries.

In partial embolism the segment of the retina, to which the occluded branch goes, becomes opaque, and is sometimes the seat of numerous hæmorrhages. Both opacity and extravasations ultimately disappear. The corresponding portion of the optic disc may be normal, as in the case shown in the figure, or it may be atrophied. The corresponding vein is at first distended, afterwards smaller than normal.

Embolism of the trunk of the central artery commonly causes complete and persistent loss of sight. When the occlusion is of a single branch, there may be a complete initial loss of sight, due probably to the plug causing a temporary obstruction in the trunk of the artery before it passed on to the branch in which it was arrested. Occa-

[1] "Ophth. Rev.," vol. i. pp. 43 and 139. See also in this connection the footnote on p. 39. Mules also has recently recorded a case where plugging of a branch of a retinal artery disappeared, under massage of the globe, about an hour after its occurrence. The visual field was restored forthwith, with the exception of a small area corresponding to the immediate neighbourhood of the embolus. ("Trans. Ophth Soc.," vol. viii. 1888, p. 151).

sionally, in such cases, the blindness has remained complete, although the ophthalmoscope afterwards demonstrated that only one branch of the artery was occluded. The general retinal anæmia may, in such cases, have been so prolonged that the nerve elements suffered a shock, damaging their nutrition beyond the power of recovery on the succeeding restoration of the circulation. Commonly, in such cases, the permanent loss is of a portion of the field corresponding to the distribution of the branch plugged. When this is one of two primary divisions of the artery, the loss may amount to one-half of the field; when of a smaller branch, to a quadrant, or the like. There was a loss of nearly one-half in the case figured in Pl. X. 2, in which one branch running upwards and outwards is completely obliterated, and others running upwards and inwards are partly obliterated. The loss was that shown in the adjacent diagram of the field of vision (Fig. 5).

Occasionally, sudden blindness has occurred, and the retinal arteries have appeared narrow, recovering their normal size

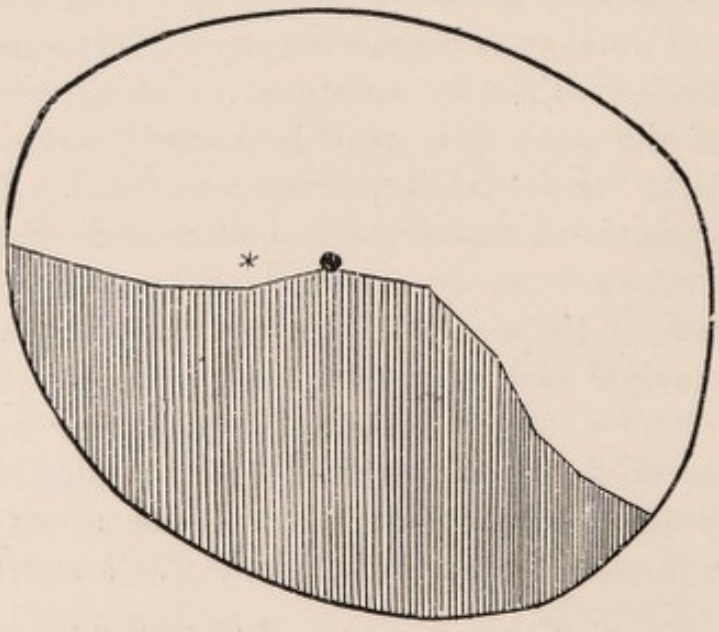


FIG. 5.—DIAGRAM OF RIGHT FIELD OF VISION IN PARTIAL EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA.

The descending branches of the central artery were normal, but those proceeding upwards were empty. (Pl. X. 2.) The shaded area indicates the portion of the field in which sight was lost. The asterisk indicates the position of the fixing point, the dot that of the blind spot.

after a short time, with restoration of vision, as in the cases of Wood White and Eales, discussed above.[1]

Arterial anæmia, similar to that which results from embolism, has been regarded as the effect of a supposed hæmorrhage around the artery compressing it within the nerve. The ophthalmoscopic distinction of this from embolism is uncertain, and probably depends rather on the incompleteness of the ultimate obstruction than on any differences in the early retinal appearances. It is said to occur in cases in which there is a general tendency to hæmorrhage, and to be the precursor of cerebral extravasation. But it is at present an hypothetical lesion. An interesting case, possibly of this character, is related by Hutchinson.[2]

THE OPTIC NERVE.

The alterations in the optic nerve, as seen at its entrance into the eye, are among the changes in the fundus oculi of greatest importance to the physician. It may be well, before describing those changes, to consider some points, regarding its structure and aspect, a knowledge of which is necessary to understand the pathological changes.

In the optic disc we can see the termination of a nerve—a structure consisting of nerve fibres, a little supporting connective tissue (especially abundant around the central vessels), and a number of blood-vessels, for the most part capillaries, which confer on the disc its tint. The nerve fibres radiate and spread out in the retina, but not equally on all sides, being few on the temporal side, towards the macula lutea, and numerous on the nasal side and especially above and below. The minute vessels of the disc are derived partly from the posterior ciliary (choroidal) arteries, and partly from the central retinal artery, twigs from both of which commonly unite in forming the "circle of Haller," a series

[1] The cause of obstruction in such cases is possibly a temporary spasmodic contraction of an artery, and not really embolism.

[2] "Ophth. Hosp. Rep.," October, 1874, p. 51.

of vessels which surrounds the optic nerve behind the disc. The connective tissue between the bundles of nerve fibres is small in quantity, but contains scattered nuclei. The opening in the sclerotic is funnel-shaped, the wider part being posterior. The termination of the nerve fits pretty closely into the inner, smaller part, while the space between the nerve and its outer sheath passes up into the posterior part of the opening (Fig. 17).

The separation of the optic nerve fibres to radiate into the retina leaves the central hollow known as the "physiological cup," the size and depth of which are determined by the arrangement of the nerve fibres. The vessels are chiefly developed among the nerve fibres and towards the surface of the disc, and hence the central cup is pale. It is often mottled grey and white, from the reflection of the white trabeculæ of the "lamina cribrosa," which occupies the sclerotic foramen, and through the meshes of which the greyer, now non-medullated, nerve fibres pass. The tint of the circumferential portion of the disc is deepest where the nerve fibres are most numerous, and hence the nasal half of the disc is redder than the temporal half. The arrangement of the nerve fibres also causes the side of the central cup to be steep on the nasal, and shallow on the temporal side, the difference being proportioned to the inequality with which the nerve fibres are distributed. When the fibres are almost all packed on the nasal side, the cup may be very large, and extend on the temporal side to the margin of the disc. On the other hand, the arrangement of the fibres and vascularity are often such that little or no paler central cup can be perceived.

The boundary of the "disc," as commonly recognized, is the choroidal ring, *i.e.*, the edge of the opening in the choroid corresponding to that in the sclerotic. The latter is usually the smaller of the two, and hence a narrow rim of sclerotic commonly appears within the choroidal edge, and is known as the "sclerotic ring." It is often visible only on one side. At the passage of the nerve fibres over the edge of the sclerotic, they often curve a little above the level of the

retina, and this slight prominence has suggested the name of "optic papilla" as a designation for the area of entrance of the optic nerve. Occasionally, in high hypermetropia, the prominence is considerable; in rare cases this elevation is very marked, and is accompanied by decided blurring of the disc-margin, so as to closely simulate the appearance of an early papillitis.

The trunk of the optic nerve possesses a double sheath: the inner is delicate, closely invests the nerve, and is continuous with the pia mater of the brain. The outer sheath is thicker and fibrous, blends in front with the sclerotic, and is continuous at the optic foramen with the dura mater. There is not, as was once thought, a reflection of the arachnoid at the optic foramen, and thus the vaginal space of the optic nerve—*i.e.*, that within the outer sheath—is continuous with the subarachnoid and subdural spaces around the brain. This vaginal space is traversed by tracts of tissue connecting the two sheaths. At the anterior extremity of the nerve, the space passes within the posterior part of the sclerotic opening, and is, according to some authorities, closed; but, according to others, it is continuous with lymphatic spaces in the substance of the optic nerve, and probably also in the retina.

The optic nerve, at its entrance into the eye, becomes liable to certain pathological changes in common with the retina, and also with the choroid. When the retina is generally inflamed, and when it is atrophied, the optic nerve participates in the change. In widespread choroidal disease, the retina ultimately undergoes much atrophy, and the disc then suffers secondarily, from the fact that its nerve fibres are processes from the retinal ganglion cells. Localised choroidal atrophy, when occurring around the disc, also causes secondary nerve-changes from interference with the blood-supply of the disc. But the choroid also undergoes inflammatory changes independently of the general retina.

The pathological conditions of the papilla resolve themselves, from their clinical features, into two groups,—increased vascularity, commonly with increased prominence;

diminished vascularity, commonly with shrinking. The states characterized by the former are more or less inflammatory, and are often included under the generic term "optic neuritis." Those characterized by the latter signs are accompanied by wasting of the nerve tissues, and are included under the generic term "optic nerve atrophy."

It must be remembered that the term "optic nerve" is employed in two senses—to designate the whole nerve, and also its intra-ocular termination, as seen with the ophthalmoscope. To prevent the confusion arising from this double use of the words, it has been proposed by Leber to use the terms "optic nerve" and "optic neuritis" when speaking of the whole nerve, employing only the words "papilla" and "papillitis" to designate the intra-ocular termination of the nerve and its inflammation. This distinction has not, however, come into general use in this country. The custom of employing the term "optic neuritis" as a designation for the intra-ocular inflammation, makes it inconvenient to restrict it to inflammation of the nerve trunk. For the former condition the synonym "papillitis" is very useful, and "retro-ocular neuritis" is employed to express inflammation of any part of the nerve behind the eyeball.

The characters of these morbid states may be thus tabulated clinically:—

A.—Morbid states of the optic nerve, characterized by increased vascularity or signs of inflammation.

1. Simple congestion of the disc; undue vascularity, redness, slight blurring but no obscuration of the edge, and no swelling.
2. Congestion with œdema of the disc (slight neuritis or papillitis); increased redness with swelling (usually slight); obscuration of the edge of the disc, complete to the direct examination, incomplete to indirect examination.
3. Neuritis, or papillitis; increased redness and swelling, with obscuration of the edge of the disc, complete in degree, partial or total in extent.

B.—Diminished vascularity and signs of wasting.

1. Simple atrophy; increased pallor and shrinkage from the first; "primary atrophy."
2. Congestive atrophy; secondary to congestion; pallor slowly succeeding simple congestion.
3. Neuritic atrophy, succeeding pronounced neuritis; "consecutive atrophy," "papillitic atrophy;" pallor slowly succeeding swelling with gradual shrinkage.
4. Atrophy succeeding choroiditis and retinitis; "choroiditic" and "retinitic atrophy," shrinkage of the disc, with lessened size of the large vessels.

A.—*MORBID STATES CHARACTERIZED BY INCREASED VASCULARITY OF THE DISC, OFTEN WITH SIGNS OF INFLAMMATION.*

Simple Congestion.

Increased redness is the universal expression of tissue hyperæmia, but alone it is of little value as a sign of hyperæmia of the optic disc, on account of the great variation in the amount of natural redness. Attention to tint of disc alone is a prolific source of error. Nevertheless, abnormal redness of the disc does occur as a morbid state, and, although in itself a sign of little value, it derives importance from certain concomitant conditions. It is significant (*a*) when it possesses special characters to be immediately described; (*b*) when developed under observation; and (*c*) when it is notably greater in one eye than in the other: but even in the latter case there is room for error in the possibility of a natural difference, or that the paler eye may present an abnormal decrease in redness.

Certain characters aid very much in the recognition of the pathological vascularity. First, morbid redness has usually a tendency to invade the physiological cup, and often, especially when the cup is small and shallow, to obscure it altogether. But this may be a normal condition, and is only significant when it occurs under observation, or in connection with other signs. Secondly, pathological redness has a tendency to render the sclerotic ring or the edge of the

choroid indistinct; to blur the sharpness of the outline of the disc. The change, when *very slight*, may be best appreciated by examination with the direct image. It is due to the circumstance that the seat of the vascularity is the layer of nerve fibres, which passes over the edge of the disc: with it there is a little swelling of the structures or effusion of fluid, which conceals that which is beyond. The term "congestion" is best restricted to those cases in which the increased vascularity exists alone, with so little structural change, that the edge of the disc can be well perceived by both methods of examination. It is rare.

The redness differs from the ordinary tint, being brighter, softer, somewhat velvety in aspect, sometimes finely stippled. The retinal vessels are usually unchanged; but their walls are often conspicuous, by contrast with the redness of the disc, and any white connective tissue about them at the point of emergence is also unduly conspicuous. Occasionally, when the hyperæmia of the disc is the expression of graver changes behind the eye, the arteries may be narrowed, in consequence of retro-ocular pressure.

The condition thus described as "simple congestion" of the disc is usually a chronic state. It is rare, I think, that such simple hyperæmia is the first stage of an actual neuritis. In the latter, swelling comes on *pari passu* with the hyperæmia—*i.e.*, congestion with œdema, rather than simple congestion, is the first stage of neuritis. Simple congestion is occasionally seen as a substantive condition, and is said, in rare cases, to precede atrophy. Congestion is not unusual in cases of hypermetropia, and may go on to the semblance of slight neuritis. It may occur as a consequence of injuries, &c., with injection of the eyeball, particularly if accompanied by iritis. The affection of sight which results from the use of tobacco may be attended with this state. It occurs also, probably, from other toxic agents, as lead.

Optic Neuritis or Papillitis.

Congestion with Œdema is really the first stage of papillitis. The normal rosy tint of the disc is increased;

its edge is blurred, but may be recognizable on indirect examination. By the direct method, the morbid appearance is much more marked. The edge of the disc is lost, and the opacity is seen to be in part the result of an undue distinctness of the radiating striation of the nerve fibres as they course on to the retina. It must be remembered that this striation is often visible as a normal condition, especially above and below. Where the aggregation of the fibres is very close, the central cup being of large size, the appearance of commencing œdema may be closely simulated. In the latter condition, however, there is from the first more or less invasion of the central cup, which soon becomes obscured. There is distinct swelling, but at first it may not exceed that sometimes met with in health. The centre of the papilla may be redder than the periphery, because the slighter œdema there allows the vascularity of the disc to be more visible. The retinal vessels may be normal, or the veins may be enlarged.

It is important to note that the direct examination renders these changes more distinct. If the obscuration of the edge of the disc is apparent only, in consequence of the similarity in tint of the disc and the adjacent choroid, the edge of the disc is more distinct on direct than on indirect examination. When the indistinctness of the edge is due to the opacity of the structures in front of it (except in the very slightest form), the edge is less distinct on direct than on indirect examination. This is no doubt due mainly to the fact that the illumination is stronger and the plane of focus is less exact in the indirect method, so that the choroidal edge and the tissue in front of it are in view at the same time; whereas the direct method of examination, by its higher magnification and more exact focussing, reveals the tissues in front of the edge so as to prevent the discernment of the latter. This fact will often be found of service in distinguishing between a normal redness of the disc, and an abnormal obscuration of the edge. Of course, it will not distinguish the latter from the cases just mentioned, in which there is a slight physiological obscuration of the edge by the nerve fibres.

This condition of œdema is usually an acute affection, and is really the first stage and slightest degree of neuritis. It is said sometimes to result from pressure on the retinal vein, causing passive congestion of the retinal vessels, and it has been described as the result of the general passive congestion of heart disease. It is seen also in renal disease and some toxæmic states, and may not then progress further, but must be regarded as the earliest stage of neuritis. In these cases it may be associated with retinal hæmorrhages.

Neuritis (Papillitis).[1]—From congestion with œdema to actual inflammation, the transition is one of degree. It seems better to restrict the term neuritis, or papillitis, to those cases in which the swelling and opacity are sufficient to conceal the edge of the disc, both on direct and indirect examination. This condition is found in most cases to result not merely from vascular congestion and œdema, but from changes in the nerve fibres and connective tissue, such as we regard as evidences of inflammation. The change may completely veil the whole or only half the disc; and from such a slight degree of neuritis to the most intense form we may have every gradation, characterized by very considerable differences in appearance.

These variations in the appearance of the disc in different cases, and supposed differences in their origin, have led to the establishment of two varieties of the affection, simple neuritis (formerly called "descending neuritis") and the "choked disc," "Stauungs-papille." The grounds on which these divisions have been made are, as will be shown, uncertain, and it is better in the first place to consider the common features which all forms of papillitis possess. The supposed varieties and theories of this origin will be then better understood.

Papillitis usually presents certain stages in the course of its development. The transition from one to the other is gradual, and cases are seen which present every degree. At any stage the morbid process may stop, remain stationary

[1] Plates I., II., III., IV., V.

for a time, and then recede. This may occur spontaneously, or as the result of treatment. Thus certain *forms* of neuritis may be distinguished according to the intensity of the changes, but our knowledge of the conditions on which they depend seems insufficient at present to distinguish them otherwise than as degrees of intensity, on whatever differences of mechanism they may ultimately be proved to depend.

The first stage of optic neuritis is that which has been already described as "congestion with œdema"—a condition of increased redness, swelling, and cloudiness, masking the edge of the disc to direct, but sometimes leaving it perceptible to indirect, examination. In this condition the microscope reveals little or no indication of true inflammation. The second stage, that of actual, but slight, neuritis, is characterized by greater opacity and by the absolute disappearance of the edge of the disc even to indirect examination. The transition from the disc to the retina is gradual; the

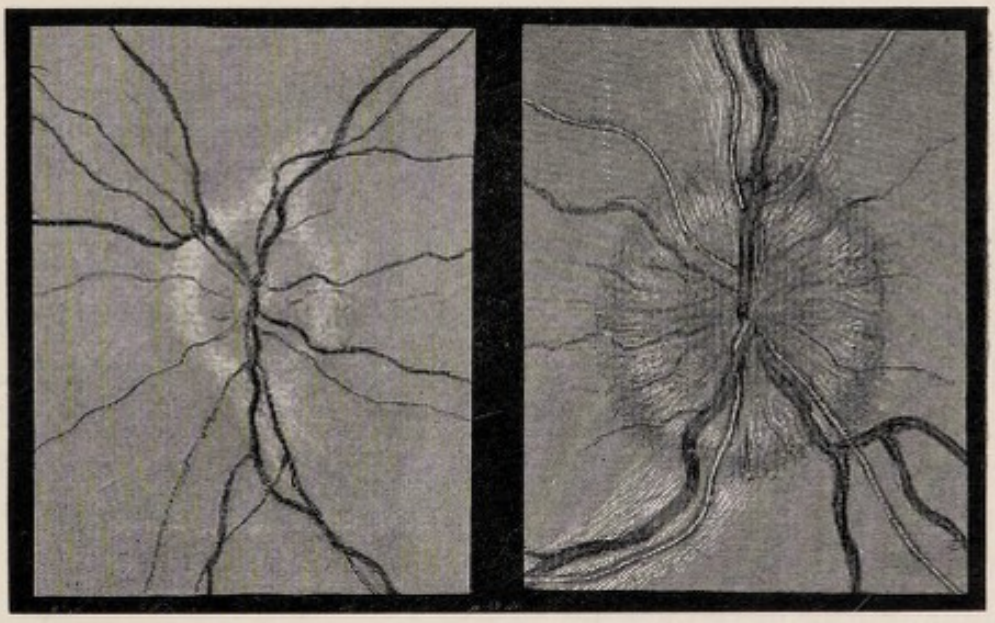


FIG. 6. FIG. 7.

FIGS. 6 and 7.—Early optic neuritis from cerebral syphiloma. The changes were confined to the left disc. The aspect to indirect examination is shown in Fig. 6, while Fig. 7 presents the same condition to direct examination. The contrast between the two, as described in the text, is, perhaps, a little exaggerated, but less than would be thought. The two drawings were carefully made at the same time. (W. R. G.)

edge is "blurred." There are then, besides signs of œdema, changes in the tissue elements which indicate inflammation —increase of nuclei, escape of leucocytes, and even commencing degeneration of the nerve elements.

It is, however, impossible, by the ophthalmoscopic examination, to draw a hard-and-fast line between "congestion with œdema" and actual "neuritis." Considerable swelling and decided obscuration of the disc-edges may exist for some time and then quite disappear, the disc clearing up without a trace of any tissue change and without any impairment whatever of function. Clinically, such a case is simply an early papillitis, and it is just in this stage that its recognition is most important.

As the neuritis increases, the red tint becomes more marked, so that it may be almost the same as that of the adjacent choroid, or it may become a reddish grey, which is very characteristic. The swelling is easily recognized, even on indirect examination, by the relative displacement of different parts on lateral or vertical movement of the lens, and may be estimated by lens-measurement in the direct examination. The striation of the periphery, perceptible in the first stage, increases, but becomes redder. It is due not only to the swelling and opacity of the nerve fibres, but also to the minute vessels which course between them. In the centre of the disc the redness is stippled or uniform, not striated, and the centre is commonly distinctly darker red than the peripheral portion. The striated edge passes, by gradation, into the tint of the adjacent fundus. The physiological cup often disappears during the stage of œdema; if large, a trace of it may remain to the stage of commencing neuritis, but is rapidly encroached upon and covered in by the swelling of the papilla and by exudation (*see* Pl. I. 2).

White lines and spots are not uncommon, especially in the cases in which the changes remain of slight degree. They often correspond to the position of arteries (Pl. I. 3). The swelling and obscuration may involve all parts of the disc equally, especially in the more acute forms of neuritis, or it may be more marked on the nasal than on the temporal side

of the disc. The difference may be so great that the position of the edge of the disc may be distinct on the temporal side, while the nasal edge is completely obscured by opaque tissue (Pl. III. 1, 2, 4). Hæmorrhages are not uncommon in this stage, sometimes on the surface of the swelling, or even on the least changed part of the disc (Pl. III. 1) or just beyond its edge (Pl. III. 4). They are always small. The arteries usually present little change in the slighter stage of neuritis, although often recognized with difficulty on account of the colour of their blood corresponding to the tint of the disc. They are a little concealed at their emergence, on passing through the inflammatory tissue. The veins lose their central reflection as they pass down the sides of the swelling, and appear dark. They may or may not be dilated.

As the papillitis goes on, the swelling increases, and often becomes so great, that there may be a difficulty in seeing its surface by the direct method, without the use of a convex lens. The veins, as they curve down the sides of the swelling, appear still darker and foreshortened, and are sometimes concealed, just beyond its edge, in the adjacent swollen retina (Pl. I. 4, II. 1, III. 6). The veins are now enlarged, often considerably, and the arteries are narrowed, and sometimes concealed. All the vessels may be lost to view at the centre of the swelling (Pl. II. 3, III. 5), where there is a depression, sometimes large (Pl. V. 1). The swelling increases, not only in height but in lateral extent, and partly displaces, partly invades, the adjacent part of the retina, often having a diameter two or three times that of the optic disc. There are rarely signs of any general disturbance of the retina. Extravasations of blood may occur on the surface of the swelling, and not uncommonly, white, flake-like spots appear upon it, often concealing the vessels (Pl. I. 3, II. 1, IV. 2). Occasionally a white spot is surrounded by a halo of hæmorrhage (Pl. IV. 2). Sometimes similar spots exist in the retina close to the edge of the disc.

Many cases proceed no farther than this stage. If the condition begins to lessen, signs of passive distension of the veins, if not present before, develop during the subsidence of the neuritis, especially if this is slow. Neuritis of this stage may clear completely (Pl. II.), the inflammatory products being for the most part removed, and those which remain merely causing a little increase of tissue in the middle of the disc. The subsidence is shown by a diminution in the height and extent of swelling, and also in its redness. At first it may appear somewhat more opaque (Pl. IV. 3), but becomes less so as the swelling subsides. The position of the edge of the choroid becomes appreciable, and gradually clearer, first on the temporal, and then on the nasal side. The disc has a "filled-in" aspect (Pl. IV. 5), and both arteries and veins may be narrowed and partly concealed on its surface. This is especially the case when the new tissue-elements in the disc have been sufficiently abundant to develop signs ot strangulation during the inflammatory stage (Pl. IV. 1, and VI. 1). When this is not the case, the disc may rapidly clear in the centre, as well as in the periphery, and the physiological cup be quickly reproduced. Often the vessels are a little narrowed, and white lines are seen beside them. Commonly, when the inflammatory swelling has been marked, a disturbance of the pigment-epithelium leads to a narrow, irregular zone of atrophy adjacent to the disc.

Whether or not there are signs of mechanical congestion in the stage of neuritis just described, a further increase in the inflammation is invariably accompanied with signs of considerable compression of the vessels, with a rapid and great increase in the swelling, resulting in the condition which has been termed "choked disc," from the German "Stauungs-papille." The tumour formed by the swollen papilla becomes much more prominent, and extends laterally in all directions, even as far as the macula lutea on the temporal side. The form of the swelling varies; sometimes it remains conical, but usually the sides

become steeper, and the top more or less flattened. The sides may even overhang so that the tumour has a fungiform shape, and the vessels, as they pass over the side, may be concealed by the edge of the swelling, and reappear in the fundus in a different position. This intense strangulated neuritis is represented on the next page (Fig. 8, and at Pl. IV. 1).

The arteries are much narrowed, and often altogether invisible on the swelling, being buried in its substance, and appearing first in the retina, a little distance from its edge. The veins may be concealed on the papilla, at least in part, but some of them are usually visible towards its edge, and are greatly distended. When the amount of swelling is extreme, all the vessels may be concealed, as in Pl. IV. 1. Hæmorrhages are frequent and extensive, and are commonly situated on the edge rather than on the surface of the swelling (Pl. IV. 1 and VI. 1). The overhanging edge may be infiltrated with blood. The veins may be concealed beyond the edge of the swelling, and often present many curves and twists, sometimes corkscrew-like from their elongation. The tint of the strangled swelling is usually a full red, mottled and streaked from enlarged vessels and small extravasations. The striation due to the nerve fibres is commonly lost. The retina adjacent is often the seat of hæmorrhages, which may extend along the vessels from the disc. Not infrequently secondary changes occur in the retina over a wide extent. Extravasations, usually striated because situated in the nerve-fibre layer, may be scattered over the whole fundus (Pl. IV. 1). The veins are often distended, and may be tortuous for a long distance from the disc. The retina, in rare cases, presents areas of opacity, diffuse and cloudy, or localized and white, often occurring along the course of the vessels (Pl. VI. 1). When the swelling of the retina is very intense it may become thrown into folds near the disc. On examining such an eye, paler streaks may be seen running in different directions, frequently concentric to the edge of the papilla, probably due to the reflection of

FIG. 8.—ACUTE OPTIC NEURITIS IN A CASE OF CEREBRAL TUMOUR.[1]

There is great swelling of the disc, which is surrounded by radiating hæmorrhages, and, at the macula, is a star-like arrangement of white spots. No albuminuria, and no history of syphilis.

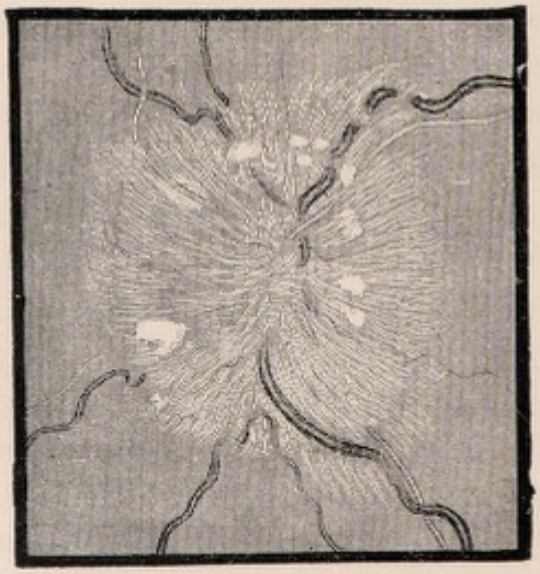


FIG. 9.—ACUTE OPTIC NEURITIS.

The veins and arteries are both concealed by the swelling. The veins are distended, while the arteries are narrowed. Numerous white patches are scattered over the swollen papilla.

[1] After Edmunds, "Trans. Ophth. Soc.," 1884, p. 291.

the light from the summit of the folds. (*See* p. 63 and Pl. V. 1.)

The time taken for the development of these changes varies within wide limits. A neuritis may remain for months and even years in the slighter degree, or most intense strangulation may be developed in a few weeks.

Subsidence of Neuritis.—The gradual subsidence of the slighter degrees of neuritis has been already traced. In the more intense forms, in which strangulation has occurred, the venous distension gradually lessens after the strangulation has existed for a time, and the veins become narrower. In Pl. IV. 1, they are much smaller than in the earlier stage of strangulation shown in Pl. VI. 1. This is probably because the compression of the arteries has lessened the blood-supply to such an extent as to permit the veins to recover nearly their normal calibre. When the strangulation is less intense, the redness of the swelling lessens, hæmorrhages, as a rule, cease to occur,[1] and some of the blood already extravasated disappears. The tumour lessens in height and in extent, and, if fungiform, again becomes conical (Pl. IV. 3). The highest portions of the swelling gradually become pale; the sloping sides and adjacent part of the retina may present a darkish discoloration, into which the central pallor passes gradually (Pl. II. 5, III. 6, IV. 3). The centre of the swelling soon presents a distinct depression, from which the vessels emerge, often concealed by whitish tissue. The course of the veins becomes more distinct, but the arteries may be still concealed, their more rigid, straight course having caused them to be buried in the new tissue more deeply than the veins, which were pushed up before it, and the paler tint of the arteries also renders them less conspicuous. The concealment of the veins beyond the edge of the disc is even greater than it was before, in consequence of their curve into the retina being increased as the swelling subsides (Pl. IV. 4 and 5). Slowly the pallor increases and the swelling becomes less and less, although the

[1] Very rarely fresh hæmorrhages form adjacent to the papilla during the stage of subsidence, as in Pl. IV. 4.

constriction of the vessels may increase, in consequence of the cicatricial contraction of the newly-formed tissue. Occasionally, when large vessels appear on the papilla during the neuritis, these become tortuous, and gradually disappear during subsidence (Pl. II. 5). As the pale swelling narrows to near the limits of the disc, the edge of the choroid and sclerotic appear, dimly at first (Pl. IV. 4, left edge), then more distinctly. The disc has a white "filled-in" look (Pl. IV. 5, VI. 2), the vessels are constricted, and, in severe cases, it may be long before any central depression is developed on the disc. Ultimately the contraction of the fibrous tissue, as in other cicatricial processes, attains an extreme degree, and the disc may again become hollow (Pl. II. 6). The lamina cribrosa is, however, usually permanently veiled, an important characteristic of this form of atrophy. The pigment of the retina is frequently disturbed near the disc, and a zone of irregular pigmentation, with slight choroidal atrophy, is left; but if the neuritis was moderate in degree, the disc may ultimately come to have a clean-cut edge. It is usually very white at first. When it has reached the retinal level, however, although it may appear white in the indirect image, a faint grey tint is usually perceptible on direct examination, and as the contraction increases, this grey tint becomes more marked, and the ultimate appearance of the disc is usually distinctly grey to direct examination. Very rarely the inflammation may subside irregularly, clearing from one part of the disc, while the other still presents the characters of neuritis.

The retina undergoes certain changes during this period of subsidence; hæmorrhages upon it are usually soon absorbed, but sometimes undergo transformation into spots of pigment. Some extravasations lead to the formation of white spots in the retina. This is especially the case near the disc, where the nutrition of the retina is always a good deal disturbed by the adjacent inflammation. These white spots, which depend on fatty degeneration, either of fibrin or of the retinal elements, and persist after the blood has been removed, commonly originate close to the borders of the neuritic

swelling; but, as the latter subsides and contracts, they are left behind, and are often a disc's breadth or more from the edge of the sclerotic ring, and they may then puzzle the observer from their resemblance in character and position to the spots of albuminuric retinitis. A group of such spots, midway between the disc and macula lutea, is seen in Pl. IV. 3. If the œdematous swelling involves the macula, it may cause spots identical in appearance with those which in renal disease form the familiar stellate figure around the macula. A striking instance of this is shown in Pl. VI. 2. The distinction, as will be subsequently explained, rests on the absence of albuminuria and on the evidence the disc affords of a considerable present or antecedent neuritis.

When a neuritis has lasted a long time, and the veins have been persistently stretched over the swelling, they may be so permanently elongated that the subsidence of the neuritis, instead of being attended with a diminution in their tortuosity, is accompanied by an increase in their curves. This is shown in Pl. VI. 4 and 5, in which also a very rare circumstance is presented—the occurrence of recent extensive hæmorrhages during the stage of subsidence.

Second Attacks of Neuritis.—If a disc has become completely atrophied it is very rarely again the seat of inflammation. In one case, however, of a boy aged twelve (under the care of Dr. Hughlings-Jackson), who had double optic atrophy and absolute blindness, due to intra-cranial disease some years previously, distinct double papillitis occurred in the atrophied discs, associated with symptoms of intra-cranial tumour. When, however, atrophy is partial or absent, in rare cases second attacks of neuritis may occur. In one case, for instance, a patient suffered without doubt from a cerebral tubercle, and died from an attack of tubercular meningitis. The former had probably become quiescent, and the neuritis which it caused subsided, leaving partial atrophy. The discs again became swollen and obscured with the symptoms of meningitis.

Pathological Anatomy.—In the condition described as

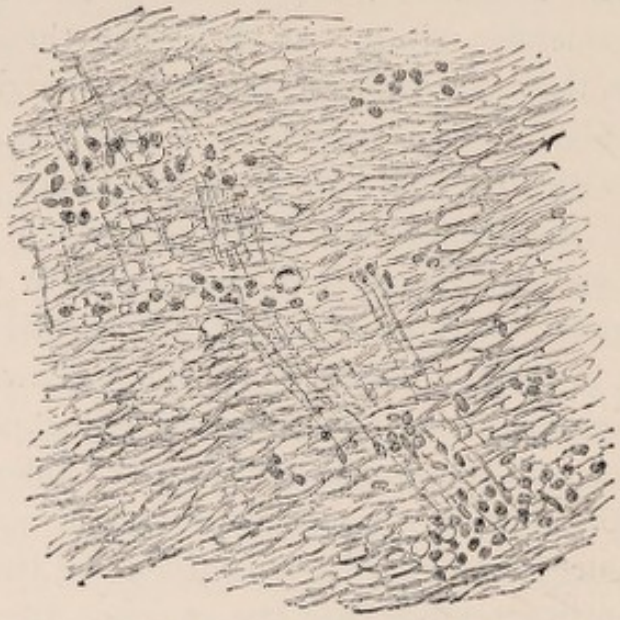


FIG. 10.—OPTIC NEURITIS; NERVE-FIBRE LAYER.

The fibres are separated by numerous round and oval spaces, due to œdema. The nuclei are unduly numerous, and lie in groups, which indicate the fasciculi (× 150.)

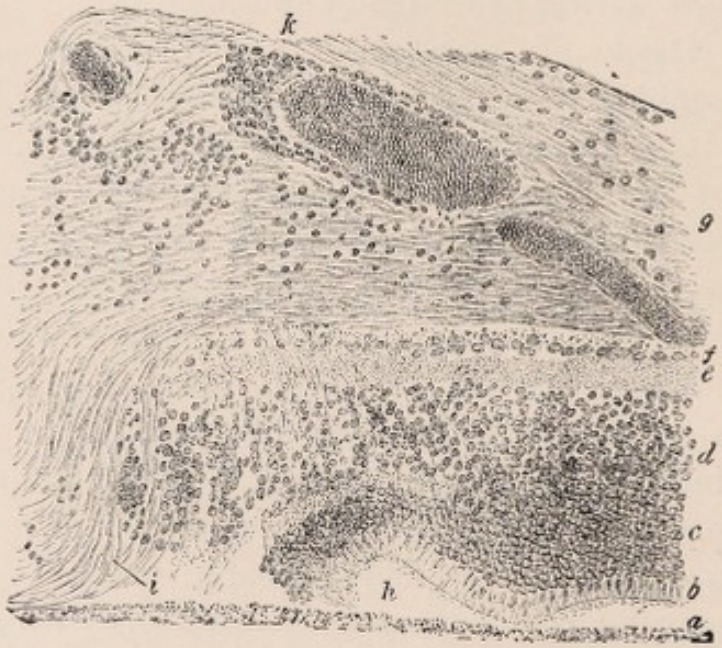


FIG. 11.—SECTION THROUGH THE OUTER PART OF AN INFLAMED PAPILLA.

(*a*) Pigment-epithelium. (*b*) Layer of rods and cones. (*c*, *d*) The nuclear layers. (*e*) The inner molecular layer. (*f*) Ganglionic cell layer. (*g*) The greatly swollen nerve-fibre layer, containing many leucocyte-like cells, many of them surrounding the vessels. (× 150.)

"congestion with œdema," the microscope reveals less prominence than was observed during life, because the swelling depended on distended vessels and effused serum. The nerve fibres are separable with abnormal readiness, and are divided by spaces which during life were occupied by serum (Fig. 10). The fibres themselves may present slight varicosity. There is no increase in the connective-tissue elements, and there are no products of degeneration of the

FIG. 12.—SECTION THROUGH AN ARTERY AND VEIN IN THE SAME PAPILLA.

(*a*) Distended vein; (*b*) contracted artery with thick walls.

FIG. 13.—OPTIC NEURITIS; COLLECTION OF LEUCOCYTES IN A PERIVASCULAR SPACE. (× 150.)

nerve fibres. The retina is normal to the edge of the choroid, its nerve-fibre layer being alone increased in thickness by the conditions mentioned as causing the swelling of the papilla. Sometimes the retinal layers may be displaced outwards a short distance.

FIG. 14.—SECTION THROUGH AN INFLAMED PAPILLA.

The vessels are distended with corpuscles, and several of them surrounded by leucocytes. The nerve-fibres, separated by œdema-spaces, course upward and to the left, and at right angles to them are seen some fine connective-tissue (supporting) fibres. (× 120.)

In the stage of developed neuritis, the microscope reveals a considerable swelling of the papilla, often two or three millimetres above the level of the choroid. There is usually a central depression, which may be larger and deeper than the ophthalmoscopic examination suggested. The swelling may be very distinct to naked-eye examination (Fig. 30, p. 63), and hæmorrhages may be seen upon it. Thus, mere inspection of the fundus after removal may

FIG. 15.—GRANULE-CORPUSCLES, &c.

From the substance of the papilla in a case of optic neuritis. (Glycerine preparation; × 100.)

FIG. 16.—VARICOSE NERVE FIBRES.

From an inflamed papilla in a case of tubercular meningitis. (× 200.)

show the previous existence of papillitis. The swelling is due to several conditions, the relative degree of which varies much in different cases:—(1) The vessels, large and small, are distended with blood. (2) Spaces between the nerve fibres sometimes indicate the persistence of œdema. (3) Many nuclei are seen, some of which are leucocyte-

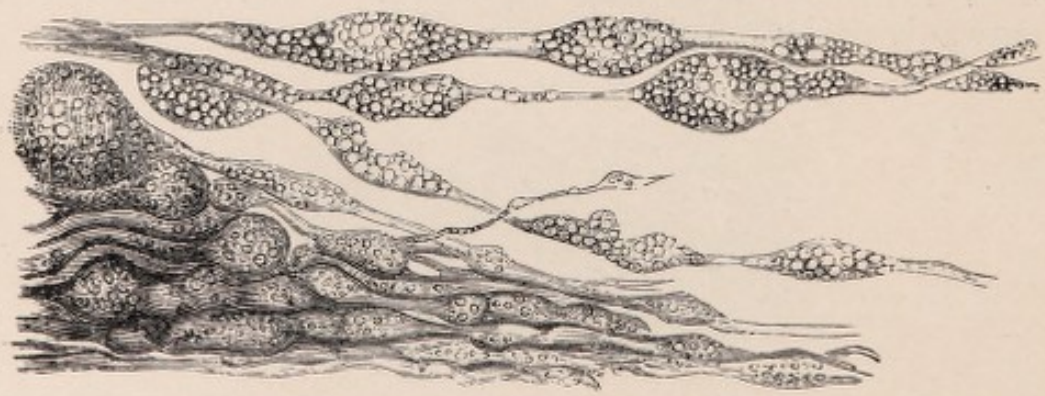
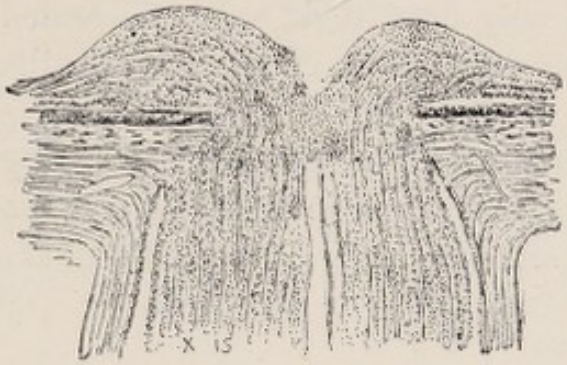


FIG. 17.—DEGENERATION OF NERVE FIBRES.

From the substance of an inflamed papilla in a case of tumour of the lower part of right middle cerebral lobe. Highly magnified. (After Pagenstecher and Genth.)

like corpuscles, most abundant around the vessels, which may be encrusted by a thick layer (Figs. 11 *k*, 13, 14 *a*, &c.); they are sometimes grouped into dense masses (Figs. 11 and 19). Similar corpuscles lie in greatly increased numbers between the bundles of nerve fibres. Some of these are nuclei belonging to a system of connective-tissue fibres which run at right angles to the nerve fibres (indicated in Figs. 11 and 13). These fibres may be themselves



Optic Neuritis in a Case of Cerebral Tumour.

Fig. 18.—Section through the centre of the papilla, showing the swelling of the outer part and a central depression, almost to the choroidal level The nerve fibres can still be traced, separated by cells. The same infiltration is to be seen in the nerve. The sheath is not distended, but its lining membrane is infiltrated with nucleated cells.

swollen. (4) The nerve fibres present changes, which contribute, in varying degree, to the production of the swelling. They are irregularly thickened, and the enlargements may be varicose, moniliform, or knob-like (Fig. 16), often containing granules or fatty globules from degeneration of the myelin. The swellings may attain a large size, as in the accompanying figure (Fig. 17). Free aggregations of fatty

Fig. 19.—The same papilla near the edge. On the left the deeper layers of the retina are seen thrown into folds. (*See* p. 64.)

globules and granules may also be found, commonly enclosed in a cell wall ("granule-corpuscles") (Fig. 15); they may assume a colloidal appearance ("corpora amylacea"). Many of these are simply the detached degeneration-swellings of the nerve fibres. They are best seen in glycerine preparations. These products of degeneration give rise, by their aggregation, to some of the larger white spots seen with the ophthalmoscope. (*See* Fig. 9.) Other spots are apparently due to aggregations of leucocyte-like cells.

The vessels may have their walls thickened by nucleated tissue, and sometimes by a clear, finely fibrillated substance (Fig. 24).

The vessels do not usually present any evidence of compression in the sclerotic ring, but commonly appear to be narrowed, often considerably, in the thickest part of the

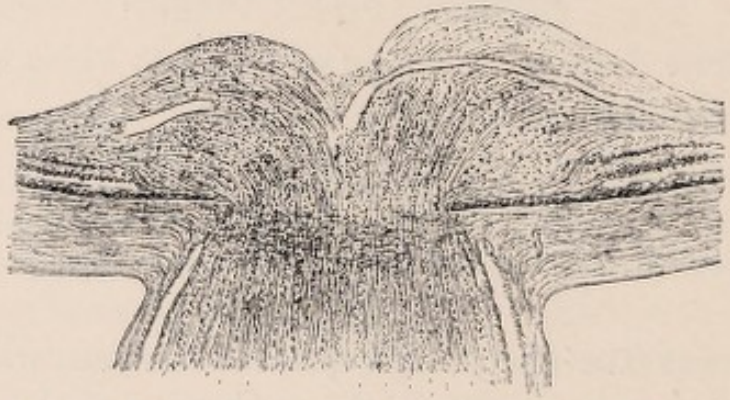


FIG. 20.—SECTION OF THE PAPILLA IN A CASE OF CEREBRAL TUMOUR.

There is considerable swelling, greater on one side. The commencement of the retina is displaced some distance from the edge of the sclerotic ring. Infiltration of nucleated cells in the papilla and nerve-sheath, but the latter not distended. (× 20.)

FIG. 21.—SECTION THROUGH THE MIDDLE OF THE SAME DISC.

The central depression remains, although much narrowed. The central vein is seen divided longitudinally. Neither in the sclerotic ring nor behind it does the vein present any trace of compression. (× 8.)

swelling, and the veins are again enlarged as they pass down the sides. The veins are usually very large, the arteries narrow. The former, after curving down the sides of the swelling, descend into the substance of the swollen retina, even into the outer nuclear layer, and rise again into the layer of the nerve fibres. Sometimes two such curves may exist (Fig. 25). The retina is displaced from the edge of the choroid, often as far as a millimetre from the sclerotic ring. Its layers, adjacent to the papilla, usually present con-

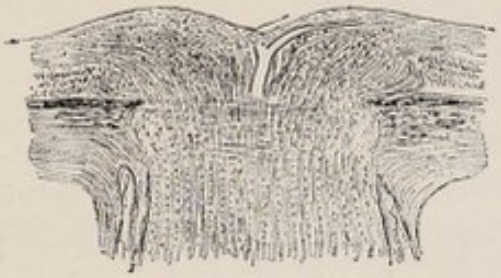


FIG. 22.

FIG. 23.

FIGS. 22 AND 23.—SECTIONS THROUGH THE PAPILLA IN A CASE OF OPTIC NEURITIS DUE TO CHRONIC CEREBRITIS.

(Case published by Dr. H. Jackson in "Ophth. Hosp. Rep.," vol. viii. p. 445.) The papilla is slightly swollen, and has displaced the retinal layers. In Fig. 20 a vein is seen becoming compressed in passing through the inflamed retina, but it will be noted that in Fig. 21 there is no sign of compression, as the central vein passes through the sclerotic ring. (× 15.) *See* also chapter on "Softening of the Brain."

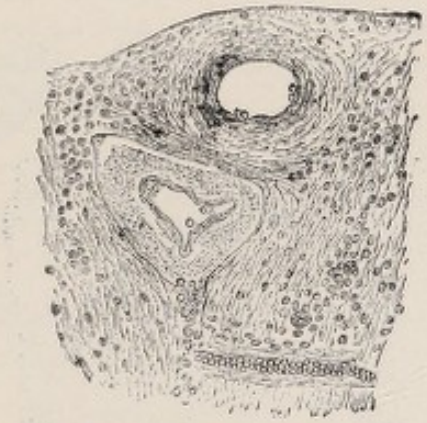


FIG. 24.—PART OF A SECTION OF AN INFLAMED PAPILLA IN A CASE OF OPTIC NEURITIS.

An artery (below) and a vein (above) exhibit thickening and fibrillation of their outer coats. Below is a small vessel showing similar changes. The surrounding tissue is infiltrated with nuclei. (× 100.)

siderable change. The nerve-fibre layer is thickened by a slighter degree of the changes which cause the swelling of the disc. The nuclear layers are increased in thickness and often blended together, and the nuclei are more or less separated and grouped into vertical columns by the displaced fibres of Müller. The retina may present (as here) slight curves due to its displacement, most marked in its outer (deepest) layers, and causing detachment of the retina, the

FIG. 25.—SECTION THROUGH THE SWOLLEN PAPILLA IN A CASE OF OLD CHRONIC MENINGITIS, WITH INFLAMMATORY GROWTHS IN THE CONVEXITY OF THE BRAIN. (*See* Pl. VI. 2 and Fig. 35.)

At the edge of the swelling a large vein forms two vertical curves in the substance of the thickened retina, the lower curve reaching the inner nuclear layer. (× 15.)

FIG. 26.—SECTION THROUGH THE RETINA.

Some distance from the disc in the same case. The vein occupies two-thirds of the thickness of the retina, and in one or two places has encroached on the nuclear layers. (× 50.)

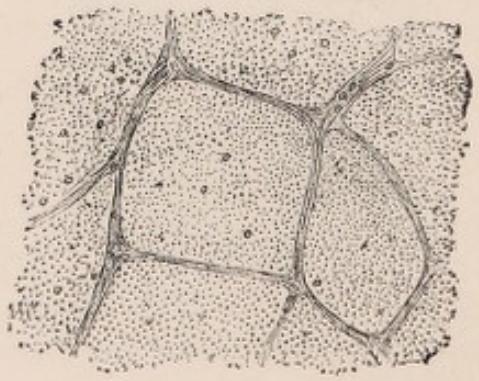


FIG. 27.—SECTION THROUGH A HEALTHY OPTIC NERVE.

For comparison with the subsequent figures.

space between the bacillary layer and pigment being occupied by serum. These curves may be visible with the ophthalmoscope as pale bands, parallel to the edge of the papilla (Pl. V. 1).

The pigment-epithelium may persist up to the edge of the sclerotic, or it may disappear in the area from which the retina has been displaced (Fig. 25). Often there is choroidal atrophy close to the edge of the sclerotic.

The changes in the papilla always become much slighter at

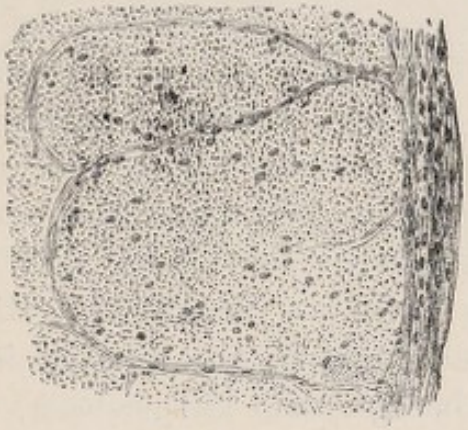


FIG. 28.—TRANSVERSE SECTION THROUGH THE OPTIC NERVE HALF AN INCH BEHIND THE EYE.

In a case of early optic neuritis. Thickening and infiltration of sheath. Very little change at present in the nerve. (× 150.)

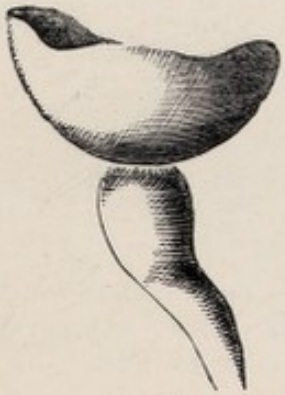


FIG. 29.

FIG. 30.

POSTERIOR SEGMENT OF EYEBALL AND OPTIC NERVE.

From a case of chronic traumatic meningitis, showing the distension of the sheath of the nerve and the swelling of the papilla. (Natural size, after Pagenstecher and Genth.)

the sclerotic ring, and may appear to cease there. Commonly, however, large numbers of nuclei lie among the nerve bundles in and just behind the lamina cribrosa, where such nuclei are in health most abundant. The sclerotic ring may appear distended, the nerve tissue occupying closely its funnel-shaped area. The appearance of distension is partly, if not entirely, due to the shape of the ring, as may be seen by comparing Fig. 18 with Fig. 22. In the latter the appearance of excavation of the edge of the sclerotic is present on the left side only, and an interval exists between it and the nerve fibres, occupied only by the fibres of the lamina cribrosa.

The sheath of the optic nerve is often distended with fluid, sometimes slightly, often considerably. The distension is greatest a short distance behind the eye, and narrows close to the sclerotic, having thus a pyriform shape (Fig. 29). In cases of old neuritis the sheath may be enlarged but empty, showing previous distension. Microscopically the nerve may appear normal, the nuclear increase near the lamina cribrosa being absent farther back. More commonly signs of inflammation may be traced throughout the nerve; the nuclei are increased in quantity, the trabeculæ thickened

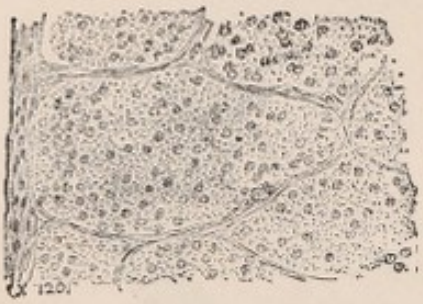


FIG. 31.—SECTION THROUGH THE OPTIC NERVE, JUST BEHIND THE SCLEROTIC.

(Pl. I. 3.) The nerve fibres present only an irregular granular appearance, the axis cylinders being no longer distinct. The sheath presents many compressed nuclei. (× 120.)

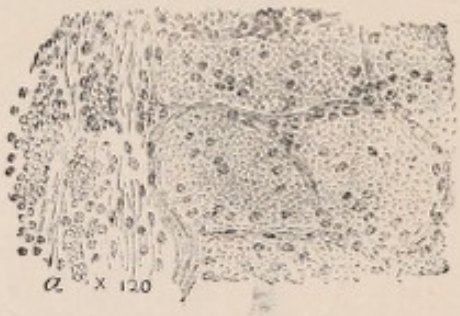


FIG. 32. — TRANSVERSE SECTION THROUGH THE SAME OPTIC NERVE, JUST IN FRONT OF THE COMMISSURE.

The sheath of the nerve (on the left) contains dilated vessels, and large numbers of leucocytes, which are also very abundant in the interfascicular septa. (× 120.)

and the vessels distended (Figs. 31 and 32). The inner sheath is often crammed with nucleated cells, and the connective tissue between the inner and outer sheath increased (Fig. 28). The nerve fibres may present evidence of degeneration (Figs. 31 and 32). These changes, slight or considerable, may often be traced back as far as the chiasma, in front of which they are sometimes much more intense than anteriorly, and are most intense near the surface of the nerve. This is seen

FIG. 33.—LONGITUDINAL SECTION OF THE OPTIC NERVE, FROM A CASE OF OPTIC NEURITIS,

Showing the irregular outline of the degenerating nerve fibres, and the accumulation of cells between the fasciculi. (× 120.)

especially in cases of meningitis, and affords evidence of extension of inflammation from the meninges. An increase of nuclei is sometimes to be traced into the chiasma, and even into the optic tract (Fig. 34), where the corpuscles may even be aggregated into the groups that have been termed "miliary abscesses" (Fig. 35).

During the progressive subsidence to atrophy, there is a

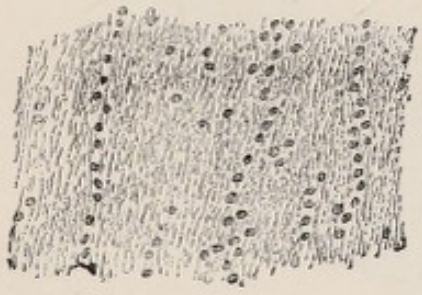


FIG. 34.—LONGITUDINAL SECTION THROUGH THE OPTIC TRACT, IN A CASE OF OPTIC NEURITIS.

There is increase in the connective tissue corpuscles between the fibres. (× 150.)

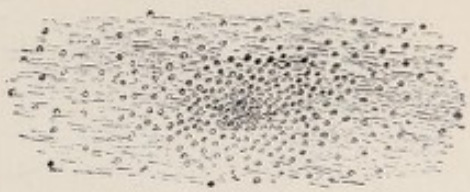


FIG. 35.—SECTION THROUGH THE OPTIC TRACT IN A CASE OF CHRONIC MENINGITIS.

Shows aggregation of nucleate cells into a "miliary abscess." (× 100.)

diminution of the cellular elements in the papilla, probably, in part, in consequence of their transformation into fibres. The products of the degeneration of the nerve fibres are slowly removed. Ultimately the substance of the papilla appears to consist of a felty mass of interlacing fibres sprinkled with nuclei, in which at last scarcely any indication of nerve fibres is to be traced.

Symptoms.—Subjective symptoms may be entirely absent, even when the inflammation of the papilla is of considerable intensity, as was first pointed out by Hughlings-Jackson. Vision may be unimpaired—acuity and colour-vision being perfect, and the field unrestricted. An increase in the size of the blind spot may usually be ascertained by mapping it out with the perimeter, but of this the patient is unconscious. The degree of neuritis which may exist, with no impairment of acuity of vision, is remarkable. In the cases shown in Pl. I. 5, II. 1, 3, III. 1, 2, IV. 4 and 5, when the drawing was made, the acuity of vision was scarcely or not at all impaired. It is often said that "descending neuritis" causes

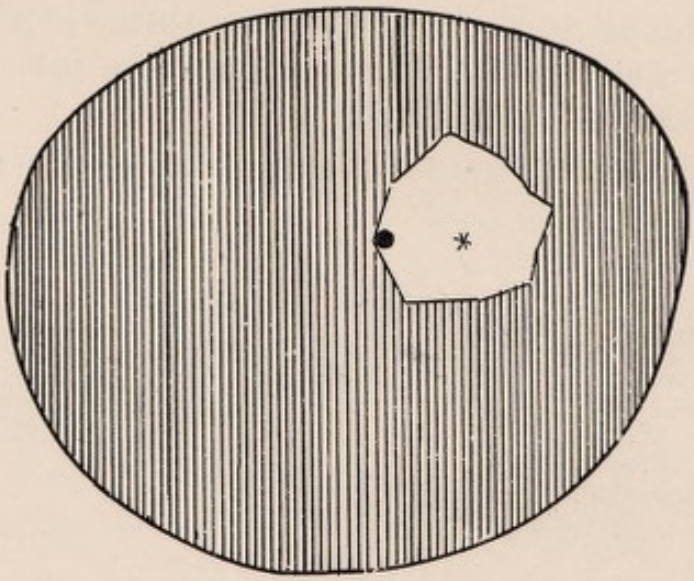


Fig. 36.—Diagram of the Field of Vision in a Case of Subsiding Optic Neuritis in Cerebellar Tumour, Left Eye.

The outer boundary of the figure is the limit of the average normal field. Vision was lost in the shaded area, preserved only within the inner line around the fixing point, the position of which is indicated by the asterisk.

much earlier affection of sight than limited intra-ocular papillitis. But acuity of vision may be unimpaired even with an apparently descending neuritis. In more intense cases, however, sight is impaired or lost, and this constitutes the chief subjective symptom. Photophobia and pain in the eye are very rare in optic neuritis. Pain in the head may occur in cases of apparently primary papillitis; it is, of course, a very common accompaniment of symptomatic inflammation, but is then generally to be accounted for by the intra-cranial disease.

The affection of vision usually occurs earlier in the one eye than it does in the other. It may come on rapidly or slowly; never suddenly. Sometimes the rapidity of its progress may be great; sight may fail completely in the course of a few days.

Restriction of the visual field usually accompanies considerable change in the acuity of vision. It may be extensive, and often reaches its height during the stage of subsidence. Only a small area around the fixing point may remain, as in the diagram (Fig. 36), from a case of subsiding

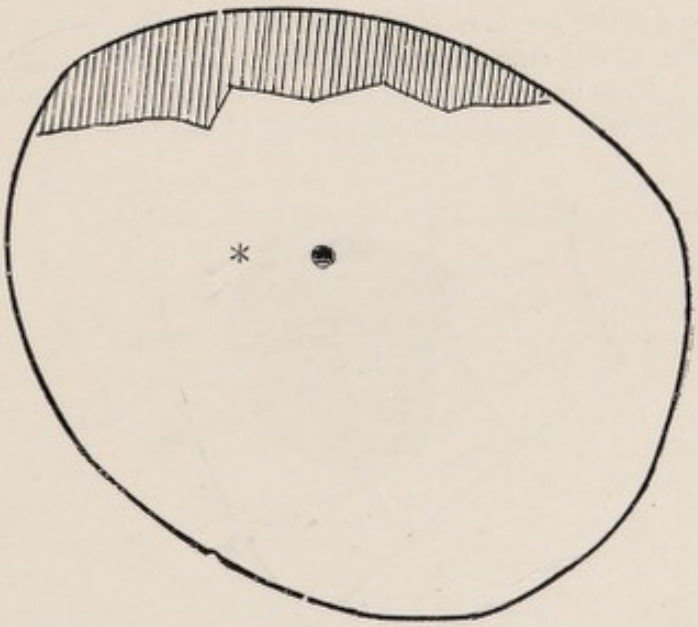


FIG. 37.—DIAGRAM OF FIELD OF VISION, SHOWING LIMITATION ABOVE ONLY.

From a case of unilateral optic neuritis, probably due to cerebral syphiloma. There was amblyopia, but no neuritis of the other eye. Both discs subsequently became atrophied.

neuritis in cerebellar tumour. Occasionally the limitation of the field of vision may be irregular, one part being more affected than the rest, as in Fig. 37, in which the upper part only is restricted. In some cases, a change in the field of vision, due to the intra-cranial disease, may accompany the peripheral limitation due to the optic neuritis, as in the diagrams (Figs. 38 and 39) of the fields of vision in a case

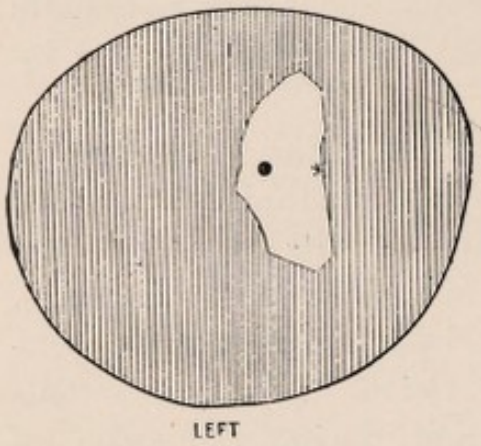


FIG. 38.

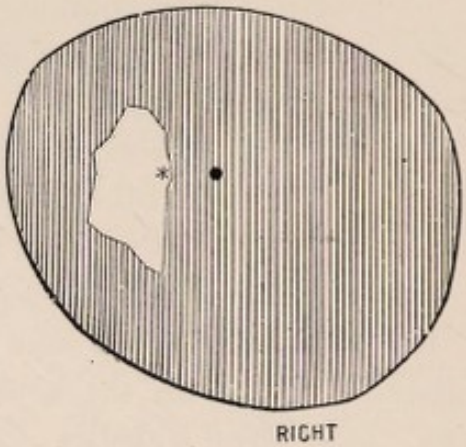


FIG. 39.

DIAGRAMS OF THE FIELDS OF VISION IN A CASE OF HEMIANOPIA AND DOUBLE OPTIC NEURITIS.

Probably due to a cerebral syphiloma. The asterisk represents the fixing point, the dot the position of the blind spot. The outer boundary of the shading is the normal limit of the field, the shading the area in which sight was lost. There is seen to be loss of the whole right half of each field, with concentric limitation of the left halves.

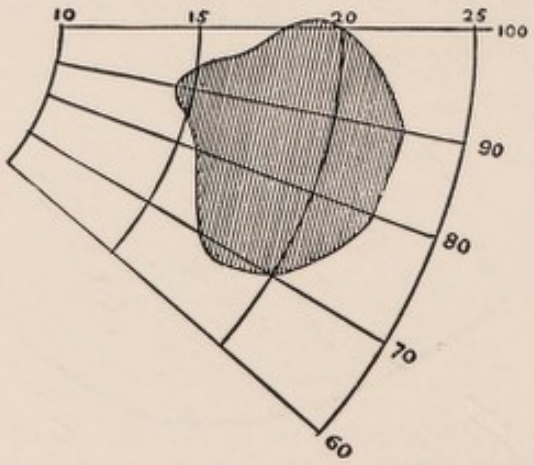


FIG. 40.—DIAGRAM OF THE BLIND SPOT (SHADED AREA) IN A CASE OF OPTIC NEURITIS.

From a case of tumour (probably a syphiloma) in the left ascending parietal convolution (*see* also Pl. II. 3).

in which hemianopia, owing to the intra-cranial disease, accompanied the peripheral limitation. Not infrequently there is marked peripheral amblyopia, and a small object cannot be recognized in the periphery, although a large object, as the hand, is well seen. The increase in size of the blind spot is proportioned to the size of the papillary swelling. The accompanying diagram (Fig. 40) shows its area in a case of optic neuritis figured in Pl. II. 3. It is a little, but not much, larger than normal.

When there is distinct amblyopia there may be a defect in colour-vision, and the latter may exist even when acuity of vision is very little impaired. The order of loss is sometimes (as in atrophy, *q.v.*) that of the normal peripheral arrangement of the colour fields in the accompanying figure (Fig. 41), red and green being lost before yellow and blue. Thus in a girl aged eleven, who had optic neuritis of both eyes, there was little limitation of the field for white. The only colour which she could name accurately was yellow. Light shades of other colours were called white, deep shades black. More

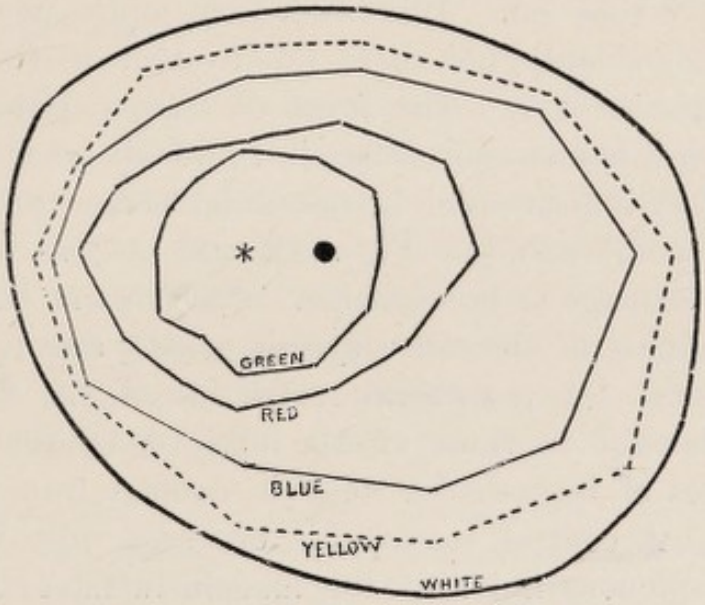


FIG. 41.—DIAGRAM SHOWING THE FIELDS OF COLOUR-VISION IN A NORMAL EMMETROPIC EYE ON A DULL DAY. (NETTLESHIP.)

The fields are each rather smaller than on a bright day. The asterisk indicates the fixing point, the black dot the position of the blind spot. (Usually the blue field is larger than the yellow.) *See* the section on "Atrophy of the Optic Nerve."

frequently, however, the loss is irregular; I have known yellow alone to be lost. In a case of severe neuro-retinitis due to chlorosis, at one time, yellow was alone lost in one eye, and in the other eye yellow, blue, and green were lost, red being seen. Sometimes colour-vision is little affected, even when there is considerable peripheral limitation of the field of vision.

When sight is completely lost, the sensibility of the retina to electrical stimulation may or may not be impaired. It may be lost with vision, and return with some recovery of sight.

It is important to discern the mechanism by which sight is impaired, since the prognosis must, in the main, depend thereon. The loss of sight which occurs in cases of idiopathic papillitis is, of course, due to the visible process. But neuritis in intra-cranial disease may be accompanied with loss of sight, due either to the intra-ocular changes, or to disease in the course of the optic fibres or in their central connections.

One distinction is afforded by the manner in which loss of sight comes on. Blindness from optic neuritis never comes on suddenly, not more rapidly than in the course of two or three days. The form of loss is important. A symmetrical hemianopic defect in the fields (such as in Figs. 38 and 39) indicates an intra-cranial cause; and a loss of each temporal half (as Figs. 42 and 43) is evidence of probable damage to the chiasma. Such loss of half one field and blindness of the other eye, is usually due to disease at the chiasma. A peripheral restriction of one field usually means damage in front of the optic commissure, and, in most cases of intra-ocular neuritis, damage from the visible changes. A central scotoma is observed only when there is a conspicuous lesion at the macula lutea, or in cases of primary retro-ocular neuritis.

Lastly, important assistance is derived from the degree of intra-ocular change—is it sufficient to account for the loss of sight? The question is often difficult to answer. It is only severe papillitis that gravely impairs sight by involvement of

the conducting fibres and their later compression. The latter, from the cicatricial contraction of the inflammatory products, may lessen sight which has been normal during neuritis of moderate severity, and always augments the loss which has resulted from the acute inflammation.

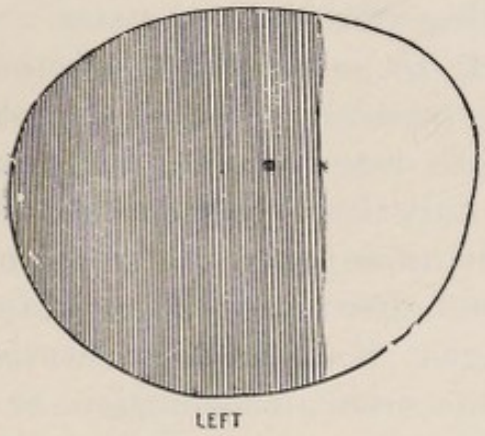


FIG. 42.

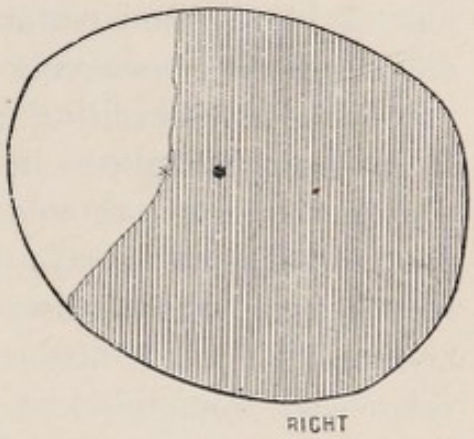


FIG. 43.

DIAGRAMS OF THE FIELDS OF VISION IN A CASE IN WHICH SIGHT WAS LOST PROBABLY FROM PRESSURE ON THE CHIASMA.

The shaded area indicates the part in which vision was lost—viz., the temporal portions of both fields,—the nasal portions alone persisting ("temporal hemianopia").

CAUSES.—The most common causes of optic neuritis are encephalic diseases, and of these tumour is incomparably the most frequent. Neither the nature, size, nor the seat[1] of the tumour appears to exercise much influence on the occurrence of neuritis. The next most frequent cause is certainly meningitis, and then come abscess of the brain, cystic disease of the brain, and softening of the brain from embolism, when the plug comes from an inflamed valve.

[1] From an analysis of cases made by Edmunds and Lawford it would appear that, *ceteris paribus*, tumours near the convexity of the brain are somewhat less liable to cause optic neuritis than those situated near the base. Cerebellar tumours seemed particularly prone to excite optic neuritis, often of a severe type. Of twelve cases of primary tumour of the cortical motor area, on the other hand, not one was associated with optic neuritis ("Trans. Ophth. Soc.," vol. iv. 1884, p. 172). But there is no part of the brain in which a tumour may not cause optic neuritis. The relation needs a large number of cases to permit a conclusion to be reached.

In some cases it appears to result from an irritative process in the brain, revealed only by the microscope (*see* Part II., "Inflammation of the Brain"). It also accompanies, in rare cases, acute diseases of the spinal cord. Other causes, outside the nervous system, are—albuminuria, lead and tobacco poisoning, certain febrile diseases, anæmia (especially from loss of blood), and certain other morbid blood-states. It seems also to occur very rarely as an idiopathic affection, without obvious exciting cause, especially in gouty subjects. It has been thought to be due to disturbances of menstruation and exposure to cold. In all these cases it is usually double, but is not always simultaneous in the two eyes; now and then, in cerebral disease, and after acute febrile diseases and loss of blood, it may be single. Unilateral optic neuritis may occur from mischief in the orbit—inflammation, or a growth in or near the optic nerve.

In the general diseases, such as albuminuria, lead poisoning, anæmia, &c., optic neuritis is often associated with encephalic symptoms. In the case of lead poisoning shown at Pl. V. 6, the neuritis was associated with great mental excitement, and so also in the case of albuminuria, Pl. VII. 2, while in the similar case figured in Pl. VII. 3, intense headache was present. It seems probable that, in such cases, the neuritis and cerebral disturbance are due to a general effect of the toxæmia on the nerve tissues.

Duration.—The duration of optic neuritis varies very much in different cases. The cases of most rapid course may reach their height in three or four weeks, maintain it for about the same time, and then subside. These are of two classes—the most trifling, and the most severe. The former are, for the most part, those which depend on a cerebral condition which soon subsides, such as a transient attack of meningitis, or syphilitic or scrofulous disease, which is influenced by treatment before the neuritis reaches its height. Now and then, however, a neuritis rapidly subsides, although the cerebral disease progresses. But this is the rare exception. In these transient cases the subsidence may occupy the same

time as the development—two or three weeks—and be complete, so that at the end of six or eight weeks the discs are again normal. In some very intense cases, such as that of apparently chlorotic neuritis figured in Pl. VI., the development of the affection may be equally rapid, an intense degree of swelling being soon attained, and subsidence commencing in a few weeks; its diminution is slow, and often months pass before the edges of the disc are again perceptible. The longer a considerable degree has lasted, the slower is its subsidence. In two cases of cerebral abscess, where the pus was evacuated by an operation, the neuritis, which was great, but recent, subsided in a little more than a fortnight.

On the other hand, the course of neuritis may be so chronic that months, even a year, may pass without the least change in the condition of the discs being perceptible (*e.g.*, cases 23, 24 and 26 in first two editions of this book). Most cases of such extreme chronicity have accompanied symptoms of brain disease which were not, in themselves, suggestive of a tumour, or the like. But an equally chronic neuritis may accompany tumour, as in that shown, for instance, in Pl. III. 1 and 2. No alteration could be seen, on comparison with the drawing, for eighteen months, and two years later the neuritis was still marked, the red, congested half being little altered, although on the clearer half the disc had become grey, and sight was lost. It is to be noted that in this case the symptoms of tumour, although intense, also progressed very slowly, and the chronicity of the neuritis may probably be taken as an indication of chronicity of the cerebral disease. The converse proposition, however, that all forms of very chronic brain disease entail a chronic form of neuritis, does not, by any means, hold good.

In most cases the duration of neuritis is intermediate between the extremes mentioned, reaching its height in a month or two, remaining for some weeks or months with little change, and then subsiding. A rapid strangulation usually precedes subsidence at no long period, the products of inflammation perhaps themselves checking the inflammatory process by the compression of the vessels.

THE RELATION OF OPTIC NEURITIS TO ENCEPHALIC DISEASE.[1]—The first definite theory of the mechanism by which intra-cranial disease acts was put forward by von Graefe in 1859,[2] and further developed by him in 1866.[3] It was founded on the observation that in some cases of intra-ocular neuritis, with hæmorrhages, in cerebral tumour, no signs of inflammation were perceptible, on naked-eye examination, in the trunk of the optic nerve; whereas, in a case of meningitis, in which the ophthalmoscopic changes had been less intense, inflammation of the nerve trunk was found by Virchow—inflammation which was naturally assumed to have been communicated to the optic nerve from the inflamed meninges, and to have descended the nerve to the eye. This condition von Graefe designated "descending neuritis," and gave, as its characteristics, a slight degree of change in the discs and a tendency to invade the adjacent retina. On the other hand, the cases of tumour, with great intra-ocular change, hæmorrhages, &c., and no evidence of inflammation in the optic-nerve trunks, he explained by the theory that they were due to the effect on the circulation of the eye of the increased intra-cranial pressure, which he assumed to be invariable in these cases, and to obstruct the return of blood from the eye by compressing the cavernous sinus. He suggested further that this mechanical effect was greatly intensified by the rigid sclerotic ring, increasing the mechanical effect. Hence he applied to the condition—considerable swelling with hæmorrhage and vascular distension—the term "stauungs-papille" (*stauung*, a damming back), in distinction from the "descending neuritis." Clifford Allbutt introduced the term "choked disc" as a synonym for "stauungs-papille."

[1] The reader may be surprised to find so few references to researches on the pathology of optic neuritis since the appearance of the last edition. The reason for this is because the subject has not been carried farther. The uncertainty that surrounds it has been lessened by only one discernment. That is the important fact that neuritis subsides in most cases, if the intra-cranial pressure is diminished by early trephining. The significance of the fact is considered in the text.

[2] Société de Biologie of Paris, Nov., 1859 ("Gazette Hebdom.," 1859), and "Arch. f. Ophth.," vii. 1860, pt. 2, p. 58. [3] "Arch. f. Ophth.," xii. p. 100.

It was soon pointed out as strange that an actual inflammation should result from a mechanical congestion, and still more that it should be limited to the papilla. But graver difficulties awaited this theory of the "stauungs-papille." It was found that the ophthalmoscopic signs of the two forms of neuritis could not always be relied upon. The condition supposed to be characteristic of descending neuritis was discovered, in some cases, to be but the first stage of that supposed to indicate mechanical obstruction. It was found, also, that when the character of one of the two forms was clear, the necropsy might show the case to be really one of the other variety. Cases of this character were frankly published by von Graefe, although he still held that the distinctions were, in the majority of cases, accurate, and the supposed mechanism of the "stauungs-papille" effective. This theory, however, was destroyed in its substantive form, in 1869, by the demonstration by Sesemann[1] that the communication between the orbital and the facial veins was so free that the effect of pressure on the cavernous sinus was at once relieved, and did not cause more than a very transient fulness of the retinal veins, and that even obliteration of the cavernous sinus produced no intra-ocular changes. This has since been well corroborated, as, for instance, by a case recorded by Hutchinson, in which no distension of the retinal veins was produced, although the cavernous sinus was completely obliterated by the pressure of an aneurism. It has, indeed, been said that the openings from the orbital into the facial vein are often larger than the communication with the cavernous sinus.

It was discovered by Schwalbe[2] that the subvaginal space around the optic nerve is, at the optic foramen, continuous with, and can be injected from, the subdural space around the brain.[3] This gave significance to some earlier observations of

[1] "Reichert u. Du Bois Reymond's Archiv," 1869, p. 154.

[2] "Centralblatt f. Med. Wiss.," 1869, p. 465. "Arch. f. Mikroskop. Anat.," Bd. vi. 1870, p. 1.

[3] It has been stated by Parinaud that the communication is with the subarachnoid, not with the subdural space ("Ann. d'Oculistique,' vol. lxxxii. 1879, p. 25).

Stellwag von Carion[1] and Manz[2], that the sheath of the nerve may be distended in optic neuritis from tumour and meningitis. The two facts suggested to Schmidt[3] that intra-cranial pressure may influence the intra-ocular termination of the optic nerve by this mechanism, since, as already described, the distension of the sheath is greatest just behind the globe. The theory received support from Manz,[4] who showed how frequent is distension of the sheath in optic neuritis, and believed it to be invariable in cases of increase of intra-cranial pressure or increase of subarachnoid fluid. He urged that the simple pressure on the nerve and vessels might cause the intra-ocular changes, and endeavoured, by experiment on animals, to demonstrate this effect of the vaginal distension. Injections into the subdural space passed into, and distended, the sheath, and caused fulness of the retinal veins, and in some cases transient redness and swelling of the papilla.

Schmidt, however, found that a coloured liquid injected into the sheath passed into lymph spaces in the nerve at the lamina cribrosa, and he suggested that neuritis is produced, not by the simple pressure outside the nerve, but by the influence, perhaps irritation, of the liquid passing into these lymph spaces. The theories of Schmidt and Manz have been largely accepted in Germany as affording the most satisfactory explanation of the origin of optic neuritis. Leber[5] while adopting the view that the distension of the sheath is the immediate excitant of neuritis, doubts the theory of Manz, that the fluid acts by mechanical pressure, and rejects the effect on the lymphatic spaces assumed by Schmidt, on the ground that his own and other investigations have failed to confirm the asserted communication of these spaces with the sheath. Leber suggests that the fluid in the sheath excites neuritis, by conveying pathogenic

[1] "Ophthalmologie," vol. ii. 1856, p. 612.

[2] "Zehender's Monatsbl.," vol. iii. 1865, p. 281.

[3] Of Marburg, now Schmidt-Rimpler. "Arch. f. Ophth.," vol. xv. 1869, p. 193.

[4] "Deutsch. Arch. f. Klin. Med.," vol. ix. 1871, p. 339.

[5] Discussion at the International Medical Congress, London, 1881.

material to the optic nerve behind the eye. Deutschmann[1] has published experimental evidence in favour of Leber's view, and in opposition to the theory of "choked disc" from the distension of the nerve-sheath.

It was suggested in 1863 by Hughlings-Jackson,[2] that intra-cranial tumour causes optic neuritis by its irritating effect, acting as a "foreign body"; and this view was supported a little later by Brown-Séquard, who compared the origin of neuritis in intra-cranial tumour to the production of atrophy of the optic nerve by a distant source of irritation. It was more precisely formulated by Benedikt[3] in 1868, by ascribing the mechanism to the vaso-motor nerves, and it is sometimes termed his theory.[4] This view assumes that the tumour acts as a source of irritation, producing a reflex influence through the vaso-motor nerves upon the optic disc, and thus leading to its inflammation. It has been rejected by most writers on the grounds stated by Leber,[5]—that it involves a mechanism not known to exist, and a complex relation of the optic nerve to all parts of the brain difficult to conceive; and by Clifford Allbutt, on the ground that he has failed to find around tumours the signs of irritation. The theory is, however, still held by Hughlings-Jackson as that which best explains the phenomena of neuritis; he has always urged that the occurrence of optic neuritis is not related to increased intra-cranial pressure. Galezowski believes that neuritis is always descending, and first maintained, contrary to previous observers, that the intra-ocular change is in all cases the visible manifestation of an inflammation propagated by continuity from the

[1] "On Optic Neuritis, especially the so-called Choked Disc, and its connection with Brain Diseases." Jena, 1887, and "Ophth. Rev.," vol. vi. 1887, p. 107.

[2] "Ophth. Hosp. Rep.," vol. iv.

[3] "Allg. Wien. Med. Zeit.," 1868, No. 3.

[4] Schneller, in 1860, put forward a similar theory when he suggested that some retinal changes in intra-cranial disease might be due to a "primary affection of the centres of those nerves which regulate the course of the blood in the ocular vessels."—"Arch. f. Ophth.," Bd. vii. 1860, I. p. 71.

[5] In "Graefe u. Saemisch's Handbuch," Bd. v.

brain. Edmunds and Lawford came to the conclusion that optic neuritis, when due to an intra-cranial cause, is secondary to basal meningitis, and that the inflammation reaches the substance of the nerve-trunk through its sheath.[1] Lastly, Parinaud[2] asserts that neuritis is invariably the effect of distension of the ventricles of the brain, which causes general cerebral œdema, and of this both the distension of the sheath and the papillitis are equally part.

The clinical and pathological evidence bearing on these views may be briefly reviewed.

The first point to be borne in mind is that optic neuritis limited to, or at least most intense in, the optic papilla, may occur without any obvious intra-cranial disease. The intense neuritis shown in Pl. VI. 1, was apparently a primary papillitis, involving the retina only secondarily, due to anæmia. This patient had no symptom of cerebral disease, save some headache, during two years she remained under observation. The neuritis reached its height in about a fortnight from its commencement, a rapidity which is seen in neuritis from intra-cranial disease only in the most acute cerebral affections; this circumstance, with the absence of cerebral symptoms, excludes the supposition that there existed intra-cranial disease. Limited papillitis is now known to occur in simple anæmia. From these considerations it seems to follow that the intra-ocular termination of the optic nerve is a structure, for some reason which we do not know, peculiarly prone to suffer inflammation. The common localization of the inflammation to the papilla points also to the same fact.

The facts of medical ophthalmoscopy certainly make it difficult to connect the commencement of papillitis with mere increase of intra-cranial pressure. If we consider the cases in which intra-cranial pressure is raised to the highest point it ever reaches—chronic hydrocephalus—we find optic

[1] "Trans. Ophth. Soc.," vol. i. p. 111; vol. iii. p. 138; vol. iv. p. 172; vol. v. p. 184; vol. vii. p. 208.

[2] *See* "Graefe u. Saemisch's Handbuch," Bd. v.; "Ann. d'Ocul." t. lxxxii. p. 5.

neuritis the rare exception, and, when it occurs, never intense. The difficulty cannot be met by attributing it to the slowness with which the pressure is raised, because the growth of many tumours, which cause intense optic neuritis, is equally slow. On the other hand, as I have many times seen, in cases of tumour with neuritis there may be no sign of increased intra-cranial pressure during life or after death. "In these cases of vast tumours, the optic neuritis does not differ from that caused by small tumours at the vertex of the brain, which cannot exercise pressure of any consequence at the base." "The neuritis runs through its stages, and the swelling of the discs subsides, although the intra-cranial pressure goes on increasing."[1] There may also be signs of increased pressure in tumour without optic neuritis. But, while pressure upon the cavernous sinus cannot be regarded as the cause of neuritis, its influence on the retinal vessels cannot be altogether excluded. Experiments show that a quickly induced increase of pressure within the skull causes a transient distension of the retinal and papillary vessels. In tubercular meningitis (*q.v.*) Garlick's careful observations[2] have shown that fulness of veins is related to an increased intra-cranial pressure, while papillitis is not.

At the same time, observations show conclusively that a relief of intra-cranial pressure, as by surgical interference, is often followed quickly by a subsidence of the swelling in a previously existing papillitis. Moreover, it is at least probable that high intra-cranial pressure is in itself capable of producing œdematous swelling of the papilla in some cases of brain tumour.[3]

In the course of a descending neuritis the distension of veins may be very great, as I have several times observed both during life and after death (*see* Figs. 8 and 26). In tumour the veins at first, and often throughout (when the

[1] Hughlings-Jackson: Lecture on Optic Neuritis, "Med. Times and Gaz.," 1871, vol. ii. p. 581.

[2] "Med.-Chir. Trans.," vol. lxii. 1879, p. 441.

[3] Horsley: 'Brit. Med. Jour.," 1893, vol. ii. p. 1365; James Taylor: 'Trans. Ophth. Soc.," vol. xiv. p. 105.

neuritis does not reach a considerable degree of intensity), are little above the normal size, and present no tortuosities except those which are given them by the prominence of the papilla. The great distension of veins and narrowing of arteries occur when the inflammation has reached a considerable degree of intensity. This points to the neuritic process in the papilla as causing the strangulation by pressure on the vessels. This view is entirely borne out by pathological investigation. I have never been able to discover any evidence of constriction of the vessels in the sclerotic ring or behind it. Their calibre here is always uniform (*see* Figs. 18, 23, and 25). This statement is based on a very careful search for any evidence of such compression in a number of cases of papillitis from various intra-cranial diseases. In one case only was there an appearance of narrowing, and in this, from the unaltered course of an adjacent vessel, it was evidently due to a slight alteration in the position of the vessel at the spot, in consequence of which the sections ceased to pass through its widest part. It is always in front of the sclerotic, in the substance of the swollen papilla, that the vessels present conspicuous constriction—are pressed upon, and have their walls thickened by new tissue (Figs. 12, 24, &c.). Further, the most intense signs of "strangulation" may be seen in cases in which, as in that of neuro-retinitis due to chlorosis (Pl. VI. 1), there is reason to believe there is no intra-cranial disease; and in the case of chronic cerebritis, quoted at p. 85, in which there was no intra-cranial condition which could cause any mechanical effect, the intra-ocular signs of constriction and mechanical congestion were very marked.

Distension of the optic sheath is certainly very frequent in cases of optic neuritis. It is not, however, as has been alleged, invariable, either in cases of cerebral tumour with optic neuritis, or in conditions of increased intra-cranial pressure. It may be absent in tumour of the brain with characteristic neuritis; for instance, in one case a large glioma of the right frontal lobe, with hæmorrhage into it, was attended by optic neuritis, but with no distension of the

sheath. This may also be absent in tumour with internal effusion; great distension of the lateral and third ventricles was caused, in another case, by a tumour near the corpora quadrigemina,—there was optic neuritis, but no distension of the sheaths. A case of tumour of the cerebellum, with optic neuritis and no distension of the sheaths, has been recorded by Nettleship.[1] In another case of old neuritis, due to a tumour occupying the whole of the third ventricle and inter-peduncular space, extending in front of the optic commissure and causing enormous distension of the lateral ventricles, the optic sheaths carefully examined in situ were quite empty, but much enlarged. They had evidently been much distended. This case suggests that pressure at the base of the brain may even be incompatible with continued distension of the sheath. Distension was absent in a case of neuritis from cerebral abscess recorded by Carrier.[2] In tubercular meningitis, again, the condition to which the distension of the sheath appears to be related is not distension of the ventricles, or increased intra-cranial pressure, but increase of the subarachnoid fluid, and a relation to the occurrence of neuritis is not clear. Of six cases with changes in the papilla, in which the state of the optic sheaths was carefully noted by Dr. Garlick, in four, in which excess of subarachnoid fluid was absent, the sheath was normal, although in several there was great distension of the ventricles, while in the remaining two cases, in which there was an excess of subarachnoid fluid, there was also dropsy of the optic sheath. A case of neuritis in tubercular meningitis, without distension of the sheath, has also been described by Edmunds.[3] It has been suggested that the fluid may be merely prevented passing backwards to the cranium by the intra-cranial pressure. If the sheath of the optic nerve is the chief lymph-channel by which fluid is conveyed away from the eye, its distension in optic neuritis, by fluid escaping from the papilla, is intelligible.

[1] "Path. Trans.," 1880, p. 252.
[2] "Philadelphia Med. Times," Jan. 29, 1880.
[3] "Trans. Ophth. Soc.," vol. i. 1881, p. 112.

But this fact is, at least, uncertain: the case of tumour in the third ventricle, referred to in the last page, seems opposed to this theory. The optic sheaths had been greatly distended, but they had become empty, apparently in consequence of the pressure on the front of the base, by the large tumour in the anterior part of the third ventricle, cutting off the communication with the subarachnoid space. If the fluid were derived from the eye, this influence should have increased the distension of the sheaths, instead of causing them to become empty. There is thus strong reason to believe that the fluid in the sheath of the optic nerve passes into it from the subarachnoid space. The absence of obvious excess of the subarachnoid fluid in some cases, as in an instance mentioned by Edmunds,[1] is not of much weight as evidence against this conclusion, because a general increase of intra-cranial pressure (*e.g.*, by ventricular effusion) which would assist in forcing the fluid into the sheaths, might at the same time tend to remove it from the base of the brain, where its amount is estimated.

Of the frequent association of dropsy of the sheath and optic neuritis there can be no doubt. Nor can there be doubt of the influence of intra-cranial pressure shown by the subsidence of neuritis when the pressure is lessened even by simple trephining. It is certain, too, that distension of the optic nerve-sheath may occur even in meningitis without causing neuritis, as in a case recorded by Broadbent. But such cases prove little, because the duration of the dropsy may not have been sufficient for the inflammatory changes to arise. The occasional occurrence of papillitis without it—a fact which is well established—shows that it is not the invariable, and suggests that it is not the chief, mechanism by which papillitis is produced. But it is probable that although not the chief cause, it exerts an important influence on the process.

In examining the trunk of the optic nerve behind the eye,

[1] "Trans. Ophth. Soc.," vol. i. 1881, p. 112.

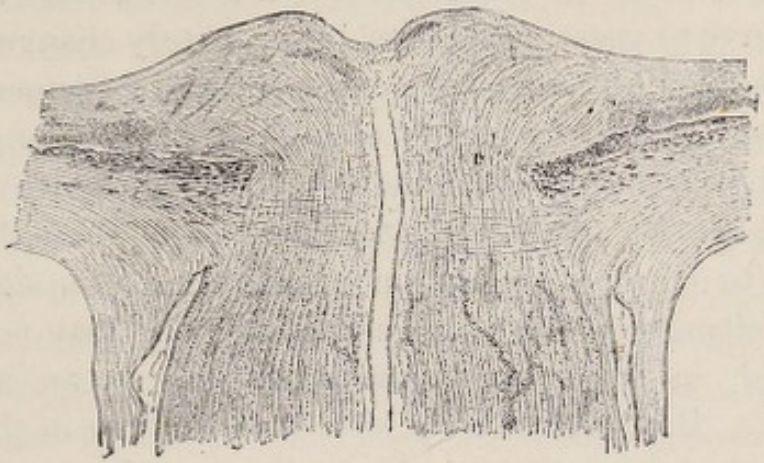


FIG. 44.

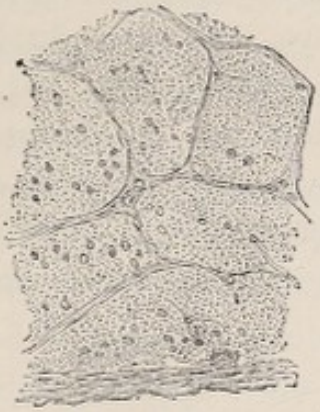


FIG. 45.

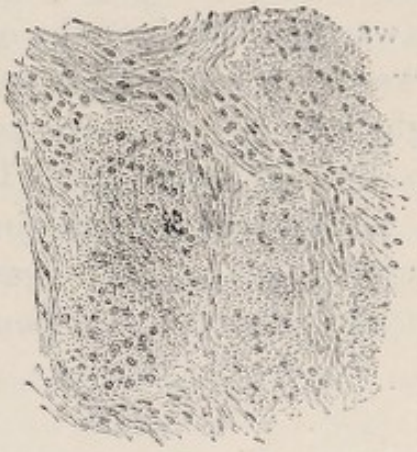


FIG. 46.

SECTIONS FROM A CASE OF OPTIC NEURITIS

Due to a tumour in the frontal lobes.

FIG. 44.—SECTION THROUGH THE DISC. Swelling of the papilla, displacement of the retina outwards. Aggregation of leucocytes along the course of the vessels, thus indicating their position. No sign of compression of vein when passing through the sclerotic ring. No distension of the sheath of the nerve. (× 15.)

FIG. 45.—PART OF A TRANSVERSE SECTION THROUGH THE OPTIC NERVE MIDWAY BETWEEN THE GLOBE AND THE OPTIC FORAMEN. Increase of nucleated cells and some degeneration in the nerve fibres. (× 100.)

FIG. 46.—TRANSVERSE SECTION JUST IN FRONT OF THE COMMISSURE. The bundles of nerve fibres are separated by much newly formed fibrous tissue, which is encroaching on the fasciculi of the nerve. The nerve fibres are degenerated, and many nuclei are scattered among them. (× 100.)

in cases of papillitis from cerebral tumour, I have found the optic nerve to present traces of inflammatory change, increase of nuclei and interstitial tissue, much more frequently than has been stated by other observers. The changes were especially marked towards the periphery of the trunk and in the pial-sheath. In not one case examined could the nerves be said to be in a perfectly normal state. The significance of the changes is open to question. They may possibly be regarded, in some cases, as indications of an ascending neuritis. But in some they were most intense in the neighbourhood of the optic commissure (compare Figs. 45 and 46), and there was evidence that neuritis had developed there by extension from the meninges. In several cases in which this was most distinct, the change in the optic nerve, midway between the commissure and the eye, was so slight that it might almost have been passed as normal (Fig. 45). (See also Figs. 26 and 29.) The indications of extension to the nerve were very distinct in two cases of intra-cranial tumour. In one there was distinct, although very slight, evidence of meningitis beneath the orbital lobes, and the

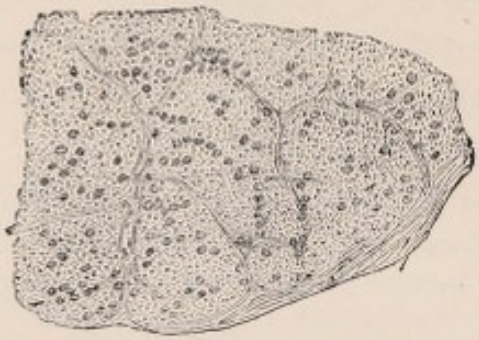


FIG. 47.

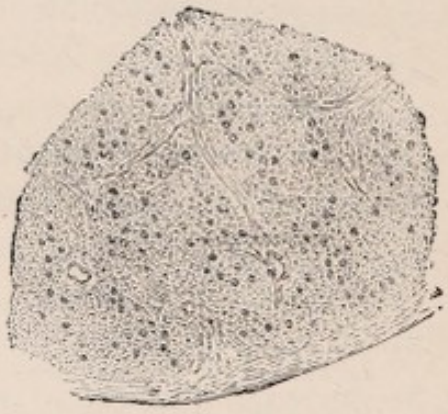


FIG. 48.

SECTIONS THROUGH THE OPTIC NERVE IN A CASE OF CHRONIC CEREBRITIS. (× 100.)

FIG. 47.—Just behind the globe, containing many nuclei within the fasciculi.

FIG. 48.—Just in front of the commissure. There is more infiltration with nuclei, and the connective tissue septa are more thickened and irregular.

inflammation had apparently extended to the nerves. In the other case (Fig. 44) the papillitis was considerable, and such as is common in cerebral tumour. The changes in the optic nerves in the middle of their course were slight but distinct (Fig. 45). In front of the optic commissure (Fig. 46) the changes from old inflammation were intense. No sign of adjacent meningitis was noted at the post-mortem examination, but there were old adhesions over the tumour at the upper part of the frontal lobe. In this case it seems probable that descending neuritis had taken place, and that the link between the intense neuritis behind, and the intense papillitis in front, was the slight change in the trunk of the nerve. Hence it seems that a very slight amount of descending change may lead, in cerebral tumour, to an intense papillitis. With this may be compared another case, under the care of Dr. Hughlings-Jackson, in which the appearance of the papilla was declared by Mr. Couper to be that of a "choked disc." No tumour, meningitis, or mechanism for "choking" was, however, discovered within the cranium. But the trunk of the nerve presented changes very similar to those in the case just mentioned, not, however, more intense at its posterior portion. Similar changes were found throughout the brain by Dr. Sutton, and it can hardly be doubted, taking the symptoms and anatomical changes together, that a condition of chronic inflammatory character, affecting the brain, had passed down the optic nerves and appeared as an intense papillitis, with signs of strangulation. The latter may have been due to the compression of the vessels within the papilla. A similar case has been recorded by Stephen Mackenzie.[1]

It seems from these facts that (1) a descending neuritis cannot be excluded from an examination of a small portion of the trunk of the nerve, and (2) that a change in the nerve revealing itself as a very slight deviation from the normal,

[1] "Brain," July, 1879, p. 269.

may serve to convey a condition of irritation to the eye, sufficient to light up considerable papillitis.

The frequency with which evidence of descending neuritis may be traced is confirmed by the observations of S. Mackenzie,[1] Brailey,[2] Edmunds and Lawford,[3] Poncet,[4] and others. Mackenzie has also pointed out that on no other theory than that of an inflammation travelling down the nerve tissue can we explain a unilateral neuritis on the side opposite to a cerebral tumour.

It has been maintained by Kuhnt[5] that the descent of inflammation from the brain to the eye is by the perivascular sheaths of the vessels, which are, he states, continuous with the pia mater of the brain, and Gayet[6] would ascribe a share also to the sheaths of the posterior ciliary vessels. The evidence of inflammation away from the vessels prevents us, however, regarding them as the exclusive agents, but pathological evidence of their participation in the transmission of the inflammation has been also brought forward by Edmunds and Brailey.[7]

It has been pointed out that the sheaths of the nerve, inner and outer, often present considerable changes, which make it probable that the inflammation passes along them to the eye. That it may do so independently of distension of the sheath is shown by two cases of optic neuritis and meningitis due to fracture of the skull, recorded by Edmunds,[8] in which the space between the sheaths of the nerve was occupied by "a dense mass of inflammatory products."

These facts suggest the following conclusions regarding the production of papillitis in intra-cranial disease:—

That in cases of cerebral tumour, evidence of descending

[1] Loc cit. and "Trans. Ophth. Soc.," vol. i. p. 94.

[2] "Trans. Ophth. Soc.," vol. i. p. 111.

[3] Ibid., p. 112.

[4] Disc. at the International Med. Congress, 1881.

[5] In a communication to the International Medical Congress at Amsterdam ("Ann. d'Oculist.," vol. lxxxii. 1879, p. 180). [6] Ibid., p. 181.

[7] "Ophth. Hosp. Rep.," vol. x. p. 138.

[8] "St. Thos. Hosp. Rep.," vol. xi. 1881, p. 71. "Trans. Ophth. Soc.," vol. iii. p. 14

inflammation may be traced in sheath or nerve, much more commonly than current statements suggest, while in cases of meningitis the evidence of such descending inflammation is almost invariable.

That the resulting papillitis may be, and remain, slight, or may become intense and present the appearances of mechanical "strangulation." The causes of this difference we do not yet know.

That such mechanical congestion does not probably result from mere compression of the vessels in, or just behind, the sclerotic ring, but always, when intense, from compression by inflammatory products in the substance of the papilla.

That while very slow increase of intra-cranial pressure has no effect on the retinal vessels, a rapid increase hinders the escape of blood and lymph from the eye, and intensifies a papillitis, whatever its origin. Relief to the pressure is often attended by a quick subsidence of the papillitis.

That distension of the sheath of the nerve alone is probably sufficient to cause papillary œdema by its mechanical effect, and may intensify the process otherwise set up, especially if the fluid possesses an irritative quality, and if (as Schmidt-Rimpler asserts and Leber denies) it can find its way into the lymphatic spaces of the optic disc.[1]

In this outline of the facts regarding the origin of optic neuritis, the hypothesis that the mechanism is a reflex vaso-motor influence has been necessarily unnoticed, because the known facts have no bearing upon it and give it no support. It presupposes a special reflex relation not known to exist, and a mechanism for the production of inflammation the efficiency of which is equally unknown.[2]

VARIETIES.—The chief varieties which have been usually

[1] The theory of Leber, viz., that papillitis is an extension of inflammation from the periphery of the nerve at the anterior extremity of the sheath, is not supported by any anatomical evidence.

[2] A fuller consideration of the theory, and the arguments against it, will be found in some remarks I made in the discussion on optic neuritis at the Ophthalmological Society, March 10, 1881 ("Transactions," vol. i. p. 105). Similar arguments were brought forward by Leber at the dis-

insisted on, are those distinguished by v. Graefe as "descending neuritis" and the "choked disc." The facts, as already mentioned, make it doubtful whether the pathological basis of the distinction is correct, and it is generally admitted that it cannot be relied upon. The aspect of the disc varies in the same case, at one time being that of a "descending neuritis," and at another time of a "choked disc." But the appearance may continue different throughout. The intermediate forms are such as to make it difficult to separate any varieties as special "forms." The characters regarded as those of descending neuritis are seen, for instance, in Pl. I. 3 and 5, and, of a wider extent and greater intensity in Pl. IV. 2. On the other hand, great swelling, vascularity, and distended veins, such as are seen in Pl. I. 4, and still more in Pl. IV. 1, characterize other forms. But in the case shown in Fig. 18, descending neuritis presented the characters of the choked disc, while the changes in Pl. III. 1 and 2, 3 and 4, slight as they are, were in each case associated with the symptoms of intra-cranial tumour.

Until we know more of the relation between pathological process and ophthalmoscopic appearance, it seems far better to found varieties purely on clinical characters. Of varieties so founded the following seem the most marked.

1. *Slight Papillitis*, including minor degrees of the condition described above as congestion with œdema, in which the changes are so slight as to dim, but not obscure, the edge of the disc on indirect examination, rendering it invisible, wholly or in part, to direct examination (Pl. III. 1, 2, 3, 4).

2. *Moderate Papillitis.*—Obscuration of the edge of the disc, or of the affected portion, complete, even to indirect examination; swelling moderate, commonly reddish; veins natural or large (Pl. I. 4, II. 1, 3, III. 5, 6, IV. 2).

cussion at the International Congress. The reflex theory has been revived by Loring ("New York Med. Journ.," June, 1882) in special connection with the fifth nerve, but still as a pure theory, which, while unsupported by facts (and even opposed by them), clearly merits detailed discussion.

3. *Intense Papillitis.*—Great swelling; veins at first large and arteries small; many hæmorrhages; the adjacent retina often involved by direct damage or by hæmorrhages. Always succeeds a slighter stage in which the evidence of strangulation may be at first little marked (Pl. IV. 1, VI. 1).

The forms in which the changes involve the adjacent retina have been termed "neuro-retinitis;" and such widespread change as is presented in Pl. VI. 1, although originating in the papilla, merits such a designation. But in most cases, even in such as Pl. IV. 1, the retina is only affected adjacent to the papilla; and the term "retinitis" seems unnecessary.

Retro-ocular Neuritis.—Consecutive pallor of the disc is not infrequently the first ophthalmoscopic sign of the disease, but this is often preceded by papillitis, which may be slight, moderate, or intense. The resulting atrophy is usually comparatively slight, but varies much in degree. Not infrequently the pallor of the disc is much in excess of what we should expect, judging from the amount of recovery of vision. But sometimes there is complete atrophy, with narrowed vessels, and blindness. The anatomical changes doubtless vary considerably, according to the nature of the attack, while opportunities for examining the nerve during the acute stage seldom occur. The connective tissue of the septa has been found thickened and vascular, with consequent pressure on the nerve fibres. In later stages, after subsidence of the inflammation, the thickened septa may diminish, with relief of pressure and return of function, or there may be a general contraction of this tissue, accompanying permanent atrophy of the nerve fibres. The nerve is most liable to be attacked in the optic canal, or just behind the eyeball, and therefore the pathological changes are most marked in one or other of these localities. In either case it is the "macular bundle" that is usually mainly involved, thus accounting for the central scotoma clinically recognized.

Diagnosis.—The diagnosis of optic neuritis is often easy,

but sometimes presents great difficulty. Of all its signs that which first attracts attention as the most conspicuous feature—the increased redness—is of least value, except in conjunction with other characters. As already more than once stated, the redness of a disc free from neuritis may nearly equal that of the adjacent choroid. The signs which are of greatest diagnostic value are (1) obscuration of the edge of the disc and (2) swelling. These, in conjunction with observed increase in redness, or change of colour, constitute the characteristic symptoms. The obscuration of the edge is especially significant. It indicates undue opacity of the tissue (layer of optic nerve fibres) in front of the edge, as already explained. Where the nerve fibres accompany the chief vessels, above and below the disc, they, in health, often slightly obscure its edge. When they are densely packed on the nasal side, especially if the central cup is very large, a slight obscuration is produced there also; but in these cases, as a rule, the large size of the physiological cup indicates the close arrangement of the fibres, the obscuration is slight, and the edge of the disc is elsewhere quite sharp. In such cases another character may occasionally be observed in a slight degree, which, in more intense form, is conspicuous in neuritis—the radiating striation at the edges of the disc. Normally this is seen where the nerve fibres are most closely aggregated, especially above and below; in morbid states it is to be observed all round the disc, although most intense where the nerve fibres are grouped, and it is then due not merely to pale lines (from swollen fibres with increased opacity), but in part, also, to red lines, fine vessels lying between the fibres.

The second indication of neuritis is the existence of distinct swelling. The prominence of one object in the fundus above the level of an adjacent object—*e.g.*, of a vessel on the edge of the physiological cup above a vessel at its bottom—is appreciated in the direct method of examination by moving the head of the observer from side to side, or up and down, as far as possible without losing sight of the objects. Their

relative position undergoes an appreciable alteration proportioned to the difference in level, and is easily recognized. By the indirect method of examination the same result may be obtained by a lateral or vertical movement of the lens, which produces the same effect as a corresponding movement of the observer's head (the "parallactic test" of Liebreich).

When the difference of the level of two objects is considerable, as, for instance, with much swelling of the disc, a convex lens behind the mirror may be necessary before a clear view of the top of the swelling is obtained, the refraction of the eye being normal and the fundus visible without a lens. In all cases the difference between the strength of the convex lenses required to render objects indistinct on the level of the retina and on the apex of the swelling, furnishes a measure of the height of the swelling.[1] Normally the surface of the papilla is a very little anterior to the plane of the retina, hence the term "papilla." The amount of this prominence varies in different cases. It is always greater where the nerve fibres are chiefly aggregated in the proximity of the retinal vessels, above and below, so that a transverse section through the disc may show scarcely any appreciable prominence, while a vertical section may present distinct prominence. The more closely the nerve fibres are aggregated in one part of the circumference of the nerve, the greater is the prominence.

Another important sign of swelling is the apparent darker tint of the vessels, especially the veins, as they pass over its sides, ceasing to be at right angles to the line of sight.

The diagnosis of retro-ocular neuritis cannot, as a rule, be made by the ophthalmoscope until a comparatively later stage of the affection. There is at first visual failure, particularly at or near the centre of the field, with sluggish reaction of the pupil to direct light-exposure. Commonly only one eye

[1] The rule commonly applied is of sufficient accuracy, viz., that each diopter of difference in the lenses necessary, is equivalent to a difference of level of 0·3 mm. Thus, if the surface of the swollen disc is focussed by +5 D, while the retina is seen best with 0, the difference of levels of 5 D equals 1·5 mm.

is affected at a time, and there may be considerable tenderness on pressing the globe backwards, or on muscular movements of the eyeball. When vision begins to recover, and central loss to white disappears, a red-green central colour scotoma may still be present.

The Diagnosis of the Cause of Papillitis.—The first question which presents itself in a given case is—Is the neuritis due to intra-cranial disease or to some other cause? The answer to this must, of course, depend on the presence or absence of indications of disease of the brain, or of such disease of the general system as is known to be accompanied by optic neuritis. The ophthalmoscopic characters of the neuritis will lead us a little way, but not far. A high degree of neuritis, with intense strangulation (such as the discs shown in Pl. IV. 1 and VI. 1), is seldom met with except in cases of cerebral tumour and rare forms of primary neuritis. The slighter degree of neuritis not uncommon in cerebral tumour, chronic meningitis, and other intra-cranial diseases, and the neuritis which occurs in Bright's disease, lead poisoning, &c., may bear a close resemblance. That of Bright's disease may present white spots in and close to the disc, but the same appearance is often seen in the neuritis of intra-cranial disease. (Fig. 7.) White spots in the retina away from the disc, with papillitis of a slight degree, and presenting no evidence of a preceding more intense affection, is strongly suggestive of renal neuritis. The small cloudy spots seen, for instance, in Pl. VII. 2 (near the left edge of the figure), are of more significance than the minute white spots near the macula, such as are shown in Pl. VII. 3, although the latter are suggestive of renal disease when they occur with a papillitis of slight degree and recent origin. Succeeding neuritis, or accompanying a neuritis which is subsiding, they are of much less significance, being often the relics of the mischief caused by simple inflammation; and how closely these may simulate the appearance of a renal retinitis is shown by Fig. 6 and Pl. VI. 2. Although an appearance of so striking an aspect is very rare, a few white spots near the macula lutea are often left by neuritis—such as are seen in

Pl. IV. 3. The signs of a previous neuritis of considerable intensity—a prominent mass of tissue in front of the disc such as is seen there in Fig. 3, or a "filled-in" disc with evident compression of vessels, as in Pl. VI. 2—rarely coincide with a similar appearance in renal retinitis, although such a coincidence is seen in Pl. VII. 4. In such a case as is there figured, the diagnosis of the cause of the neuritis could scarcely be made by the ophthalmoscope alone. But attention must always be paid to the degree of the present inflammation, or the evidence of its degree in the past, afforded by the amount of new tissue formed. So far as ophthalmoscopic observation alone can guide us, the most accurate means of deciding whether the neuritis is of renal or other origin, is probably afforded by attention to the appearance of the retinal vessels. The changes in them, so often met with in renal disease, have been briefly described on p. 14.

It is upon the independent signs of one or the other causal condition that the diagnosis must chiefly turn. In referring neuritis to cerebral mischief, it must not be forgotten that, on the one hand, optic neuritis due to a cerebral tumour may be accompanied for a time by no signs of intra-cranial disease, and, on the other hand, that an optic neuritis due to a general disease may be accompanied by symptoms suggestive of cerebral disturbance, especially headache, vomiting, and even, in some cases, convulsions. The suspicion of intra-cranial disease in cases of optic neuritis can only be discarded after long observation, if indeed it can ever be given up until some other cause presents itself. This is especially the case when the neuritis is chronic: very acute neuritis is nearly always accompanied by symptoms indicative of the disease causing it.

Neuritis due to general disease and accompanied by symptoms suggestive of cerebral mischief, is illustrated by Pl. VII. 3, from a man who complained of almost constant severe headache and occasional attacks of sickness. The ophthalmoscope showed well-marked neuritis, moderate in degree, and on first inspection no retinal disturbance was

detected. It was thought, for the moment, to be a case of cerebral tumour. On looking more carefully by the direct method, near the macula lutea were seen a number of minute white spots inconsistent with the slight degree of neuritis. The urine was at once examined, and found to be loaded with albumen, and on further examination, hypertrophy of the heart and a hard pulse were found, with some signs of uræmic mischief. He died of uræmia not long after. The history of the case shown at Pl. VII. 2 is similar, except that the evidence of cerebral disturbance here was mental change, not headache. Another case impressed itself very strongly upon me many years ago, when, as a resident in University College Hospital, I was first working with the ophthalmoscope. A man was admitted with convulsions, and comatose. An examination of the eyes showed double optic neuritis, and a diagnosis of cerebral tumour was at once ventured on. The patient died in a few hours, and the necropsy revealed contracted kidneys and a normal brain. Examination of the urine should never be neglected.

Headache and vomiting then, do not constitute proof of an intra-cranial cause of neuritis. Convulsion, alone, is also of little value unless it is of a form which indicates local brain disease, *i.e.*, local in distribution or in commencement.

In all obscure cases, search must be made for any other cause of optic neuritis, especially lead poisoning. In cases of lead poisoning renal disease is very frequent, and that cause of neuritis must be excluded before the affection can be referred to plumbism. In these cases also, doubt may be felt as to whether the mischief is not due to cerebral disease, because lead poisoning is sometimes accompanied with two forms of cerebral disturbance—delirium and convulsion. In the case presenting the neuritis shown in Pl. V. 6 there was extreme cerebral disturbance, apparently due to this cause.

One other fact must be mentioned in connection with the diagnosis of the cause of optic neuritis. In many cases in which slight neuritis of chronic course is associated with

symptoms which would scarcely suggest the existence of disease such as would cause neuritis, hypermetropia exists. This combination may be noted, for instance, in chlorosis (as in the case figured in Pl. V. 5), in epilepsy, in chorea, and in association with other slight symptoms of cerebral disturbance. It is known that, in hypermetropia, prolonged close work may occasion considerable congestion of the optic discs. Its frequent presence in optic neuritis suggests, further, that hypermetropia may be a determining factor in producing and aggravating inflammation of the nerve when other causes are present.

Prognosis.—The prognosis in optic neuritis is often a source of anxiety. In few cases can it be said that vision is not in danger of impairment and even of loss. The prognosis must be formed by a careful study of the conditions on which impairment of sight depends, as already stated. The prospect is better in the slighter degrees of papillitis, and better in proportion to chronicity of course, and dependence on causes which can be influenced by treatment. It is worse when there is reason to believe that there is also disease behind the papilla; worse in proportion to the evidence that there is a process of compression in the papilla; worse in proportion to the intensity of the changes; and worse when increased loss of sight comes on during the recession of inflammation which has already impaired vision during its height.

The cause of the optic neuritis must especially influence our prognosis. It is better in syphilitic than in scrofulous cases, and better in these than in cases of disease of other forms. Even in syphilitic mischief, however, the prognosis must be guarded if the intra-ocular changes are considerable. It is not probable that the optic neuritis is, itself, syphilitic in nature. Its subsidence depends rather on the subsidence of the syphilitic intra-cranial disease, than on the direct influence of treatment on the intra-ocular process, and it is not uncommon to have considerable failure of sight, and even complete loss, when neuritis from such disease has

attained an intense degree. Fortunately when the subsidence of the neuritis has ceased, there is a tendency to some improvement of vision, and this may be considerable in amount (*see* "Consecutive Atrophy").

TREATMENT.—Very little can be done for the direct treatment of optic neuritis. The treatment is that of the intra-cranial mischief, or general disease, which is its cause. Beyond this, local measures, leeches and the like, are little likely to influence the progress of the disease. Puncture of the distended nerve-sheath has been advocated by De Wecker, and performed by him and by Mr. Brudenell Carter. It is based on the theory that the distension of the sheath is the cause of the intra-ocular neuritis, a theory which, it has been seen, cannot yet be considered as proved. Although improvement followed the operation in a few cases, the results have not justified the procedure. In cases where there is definite optic neuritis, with retention of good vision, favourable results have followed the relief of intra-cranial pressure by the operation of trephining.[1]

During neuritis the eyes should be used as little as possible, and such conditions as intensify intra-ocular congestion should be avoided, *e.g.*, exposure to cold, and all causes of mechanical congestion, straining, cough, &c. Ice to the forehead has been recommended by Pflüger.

Optic neuritis is so frequently associated with syphilitic disease of the brain and its membranes, and the evidence which may seem to exclude the suspicion of syphilis is so often misleading, that the administration of iodide of potassium should be a rule in cases in which acquired syphilis is possible. Whenever the inflammation may be independent, in nature or degree, mercury is more likely to influence the process than any other agent.

When the causal disease is not syphilitic it is often scrofulous, and here also great good can be done by appropriate tonic treatment, to which abundant fresh air should be added.

[1] Horsley. "Trans. Internat. Med. Cong." (Berlin), 1890, and "Brit. Med. Jour.," 1893. (*See* also references on p. 79.)

Commencing neuritis may subside entirely and leave no trace, if the treatment induces the arrest of the cerebral disease. But unfortunately we are able to influence the ocular change much more slowly than we can influence syphilitic disease, and if neuritis be already intense, it is rarely that some impairment of sight can be prevented.

B.—MORBID STATES OF THE OPTIC DISC CHARACTERIZED USUALLY BY LESSENED VASCULARITY AND SIGNS OF WASTING. ATROPHY OF THE OPTIC NERVE.

Under many circumstances the fibres of the optic nerves undergo wasting or degeneration. This occurs when the eye has been greatly damaged by any cause, particularly when extensive retinal changes have supervened. As we have seen, it occurs as a consequence of the inflammation of the intra-ocular end of the nerve, or of its trunk; the wasting thus produced is termed "consecutive," "post-papillitic," or "post-neuritic" atrophy. In other cases the wasting is preceded by no visible inflammatory disturbance, and is termed "simple" atrophy. Nevertheless, in rare cases, an atrophy is preceded by the signs of simple congestion of the disc, and such cases may be termed "congestive atrophy." It is probable that the pathological condition in this form is really a chronic inflammation of the nerve itself, of which the intra-ocular signs of congestion, &c., are the indication. Lastly, atrophy may succeed choroiditis and retinal disease.

Atrophy, not consequent on any obvious ocular change, was found by Vulpian in about 4 per cent. (19 out of 500) of autopsies on old persons at the Salpétrière. In an equal number (21) there was atrophy consequent on an ocular disease.[1]

CHARACTERS.—The nutrition of the nerve fibres, and that of the capillary vessels which confer on the disc its normal

[1] Table given by Galezowski, "Sur les Atrophies de la Papille du Nerf Optique." "Journal d'Ophtalmologie," Jan., Feb., and March, 1872.

rosy tint, are so associated that atrophy of the fibres is accompanied in nearly all cases by a wasting of the capillaries, and the pallor thus produced constitutes the most salient sign of the atrophy of the nerve. The atrophied nerve commonly shrinks, and occupies less bulk than the normal nerve. This is not attended by any diminution in the size of the optic disc, since the latter is determined by the size of the sclerotic opening. The shrinking is indicated by a slight recession or "excavation" of the disc. In some cases there is a diminution in size of the retinal vessels, but this is an inconstant character. These signs will be considered in detail.

Pallor.—In judging of the colour of the optic disc it is important to examine it with a weak illumination, and by the direct method, in order to let as little light as possible be reflected. In a strong light any faintly-tinted object will appear white.[1]

It is as essential to be aware of the normal variations in colour of the optic disc, for the estimation of pathological pallor, as it is for the recognition of congestion. The variations on the negative side are not, perhaps, so considerable as are those on the positive side, but they are sufficient to render familiarity with the appearance of the normal disc necessary to prevent mistakes in estimating the slighter degrees of atrophy. The disc becomes paler in advanced life, and a slight grey tint becomes mingled with the red, but the latter is still perceptible. Thus, a tint which is normal in the old, would be suggestive of atrophy in the young.[2] Again, when the general fundus is unusually dark, the disc will seem to be abnormally pale, simply as an effect of contrast. In anæmia, also, the disc may become

[1] With very intense illumination, even a strongly-tinted object will appear white. This is because all objects reflect some of all rays, and absorb none entirely. If the waves impinging be sufficiently numerous —*i.e.*, the light very intense—so many waves of all lengths are reflected that the object appears white, the waves of the length chiefly reflected being no longer preponderant, although they become preponderant on weakening the light.

[2] In mere infants, however, the disc is said to be somewhat pale.

paler, but the change of tint from this cause is not considerable, and is insignificant in comparison with the normal variations in colour of the disc; it never constitutes an element of difficulty in the recognition of atrophy.

When a pathological pallor of the disc is pronounced, it extends over the whole area of the disc, but commencing pallor may be most marked in that part of the disc which is normally palest, *i.e.*, the temporal side, where the nerve fibres are least numerous. The change in this part, however, is only of significance in individuals in whom the "physiological cup" is small, and the temporal half of the disc normally possesses a distinctly vascular tint. In a great number of cases, in which the physiological excavation is large, and slopes gradually to the sclerotic ring on the temporal side, this portion of the disc may be normally as pale as in atrophy. The part on which attention should be chiefly fixed is, therefore, that which normally possesses considerable vascularity, the nasal portion, which is sometimes very narrow, but then deeper in tint. In this, therefore, a certain degree of pallor may mean more than in a broader nerve-fibre region. The colour may be observed to become gradually less, the red sometimes simply fading, and leaving a white colour in its place; in other cases a grey becomes mingled with the red, and gradually preponderates as the red tint fades, and ultimately a pure grey is left. If the examination is made with daylight, the tint is often a greenish-grey. These two varieties constitute in their extreme forms the white and grey forms of atrophy respectively. Intermediate forms are often seen, and to the direct method of examination some grey tint may always be distinguished, even in the discs which appear quite white to the indirect method. This grey tint tends to increase as time goes on.

The atrophy leaves the edge of the disc very distinct and sharp. The sclerotic ring is much more clear than it is normally, but it may not, at first, be recognized by the indirect method, as it is not differentiated from the white surface, as it is from the rosy tint of the normal disc. The sharpness

of the edge is due, not only to its clearness, but also to the fact that the choroid usually preserves its normal characters to the margin.[1] Pigmentary deposits on the edge of the disc are, like the edge itself, abnormally distinct.

Excavation.—In simple atrophy, the surface of the disc is depressed in proportion to the wasting of the nerve elements. The depression varies, however, in the different forms of atrophy, because the wasting of the nerve fibres is the only change in some forms, while in others it is accompanied with an overgrowth of connective tissue, which may to some extent compensate for the atrophy of the nerve elements, and may even prevent diminution in bulk of the nerve. Thus, in some cases, the depression of the disc is considerable, and in others it is slight or absent. Its special character is that it affects the whole disc, and commences at the sclerotic ring. It may often be recognized by the change of level of the retinal vessels at the spot, distinct on movement of the observer's head, or of the lens in the indirect method. Normally, it will be remembered, the depression of the centre of the disc never begins at the sclerotic ring, except on the temporal side in some cases of large normal cups. Above, below, and at the nasal side—*i.e.*, in the position of the large vessels—the normal excavation never commences at the ring, within which there is always a zone of nerve tissue, commonly the most prominent portion of the disc. Hence the change in the course of the vessels at the ring becomes an important sign of excavation. The size and form of the resulting excavation depend on two things—the amount of shrinking of the nerve, and the previous size and form of the normal cup. The wasting of the edge of the cup tends to lessen its steepness, and so to give a steep central cup a more gradual slope.

The mottling of the lamina cribrosa may become very distinct at the bottom of the excavation. Where the normal cup was large, the excavation may reveal the lamina cribrosa in almost the whole extent of the disc, the grey mottling

[1] The chief exceptions are where atrophy occurs with old myopic changes, or secondary to a choroido-retinal affection.

corresponding to the bundles of degenerated nerve fibres, the white interspaces to the meshes of the lamina.

It is believed that some share in the excavation is due to the atrophy of the small vessels, which conferred on the normal disc a certain amount of turgescence. De Wecker suggests that as the nerve has its consistence lessened, the normal intra-ocular pressure may assist in producing the excavation. This is possible but cannot be proved.

It has been said that the more connective tissue is developed in the atrophied nerve, the slighter is the shrinking of the trunk. Among the remains of the diverging nerve fibres there is little connective tissue developed, and the wasting of the fibres here is compensated for to a much less extent than in the trunk of the nerve.

The Retinal Vessels.—In some cases of simple atrophy of the optic nerve the retinal vessels become reduced in size, in others they do not. In the grey atrophy, as a rule, the vessels undergo little or no change, but they are occasionally narrowed. In simple white atrophy they present no alteration in some cases; in others, the arteries gradually become smaller, the veins undergoing little diminution. After a time the veins also may shrink. The vessels are reduced in size when there is retro-ocular neuritis near the eye, but this cannot be regarded as the cause of their shrinkage in all cases. In a case of severe papillitis, the product of the inflammation compresses the central artery, and the veins shrink secondarily from a lessened retinal blood supply. Their atrophy seems to accompany that of the retina, which is usually associated with atrophy of the nerve. Why they should shrink in some cases of simple atrophy, and not in others, is at present unexplained.

Initial Signs of Congestion.—In describing simple congestion of the disc, it was pointed out that it may terminate in atrophy. The disc has, at first, a dull-red tint, with a soft-looking surface, the redness being uniformly distributed over it. The edges of the disc are less sharply defined than in health. The congestion may persist for a long time, but, as time goes on, the disc slowly becomes paler, and ulti-

mately a condition of greyish-white atrophy is reached. Occasionally the disc presents at first, for a short time, a slight degree of œdema as well as congestion, shown by slight swelling. The pathological process, in many cases of atrophy, seems to be of the nature of a chronic inflammation. It is readily intelligible that in some cases the signs of slight inflammation should be visible during the early stage. The cases in which it is met with are especially those which result from toxic causes and disseminated sclerosis. This state of chronic inflammation behind the eye may be diffuse and affect the whole nerve, or may be partial and involve only a segment (segmental neuritis). The vessels often present much earlier and more considerable narrowing than in simple atrophy, and in the disc around them much white tissue becomes developed. It is to be noted, however, that in some conditions of undoubted retro-ocular neuritis, there may be no signs of inflammation or congestion of the disc, but only that of simple atrophy, and hence it is convenient to consider this form in the present section. The mischief is commonly at some distance behind the eye.

Atrophy after Intra-Ocular Neuritis; "Consecutive Atrophy," or "Papillitic Atrophy."—The newly-formed inflammatory tissue-elements of papillitis are in part removed, and in part transformed into connective tissue, which gradually shrinks.

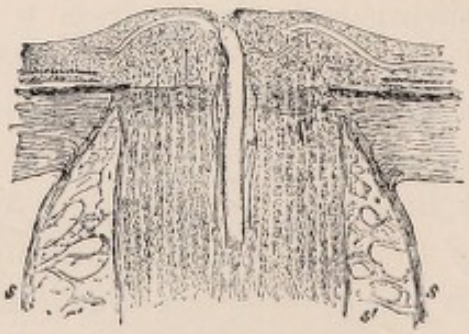


FIG. 49.—VERTICAL SECTION THROUGH THE OPTIC DISC IN A CASE OF POST-PAPILLITIC ATROPHY, DUE TO TUBERCLE OF THE CEREBELLUM.

The retinal layers are displaced, and the bundles of fibres in the optic nerve are separated. A vessel is seen divided longitudinally. Neither within nor behind the sclerotic ring is it compressed. Within the papilla, however, its branches are very narrow. (× 15.)

The pale swelling left by the inflammation (Pl. II. 5, IV. 3), large in proportion to the intensity of the process, slowly subsides, until it shrinks to the limits of the disc, and slowly reaches the level of the retina (Fig. 49). The soft edges which at first limit the pale swelling gradually become more sharply defined. The recession of the swelling from the edge of the choroid often shows that the latter, and the retinal pigment layer, have been damaged, and have undergone irregular atrophy adjacent to the edge of the disc (Pl. II. 4). The substance of the disc has a "filled-in" look, from the new tissue within it (Pl. VI. 2), and is commonly white, or rarely greyish in tint. The vessels, whether previously narrowed or not, usually become narrowed by the contraction of this new tissue, and may be partly concealed by it at their origin, or in their course over the disc. The tissue along their walls is often distinctly whiter than the rest of the disc, and when the latter is grey the contrast may be very marked. Often white lines are to be traced along the narrowed vessels for some distance from the disc, probably due to thickening of the outer coat. Ultimately, the contraction of the tissue may go as far as to lead to some excavation of the disc (Pl. II. 6), and there may be only the adjacent choroidal disturbance, and the narrowing of the vessels, to indicate the origin of the atrophy. The lamina cribrosa is usually concealed. The disc remains for a long time white to the indirect examination; ultimately, it becomes distinctly greyish, especially on direct examination,

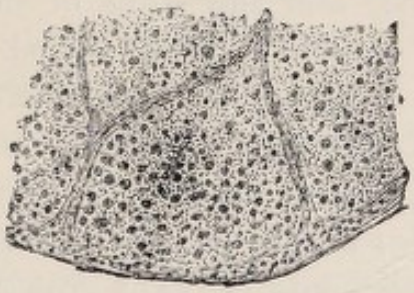


FIG. 50.—SECTION THROUGH THE OPTIC NERVE IN THE SAME CASE AS THE PRECEDING FIGURE.

The fasciculi of degenerated nerve fibres are infiltrated with nuclei, and cells of irregular shape. The septa between the bundles are a little thickened. (× 100.)

and with feeble illumination. If the inflammation has been moderate and has not damaged the choroid, the edge of the disc may be sharply defined, and its appearance may resemble that of some cases of simple atrophy, and be quite indistinguishable from that left by a retro-ocular neuritis.

Choroiditic Atrophy.—The atrophy of the disc, which is often seen after choroido-retinitis, is sometimes white or grey and resembles primary atrophy; but often it is less pale, having a peculiar opaque, reddish or yellowish-red tint, uniform in distribution, sometimes with slight blurring of the edges. Usually there is a marked diminution of the retinal vessels.

The recognition of this variety of choroiditic atrophy is of considerable importance, because, unless the result of retinitis pigmentosa, it is almost always the consequence of syphilitic disease, acquired or, more frequently, inherited. It constitutes a sign of inherited syphilis of great importance. In most cases, characteristic round spots of choroidal atrophy and disturbance of the retinal pigment are found.

Causes.—Simple atrophy of the optic nerve is either a primary change, or is secondary to some lesion of the nerve. These two varieties may be distinguished as "primary" and "secondary" atrophy, and are especially characterized by the circumstance that, in primary atrophy, the loss of sight coincides in origin and progress with the visible alteration in the disc, but in secondary atrophy the loss of sight occurs first, and the signs of nerve degeneration are not observed until a later period. It is doubtful whether the two forms can be distinguished by the ultimate aspect of the disc.

It has been proposed to divide the primary atrophies into two classes, according as the process commences (1) by degeneration of the nerve elements, or (2) by growth of the interstitial tissue, with secondary damage to the nerve fibres. The distinction has been especially insisted on by Charcot and by Abadie, on grounds of etiology, pathology, and symptoms. Our knowledge at present is scarcely sufficiently definite to

make a sharp distinction generally useful, if indeed, it is founded on a correct basis.[1] Considerable overgrowth of the interstitial tissue occurs in cases in which we must assume that the wasting of the nerve fibres is the primary element. Though secondary in nature, it may attain an exuberant degree, the result of which is to suggest its primary nature. It may entail vascular disturbance which further obscures its really secondary nature. This is conspicuous in the history of opinion regarding the changes in the spinal cord in tabes, which all now agree in regarding as essentially neural.

The recognition of the fact that the nerve elements waste first, is unquestionably of absolute pathological importance, to which all other distinctions must yield. No feature of modern pathology of the nervous system has been more conspicuous than the progressive recognition of the fact that degenerations of systems of fibres are primarily decay of the nerve elements, to which the interstitial change is secondary.

Primary Atrophy sometimes comes on without evident cause. It may be hereditary, and one very remarkable form (carefully studied by Leber) affects the males of a family soon after puberty.[2] The male sex is, apart from this variety, the more prone to optic nerve atrophy. Seventy-five per cent. of all cases occur in men, and most are in adults. A considerable number of the cases of primary atrophy are associated with disease of the spinal cord, chiefly with tabes. This form is regarded as the most typical example of the primarily neural form. It is usually a grey atrophy without diminution in the size of the vessels. The tabetic symptoms may be slight, consisting only of pains and loss of the knee-jerk, usually with loss of the light reflex of the iris, apart from loss of sight to account for it. There is usually also evidence of the common antecedent, syphilis,

[1] *See* Duwez, "Dict. Encyc. des Sciences Med.," tom. xvi. pt. 1, p. 319.

[2] *See* a paper on this subject by S. H. Habershon: "Trans. Ophth. Soc.," vol. viii. 1888, p. 190; Snell, "Trans. Ophth. Soc.," vol. xxvii; also Gowers' "Abiotrophy," "Lancet," April 12, 1902, and "Lectures," 2nd Ser. 1904, Churchill.

long before. Cases of atrophy, with no tabetic or other symptoms, but also after syphilis, must probably be placed in the same class. Charcot believed that spinal symptoms always come on later, but this opinion is certainly erroneous.

A similar atrophy occasionally occurs in general paralysis of the insane, and also in disseminated (insular) sclerosis, and rarely in lateral sclerosis of the cord. These forms have been said to be preceded by signs of congestion of the disc, but these are seldom to be perceived. The relation of primary atrophy of the optic nerves to some of these diseases has assumed a new aspect, through the evidence that tabes, and also general paralysis, are apparently the result of the action on the nerve structure of a toxine left by syphilis. Its influence is exerted chiefly on the nutritional centres of the sensory neuron, *i.e.*, on the structures which depend for vitality on the cells of the posterior ganglia. The sensory nerves degenerate, first and chiefly, at the periphery, and to the change in them the degeneration of the optic nerve bears an analogy, how close we do not yet know. The effect of the morbid influence seems to be to lessen the vital endurance of the structures, and to entail future decay. The degree and distribution of this depend on conditions of which we have, as yet, no knowledge. We can, however, conceive that the chief element is the character of the toxine, since it is to this only that we can refer the variations in the effects of other analogous agents; for instance, that which causes diphtheritic paralysis. We can thus understand that the influence may sometimes involve, and sometimes spare, the optic nerve. (*See* under Tabes.)

It must be remembered that the optic nerve is, developmentally, a direct prolongation of the central nervous system, and that, anatomically, it resembles the white matter of the brain and spinal cord. The importance of this relationship, in connection with the question of the independent origin of changes in the optic nerves and in the spinal cord, is of great importance.[1]

The atrophies of the optic nerve which are not associated

[1] *See* Gunn, "Brit. Med. Jour.," 1885, ii. p. 688.

with tabes and other spinal disease, have been ascribed to various causes, the influence of which is most doubtful. Such are: cold, various excesses, menstrual disturbance, gastro-intestinal affections, migraine. With better reason they have been ascribed in rare instances to diabetes, intermittent fever, and some acute specific diseases, and the facts regarding these will be considered in Part II. In a considerable proportion of the cases unconnected with other nerve disease, no adequate cause can be ascertained. Tobacco and bisulphide of carbon certainly, probably alcohol and lead, cause amblyopia, and may cause partial atrophy, but in the case of lead, at least, this is usually preceded by signs of congestion or even inflammation. There is reason to believe that in the case of tobacco the initial lesion is an impaired nutrition of the central retinal neurons about the macula.

The important form of atrophy occurring in several members of the same family is of great importance, yet isolated cases are met with that have the same characters as those which occur in families. They seem due to defect of vital endurance. The symptoms begin soon after puberty, often after some exciting influence which may appear adequate, such as tobacco, but they progress after it has ceased to act.[1] (*See* Part III.)

Primary atrophy usually affects both eyes, commonly one earlier than the other, but in rare cases one only.

Secondary Atrophy results from lesion of the optic centres or fibres. A cortical lesion in the brain may probably entail loss of sight of the opposite eye, and certainly one in the occipital lobe may produce hemianopia, but such damage causes little change in the disc. Possibly, slight atrophy takes place in time.[2] Even a lesion of one optic tract, causing bilateral symmetrical hemianopia, seldom produces a distinct ophthalmoscopic change. Some observers have described an ultimate slight pallor of the corresponding halves of the discs, but this is not often distinct. But in cases of long duration, the whole of the disc of the eye in

[1] Habershon; Gowers, *loc. cit.* (p. 105).

[2] *See* Bernhardt, "Berl. kl. Wochenschrift," 1872, No. 30.

which the area lost was on the temporal side (and therefore greatest), seems to become perceptibly paler than the other, the tint of the two being at first equal. This has been observed by others as well as by myself, although the difference is slight, and not easy to explain.

Pressure on the chiasma, or on the optic nerves is a common cause of optic nerve atrophy without preceding neuritis. If the chiasma is affected, temporal hemianopia results from damage to the crossing fibres in the middle, which come from the inner half of each retina, and receive impressions from the outer half of each field. The damage may be from the pressure of tumours growing from any of the adjacent structures (*e.g.*, the pituitary body), exostoses, rarely aneurism. It not uncommonly results from internal hydrocephalus—the distended third ventricle compresses the chiasma directly, pressing first on the upper and posterior aspect, where, as Michel has shown, a depression may be produced.[1] In some cases there is reason to believe that such medial damage to the chiasma results from primary interstitial inflammation of the nerves, when there is usually an irregular impairment of one field as well as the temporal loss in both, from the more extensive affection of one optic nerve.

Meningitis is another cause which, while commonly producing optic papillitis, if it extends to the nerve, may also, in rare cases, cause blindness and atrophy without intra-ocular inflammation, by pressure without inflammatory invasion. It may also cause blindness and atrophy out of proportion to neuritic mischief, and often after the inflammation of the papilla has subsided. The local neuritis in these cases may be much more intense than is suggested by the degree of intra-ocular inflammation. It is said that obstruction, by embolism or thrombosis, of one middle meningeal artery, which supplies the dura mater around the optic foramen, may be followed by atrophy of that optic nerve. The aspect of the discs in these cases is that of

[1] Compression and flattening of the chiasma from ventricular distension were noted by Cheselden in the eighteenth century. ("Phil. Trans.," No. 337, p. 281.)

purely secondary atrophy, with considerable reduction in the vessels only when the inflammation extends to the anterior part of the optic nerve. Whether the disc is white or grey depends on its original capillary vascularity.

Damage to the optic nerve causing atrophy may also occur in the optic foramen or in the course of the nerve through the orbit. Narrowing of the foramen by bony thickening, and rheumatic or syphilitic or traumatic mischief, producing pressure at the back of the orbit, close to the foramen, are occasional causes. Blows on the head commonly produce atrophy by direct injury to the nerve, but it is possible that they may cause, by the effect of the shock, a gradual degeneration. The ultimate atrophy which results from these causes has no special feature.

Disease in the orbit may cause a process of "retro-ocular neuritis." In such cases the visual failure is usually rapid, and exists for some time before any change is visible at the disc. Not infrequently the earliest ophthalmoscopic evidence is congestion of the disc, sometimes with marked swelling and narrowing of the retinal vessels, but signs of atrophy soon supervene, though vision may improve. The nerve may be thus damaged by orbital cellulitis, primary or due to the extension of inflammation to the orbit in erysipelas of the face.

Papillitic or consecutive atrophy results from intra-ocular neuritis, as already described.

Retinal and Choroiditic Atrophy.—Damage to the retina entails an atrophy of the optic nerve, which progresses, sometimes slowly, sometimes quickly, but is usually incomplete. It is believed that in some forms of toxic amblyopia, as from tobacco, the initial lesion is an impaired nutrition of the macular ganglion cells, with subsequent changes in their fibres. Now and then atrophy of the optic nerve follows some cause that seems to act by giving a shock to the retina, that leaves no trace behind—*e.g.*, the complete amaurosis, which may accompany the onset of embolism of one branch of the retinal artery, and is usually temporary. It is sometimes permanent, even though all the other

branches of the retinal artery are pervious. Atrophy sometimes follows a blow on the eye, as in a case related by Laqueur, in which a blow caused complete amaurosis without visible changes in the fundus, and simple atrophy followed. Such cases are of medical interest on account of the light they throw on the action of some general causes, such as anæmia (*q.v.*). It is rarely that any considerable degree of atrophy follows simple retinitis. Commonly, the traces of the retinal process that results in atrophy are obvious. After choroido-retinitis the aspect of the disc has been already described.

Anatomical Changes.—Atrophy of the optic nerve is never confined to the papilla; the changes are marked throughout the whole length of the nerve, and in primary atrophy are usually equally distributed. The size of the nerve varies very much; in some primary forms it is smaller than normal, somewhat translucent, but scarcely grey, and under the microscope may present merely a wasting of all the structures of the nerve, fibres and connective elements, with, especially in recent cases, products of the degeneration of the nerve fibres, granules and globules of fat, compound granule cells, and "corpora amylacea." The position of the fibres may at first be marked by rows of fatty particles. In other cases the nerve may be little diminished in size, but may present under the microscope a great increase in the interstitial connective tissue, fibres and cells, with disappearance of the nerve tubules. Commonly the change is greater in the circumferential portions of the nerve than in the central.[1] Occasionally the reverse is the case. In atrophy from pressure on the nerve, its size is usually greatly reduced, and the increase of connective tissue is very considerable.

In primary grey atrophy the nerve trunk is usually little reduced in size, and is grey and gelatinous in appearance. Microscopically, it presents an increase in the connective tissue trabeculæ, and an atrophy of the nerve fibres, first

[1] Leber: "Arch. f. Ophth.," xiv. p. 182.

of the medullary sheath, and afterwards of the axis cylinder. Products of myelin degeneration may be found in the earlier stages. Sometimes the change is peculiar; there develops round the vessels a peculiar gelatinous-looking tissue containing a few nuclei and indistinct concentric fibrillation. The normal arrangement of the trabeculæ disappears, and a section of the nerve (Fig. 51) shows islets and tracts of this tissue, in the centre of each of which a vessel can be traced. They may occupy at least one-third of the area of the section. Between them lie the fasciculi of degenerated nerve fibres with little increase in their interstitial tissue. In the case figured, the atrophy was confined to one optic nerve, and its cause was obscure. The same histological condition may be present in the grey atrophy of locomotor ataxy.[1] In other cases of grey degeneration (according to Leber's observations) the change may be more uniformly distributed through the fasciculi. The degeneration is sometimes found in certain areas much more intensely than elsewhere. In a case of locomotor ataxy,

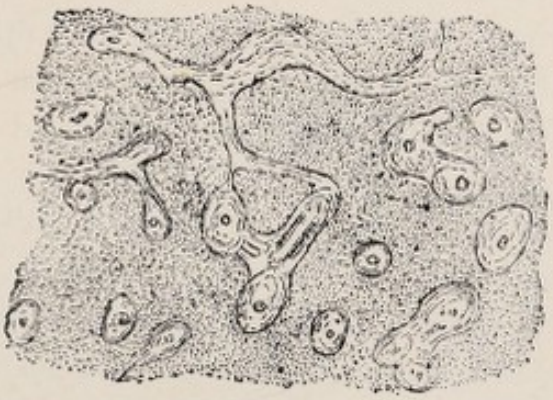


FIG. 51.—GREY ATROPHY OF OPTIC NERVE: TRANSVERSE SECTION, MIDWAY BETWEEN THE EYEBALL AND THE OPTIC FORAMEN.

The trunk of the nerve was grey, and gelatinous in aspect, and was not diminished in size. The other optic nerve was healthy. The nerve fibres are completely degenerated, a granular tissue representing them. The normal trabeculæ have disappeared, and through the section of the nerve are scattered tracts and islets of a slightly fibrillated, in places almost homogeneous, colloid-looking tissue. These tracts enclose vessels which can be distinguished, small in size, and with thickened walls, in the centre of each. (× 150.)

[1] Cf. Perrin and Poncet's "Atlas."—Atrophy of the Optic Nerve.

in which sight was not known to be impaired, I found only a great increase of tissue, consisting of nuclei and fibres, at the nodal points of the trabeculæ, and a little gelatinous-looking tissue immediately adjacent to the wall of the vessels. It would probably be justifiable to assume that this represents the commencement of the process of change.

In cases of primary atrophy of the nerve the retina is degenerated only in its inner layers—nerve-fibre and ganglion-cell layer, as Virchow first showed.[1] The other retinal elements may persist in a perfectly normal condition, even for many years. Perrin and Poncet could find no change, except in the two inner layers, in a case of ataxy in which sight had been lost for thirty years.

The degeneration from damage to the trunk of the nerve ascends to the chiasma, and descends to the eye. It is long in passing the chiasma, and, even with complete atrophy of one nerve, the optic tracts are only slightly reduced in size, that on the side opposite the affected nerve being rather smaller than the other, without naked-eye evidence of degeneration; and the microscopic changes are nearly equally distributed through the two.[2] When both optic nerves are degenerated, the optic tracts may present an equal change, traceable (as Türck pointed out) as far as, and involving, the external corpora geniculata.

Consecutive or Post-Papillitic Atrophy.—The microscope shows the substance of the disc to be occupied by nucleated connective-tissue, among which, commonly, few or no traces of nerve-fibres are to be discerned. Often, however, the nuclei, by their grouping, indicate the position of the intervals between the fasciculi of former nerve fibres. The retinal layers are displaced outwards (Fig. 20), an important sign of the preceding swelling, and both they and the edge of the choroid may present some disturbance. The atrophy of the rest of the retina is confined to the inner layer, especially affecting the layer of nerve fibres.

Symptoms.—The great symptom of atrophy is impairment of

[1] Virchow's "Archiv," vol. x. 1856.

[2] Gowers, "Centralblatt f. die med. Wissensch.," 1878, No. 31.

sight proportioned to the affection of the nerve fibres, impairment of which the patient gradually becomes aware. Examination shows a change in sight in three directions—(1) diminished acuity of vision; (2) alteration in the field of vision; (3) altered perception of colours.

1. *Diminution in the acuity of vision* is practically invariable when the atrophy is pronounced; it is almost always more considerable in one eye than in the other. In estimating it, care must be taken to ascertain and correct any errors of refraction, and defects of accommodation. It may vary from a slight degree to complete loss. It is commonly, but not always, proportioned to the degree of change in the optic nerve, visible with the ophthalmoscope.

2. *Alteration in the field of vision* may be of several kinds. It is almost as constant as the diminution in acuity. The form is commonly a restriction at the margin of the field, progressing concentrically until only a small central area is left, such as is shown in Fig 52. Such a limitation may progress much more on one side of the field than on the other, or it may progress much more in one part, so as to cause a sector-like defect. Occasionally the diminution is

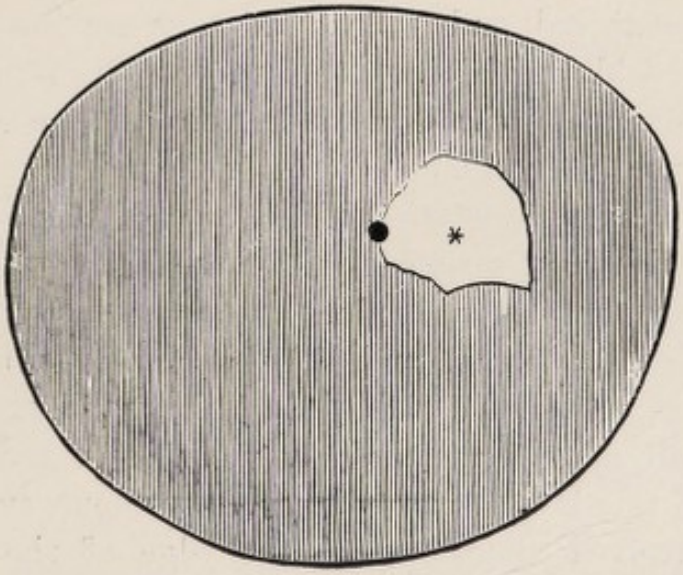


FIG. 52.—CONCENTRIC LIMITATION OF LEFT FIELD OF VISION IN A CASE OF ATROPHY OF THE OPTIC NERVE.

The outer boundary of the figure is the limit of the normal field. The inner white area is the area of the restricted field.

limited to one-half of the field, vertical or lateral. In retinitis pigmentosa, particularly the primary form, the field is often first lost in a zone midway between centre and periphery. Lastly, in some cases, the first loss is a central one, in the middle of the field, a "central scotoma," as it has been termed. Not infrequently in such cases, the chief loss is not exactly at the centre, but to one side of it (usually the outer), and is then called a "paracentral scotoma."

3. *Colour-Blindness.*—In many cases the perception of colours is perverted. There are two methods of testing colour-vision. If the patient possesses sufficient intelligence, he may be asked to identify certain colours. If the patient is unintelligent, the "confusion method" must be adopted, by ascertaining which colours seem alike. The former method sometimes gives more valuable information.

Modern physiological speculation suggests that there are four fundamental colours, related in complementary pairs, red and green, yellow and blue. The area of the field of vision, in which these colours are seen, varies for each. If coloured objects are moved from the centre of the field to the periphery, the first simple colour to be unperceived is green, the next red, and yellow and blue are lost near the edge of the field for white. Commonly yellow is lost before blue, but sometimes the latter is lost first. The amount of light influences very much the area of the fields, and Fig. 53 is an approximate indication of the size of the fields for a centimetre object on a day on which the light was dull. It will be seen that their outlines are practically concentric with the field for white, and this has been found true in normal eyes, under all conditions. But the area of the fields is still more influenced by the size of the coloured object.[1] If this is sufficiently large, any colour, even green, can be perceived almost up to the edge of the field for white.

Commonly, in atrophy of the nerve, the first defect is for

[1] *See* Gowers, Lecture on Subjective Visual Sensations, "Trans. Ophth. Soc.," 1895; and Lectures, Second Series. Churchill, 1904.

green and red, and blue and yellow are lost subsequently.[1] The order of affection is commonly that in which the fields are arranged on the retina. The simple colour first lost, in passing from the centre to the periphery of the retina, is that first lost in atrophy, green; and the last to be lost is blue or yellow. Thus a girl, suffering from disseminated sclerosis

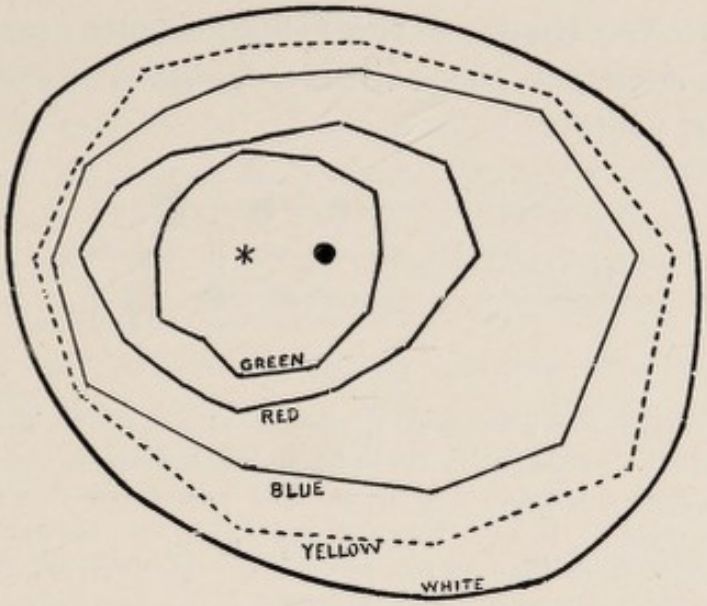


FIG. 53.—A REPETITION OF FIG. 41. DIAGRAM SHOWING THE FIELDS OF COLOUR-VISION IN A NORMAL EMMETROPIC EYE ON A DULL DAY.

The fields are each rather smaller than on a bright day. The asterisk indicates the fixing point, the black dot the position of the blind spot. (Usually the blue field is larger than the yellow.)

and commencing grey atrophy, recognized, with the affected eye, every colour except green, which she called red or brown. Yet anomalies are met with; in another case, there was only loss of perception of green. Occasionally red appears to be lost first. A patient with tabetic atrophy stated that the first loss of the sense of colour of which he was conscious, was that he could see no colour in a scarlet geranium. Red gravel looked grey to him. Soon afterwards the grass also looked grey, and he could not, at a little distance, distinguish it from the gravel. When examined,

[1] For recent views on the earlier failure of recognition of red and green, *see* Gunn, Bowman Lecture on "Visual Sensation," Trans. Ophth. Soc., vol. xx.

violet alone was seen as a colour; he said it looked blue. A medium blue was seen as white. Cases have also been met with in the stage in which perception of red was lost and that of green was preserved. The loss of perception of colour is often rather a colour amblyopia than colour blindness. Large pieces of colour may be seen when small spots are not, just as in health the fields for colour depend on the size of the object. The shape of the fields for colour may present alterations similar to those already described as occurring in the field for white.

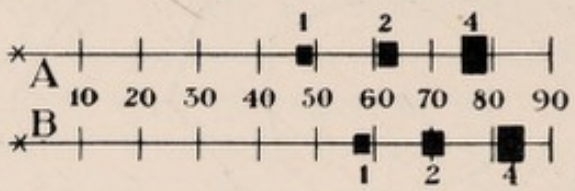


FIG. 54.

A and B represent, each, the outer horizontal radius of the field of vision of the right eye, and the limit at which red is visible in an area of 1, 2, and 4 centimetres in vertical measurement. A with the left eye closed; B with the left eye open, but its field separated by a screen from that of the right eye. (Gowers, *op. cit.*)

Abadie[1] has attributed especial, and certainly undue, importance to the loss of colour-vision as a supposed distinction of the parenchymatous from the interstitial forms. It is probably of little significance in this respect.

The relation of changes in the field of vision to the position of disease in the nerve behind the eye is not simple, since the relation of the fibres seems to change. The fibres from the central region lie in the axis of the nerve at the back of the orbit, but in the outer part[2] of the anterior half of the nerve. The discernment of this is due, primarily, to Förster, who reversed the earlier view of Leber. A peripheral defect in the field of vision is ascribed to an affection of the circumferential portion of the optic nerve.[3] Immediately

[1] "Ann. d'Oculistique," 1878, and Lebris, "Thèse sur les Differentes Formes de l'Atrophie de la Nerf Optique." Paris, 1878.

[2] *See* Samelsohn, "Centralbl. f. med. Wissensch.," 1880, p. 418; Nettleship and Edmunds, "Trans. Ophthalmological Society," vol. i. 1881, p. 124.

[3] *See* Wilbrand: "Klin. Monatsbl. f. Augenheilk." Dec. 1878, and "Centralblatt f. med. Wissensch.," 1879, p. 923.

behind the disc, however, it is probable that the peripheral fibres pass to the adjoining retina, and the more central ones to the retinal periphery, while those in the outer third belong to the macula.

Concentric diminution of the field is common in all forms of atrophy. In the simple form (as in tabes) it most frequently begins on the outer side, but may commence on the inner side or above or below. The acuity of vision may fail at the same time, or may remain normal until the field is reduced to a very small area. The patient then seems as if looking down a narrow tube—a condition sometimes called "telescopic vision"; "tube vision" would be more precise. When acuity is preserved, if the limitation is regular and sharp, central colour-vision may be normal, but the fields are reduced in area, preserving their normal relation to the field for white. When the limitation, although sharply defined, is irregular, colour-vision is usually much impaired.[1]

A loss of one-half of the field of vision (apart from cerebral hemianopia) is met with chiefly in secondary atrophy, especially when the cause is pressure on the chiasma, as by a tumour or interstitial inflammation there; the result is loss, partial or complete, of the temporal halves of the fields. Such a loss may occur in retro-ocular neuritis extending back to the chiasma, and is then associated with some irregular diminution in the remaining half-fields. We find, very rarely, indications of temporal hemianopia in tabes, in which we must regard the degeneration of the nerve fibres as the primary and essential element. Figs. 55 and 56 show the fields of a patient with tabes, who stated that he had rapidly lost vision in each eye. When he came under observation there was entire loss of the right field and loss of the temporal half of the left field, the loss including the fixing point. A similar affection of sight in tabetic atrophy has been observed by others.[2] The probable explanation is that the secondary interstitial overgrowth, which is so

[1] Nettleship, "British Med. Journal," 1880, ii. 779.

[2] *E.g.*, Treitel, "Arch. f. Ophth.," vol. xxv. 1879, p. 61.

exuberant in tabes, assumes an inflammatory character, especially at the middle of the chiasma.

Sector-like defects in the field are met with in secondary atrophy, especially in cases of injury to the trunk of the

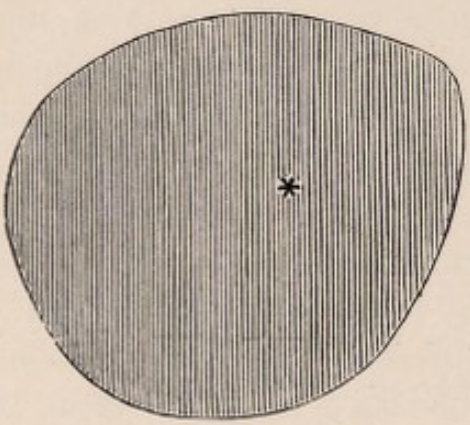


FIG. 55.

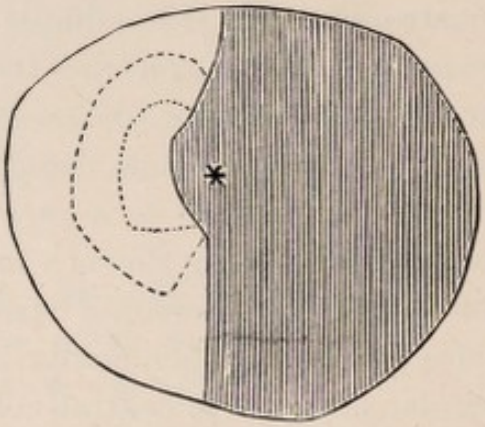


FIG. 56.

FIELDS OF VISION IN A CASE OF TABETIC ATROPHY.

The shading indicates loss. The outer dotted line indicates the field for blue, the inner that for yellow.

nerve at the posterior part of the orbit. They also occasionally occur in simple and in tabetic atrophy. A well-marked instance of this condition in spinal atrophy is shown in Figs. 57 and 58. The patient was in the earliest stage of

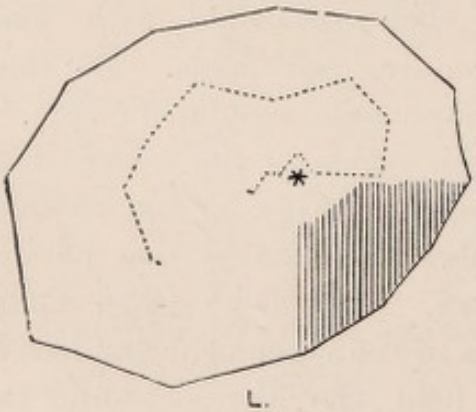


FIG. 57.

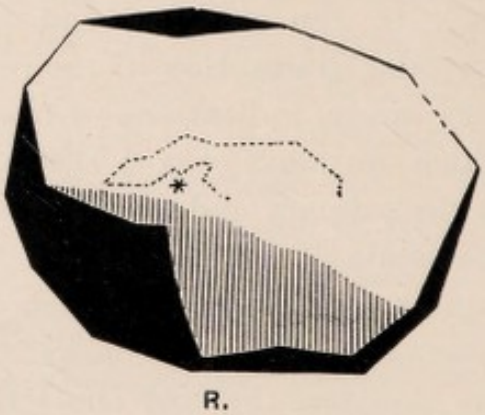


FIG. 58.

SECTOR-LIKE DEFECT IN FIELDS OF VISION IN A CASE OF SPINAL ATROPHY.

The shading represents amblyopia, the black loss. The dotted line shows the boundary of the field for red. Where it is absent the field ceased so gradually that its limit could not be ascertained.

locomotor ataxy. The optic discs were grey and the vessels small; vision was R. $\frac{1}{12}$ L. $\frac{1}{18}$. They are not easily explained, except on the hypothesis that the secondary interstitial overgrowth assumes an excessive, independent energy, in the outer part of the chiasma on each side.

Such sector-like defects in primary atrophy may be, as in this case, symmetrical, but they are sometimes unilateral, as in a tabetic atrophy where, in one eye, there was a defect of the upper and inner quadrant;[1] in two cases described by Treitel there was a defect in the inner and lower part in the right eye, similar to that figured.

Central scotomata are usually transversely oval, extending from the blind spot to the fixing point; the latter may sometimes just escape. The periphery of the field is usually normal, but may be restricted. There is always a loss of colour-vision, which is greater and occurs earlier than that for white. Red and green are first, and may be alone lost. Central scotomata for red are shown in Figs. 59, 60, and 61. They are met with in cases of axial neuritis and degeneration, and especially in cases of amblyopia from tobacco. That the latter depends on the same pathological condition (axial neuritis) is not improbable, both from the character of the affection of vision and from the fact that slight neuritis is often observed at the papilla.[2] A central scotoma is occasionally met with in consecutive (papillitic) atrophy. It may occur also in that which succeeds loss of blood, and is probably produced by neuritis. Central loss is occasionally met with in simple atrophy, but in the tabetic form it is very rare—has been said, indeed, never to occur. In a case under my care, however, there was a central

[1] Uhthoff, "Arch. f. Ophth.," vol. xxvi. 1880, pt. 1, p. 277.

[2] For the recent view of the nature of tobacco amblyopia, *see* special section. According to this, it is due to an influence on the ganglion-cells of the retina, on which the nutrition of the fibres is assumed to depend. Even this theory is not irreconcilable with the fact that slight papillitis may sometimes be seen, if the secondary interstitial process takes on exuberant activity.

scotoma (Figs. 62 and 63), associated with the symptoms of lateral and posterior sclerosis of the cord, and some cerebral degeneration; slight papillitis made it probable

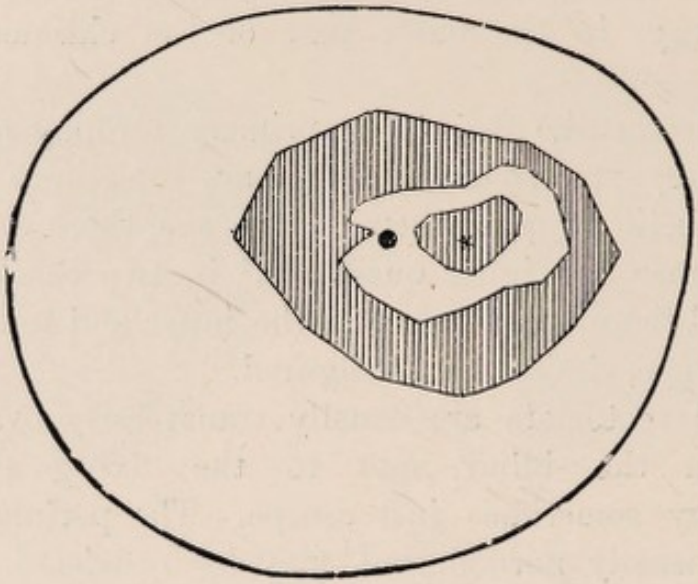


FIG. 59.—DIAGRAM OF THE LEFT FIELD OF VISION FOR RED IN A CASE OF TOBACCO AMBLYOPIA.

The outer line is the boundary of the normal field for white. The boundary of the outer shaded area is the minimum normal field for red. Red could, however, be seen only in the inner white area, and it could not be seen in the central shaded area around the fixing point (*). The black dot indicates the position of the blind spot. (For the chart from which this diagram was prepared I am indebted to Mr. Nettleship.)

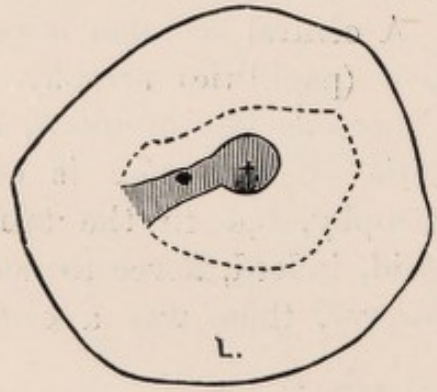


FIG. 60.

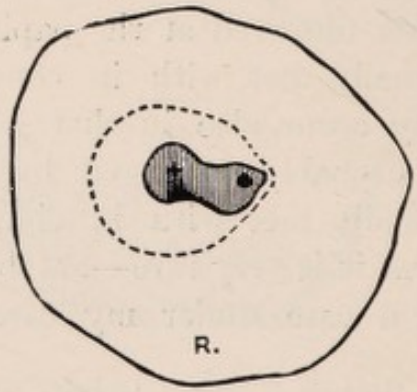


FIG. 61.

CENTRAL SCOTOMATA FOR RED, EMBRACING FIXATION POINT AND BLIND SPOT.

No loss for white, but considerable amblyopia (16 Jäger). The patient had smoked half-an-ounce of shag daily. The dotted line represents the periphera' boundary of the field for red. (Nettleship.)

that axial neuritis existed; the influence of tobacco seemed inadequate, from the facts.

Peripheral areas of persisting vision, with general loss,

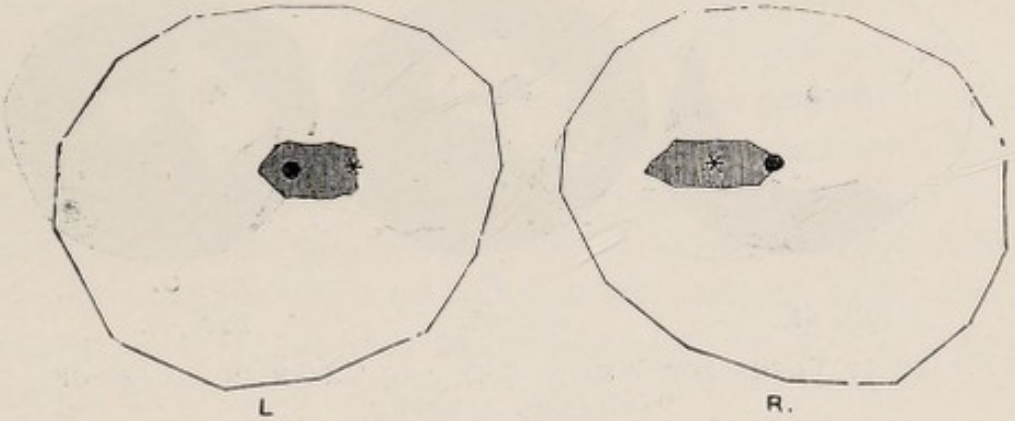


FIG. 62. FIG. 63.

CENTRAL SCOTOMATA IN A CASE OF DEGENERATION OF THE SPINAL CORD.

There was a rather larger central loss for red and green, but the peripheral defect for these colours was also considerable.

are met with only in cases of orbital inflammation with partial damage to the nerve, or in consecutive (papillitic) atrophy. The changes in the latter are often very irregular. There may be general concentric limitation of the field, or, less commonly, a central loss, rarely sharply defined. Failure of colour-vision is frequent, but is often less regular in order, than in primary atrophy. The colour fields may present very irregular defects, as in Figs. 64, 65, and 66, which represent the fields for white, red, and green in a case of post-neuritic atrophy. Those for yellow and blue were normal, except for a limitation below, and to the inner side, corresponding to the field for white. They were taken nine months after the subsidence of the neuritis, when acuity had improved to $\frac{1}{12}$. Uhthoff once met with a central scotoma for blue only.

In atrophy, as a rule, there is some correspondence between the pallor of the disc and the failure of sight. When it is considered, however, that the tint of the disc depends on its blood-vessels, and the amount of vision on the integrity of the nerve fibres which merely pass through the disc, and

have a long course on each side of it, it is not surprising to find that the correspondence between the colour and vision is not always close. Little difference may be detected in the

FIG. 64. FIG. 65. FIG. 66.

FIELDS OF VISION FOR WHITE, RED, AND GREEN IN A CASE OF PAPILLITIC ATROPHY.

tint of the two discs, although there may be a great difference in vision.

DIAGNOSIS.—The ophthalmoscopic diagnosis of simple atrophy of the optic nerve rests especially on the pallor and the excavation, and the chief difficulty is due to the degree of pallor sometimes seen as a physiological condition. The existence of amblyopia, otherwise unexplained, is strong evidence that the pallor is pathological. The pallor of the temporal half of the normal disc may be great when the physiological cup is large, and, as already stated, may easily be mistaken for atrophy. It is certain that many normal cases have been described as "atrophy of the temporal half of the disc." It is doubtful whether an atrophy is ever confined to the temporal half of the disc, in which, ordinarily, the nerve fibres are very few. Although it is true that a slight degree of atrophy may produce the most distinct changes in this half of the disc, yet some pallor is practically always to be recognized, in such cases, in the nasal as well as in the temporal half.

Beginners sometimes mistake the white crescent of "posterior staphyloma" for part of the disc, and thus think the outer part of the disc, the colour of which is of such special

significance, is white. Occasionally, especially in myopic eyes, the choroid presents a zone of atrophy, soft edged, around the entire circumference of the disc, which then has a puzzling appearance. In both these cases careful observation will show that the pale zone encloses a well-coloured disc, and will prevent mistake as to its real nature.

The excavation which accompanies atrophic pallor is usually characteristic in its features and easily distinguished from other forms of excavation. But a large physiological cup may be bounded by a narrow rim of deeply-coloured disc; the limit may not at first be apparent, and the large, deep, sometimes grey, cup may be mistaken for the disc. Examination by the direct method at once shows the source of the error.

The excavation of atrophy commences at the sclerotic ring; this is a character also of the excavation of glaucoma. But the depth of the glaucomatous cup, its steep or undermined sides, and the course of the vessels over the edge, and their subsequent concealment by it are diagnostic.

Prognosis.—The prognosis of atrophy of the optic nerve, on whatever cause it depends, is always unfavourable in proportion to the actual destruction of fibres which has taken place, and to the extent to which the causes influencing the disease are beyond control. Primary atrophy is due to a tendency to degeneration over which we have little influence, especially when the atrophy is associated with symptoms of degeneration elsewhere in the nervous system. This statement is true of tabetic atrophy, in which, however, arrest is occasionally obtained, sometimes only for a year or two, in rare cases permanently. Secondary atrophy, from damage to the nerve, is often the consequence of causes which may pass away, and the prognosis is less uniformly grave than in primary degeneration. It must, however, always be somewhat uncertain, since it is often very difficult to form an accurate opinion of the nature of the morbid process on which the degeneration depends. In the atrophy which is consecutive to intra-ocular neuritis, we know that

during the early part of the process of cicatricial contraction the damage to the nerve fibres will increase, and the sight will go on failing. If sight is lost from such contraction some time before it reaches its maximum, the prognosis is very grave. If, however, the loss of sight is incomplete when the subsidence has nearly reached the level of the retina, some subsequent slow improvement may be hoped for, and this may, in the less severe cases, be great. In a case which I have published elsewhere,[1] for example, probably of tumour in the middle lobe of the cerebellum, there was at first double optic neuritis, with great swelling. On the subsidence of the optic neuritis, six months later, vision had diminished in both eyes to $\frac{1}{15}$. After this, vision improved, until, fifteen months after the first observation, it had risen to $\frac{2}{3}$, and the pupils, which had shown formerly no reaction to light, again acted. Under all circumstances, it is unhappily true that a disc which has lost all its normal tint never regains its vascularity, and useful vision is seldom recovered.

Some prognostic indications may also be drawn from the form of the affection of sight. The gravest, that which indicates not merely damage, but destruction of nerve fibres, is considerable contraction in the field of vision. In proportion as this is extensive the prognosis is grave. Lessened acuity of vision is of less serious prognostic significance. The change in colour-vision is least grave when this depends on a toxic cause, or on neuritis; but is most grave when it is due to a primary degeneration, and occurs early. Central scotomata rarely go on to complete atrophy.

Hence, the chance of restoration of useful vision in pronounced atrophy is small, but in some cases the progress of the disease may be arrested, for a time or permanently, and even improvement obtained, occasionally considerable in degree.

TREATMENT.—The treatment of optic atrophy is essentially that of the general condition on which it depends —toxic influences, excesses of all kinds, cerebral and

[1] "Trans. Ophth. Soc.," i. 117.

spinal disease, an abiotrophic tendency, &c. The treatment of many forms of atrophy which are due to an isolated ocular condition is beyond the scope of the present work. Cerebral processes may be influenced, if of syphilitic or tubercular nature.

In cases of primary atrophy the treatment has to be directed to the general health, and nervine tonics are the chief agents to be employed. The hypodermic injection of strychnia is the only measure which, in my experience, affords some prospect of arrest in simple atrophy, such as the tabetic form. One injection may be given daily, of the nitrate of strychnia, in any convenient place. The initial dose may be $\frac{1}{30}$ gr., increased to $\frac{1}{20}$ or $\frac{1}{15}$, *i.e.*, one to six minims of a 1 p.c. solution. It has to be continued for a long time; after six months, it may be intermitted one week in five. If it fails, two injections daily of $\frac{1}{20}$ may be tried. Remarkable tolerance is established, and physiological effects are scarcely ever produced by less than $\frac{1}{10}$th gr.

When perception of light is not entirely lost, the retina may be readily stimulated by an interrupted voltaic current, so as to give rise to a sensation of light; and this has suggested repeated stimulation of this character as a means of treating optic nerve atrophy. Some improvement has been said to follow treatment with the continuous current; but general experience has been unfavourable.

THE RETINA.

Apart from the vessels and the optic disc, the changes which are of medical importance are those which are special to certain general diseases, such as syphilis, albuminuria, leucocythæmia, pernicious anæmia, and the like. They will be described in detail in Part II., in the sections on the ophthalmoscopic changes in special diseases. The only

[1] Pye-Smith, "British Med. Journal," May 18, 1872; and Gunn, "Ophth. Hosp. Rep.," vol. x. pt. 2, June 1881, p. 161. Gunn found marked improvement only in cases where the atrophy was dependent upon retinitis pigmentosa.

common feature which these morbid states possess, is the development in the retina of hæmorrhages and white spots and patches. The hæmorrhages, their characters and significance, have been already described (p. 24). It may be convenient briefly to describe the forms of white patches which the retina may present under pathological conditions.

A diffuse, slight opacity of the retina may be due to the derangement of its normal structure, resulting from the effusion of serum among the structures which compose it. Such diffuse opacity occurs in embolism, neuritis, retinal arterial degeneration, and albuminuric retinitis; but in all, and especially in the two last, it is usually associated with structural changes. Circumscribed opaque white spots are due to change other than that of simple œdema, and commonly of four varieties: (1) Fibrinous exudations which undergo coagulation; (2) the accumulation of corpuscles, similar in appearance to those of the nuclear layer, and also to the white corpuscles of the blood, so that it is doubtful from which source they are derived; (3) fatty degeneration of the retinal elements, perhaps also in part of fibrine from the serum effused in simple œdema, and of the remains of blood clot; (4) a fibroid change, a process of "sclerosis" of the retinal elements, is described as an occasional cause of a white spot, but is more frequently confined to the perivascular tissues and vessel-wall.

These conditions are frequently combined. The fatty degeneration may exist alone, as the sole cause of a white spot. Corpuscular accumulation usually involves a good deal of fatty degeneration in the cells and in the disturbed retinal elements. Sclerosis of the retinal structures is also in most cases associated with fatty degeneration.

It is often impossible to say, from the ophthalmoscopic appearance, on what change the white spot depends. Minute granular-looking spots, brilliantly white, are commonly due to fatty degeneration of retinal structures or of nucleated cells, &c. Larger white spots, if soft edged, are commonly effused fibrine or accumulations of leucocytes, especially if situated

beneath the nerve-fibre layer. Fatty degeneration of the retinal structures is, however, commonly associated. White spots in the superficial layer of the retina are due usually to degeneration of the nerve fibres.

Growths in the retina sometimes occur in cases in which there are other growths elsewhere. The disc shown in Pl. I. 4 was from a boy who had cerebral tubercles, and whose other eye was the seat of a tubercular growth behind the retina.

The occurrence of miliary tubercles of the retina has been suspected by many observers. White spots are sometimes seen adjacent to the disc in cases of tubercular meningitis, and such a spot, in one case, I found to be made up of lymphoid cells like those of the nuclear layers in which it was situated. Bouchut has seen white spots at a distance from the disc, near the vessels. Microscopically, he always found them to contain only products of fatty degeneration. He suspected them to be caseous tubercles, but there was no direct evidence that this was their nature (*see* Part II.).

Since such white spots in the retina are present in many forms of retinal disease which occur secondarily to, and are significant of, general diseases, it is of great importance to distinguish them from other appearances which have a different significance.

First, it is necessary to discern whether the white spot is in the retina or in the choroid. Most choroidal white spots are due to atrophy, and their distinction is easy. The loss of the pigment and of the chorio-capillaris permits the white sclerotic to shine through; some choroidal vessels may have escaped destruction and course across the white patch; its edge is always more or less irregular, and usually much pigmented; or the choroid may exhibit adjacent slighter disturbance. It is easy to recognize, by the "paral-lactic test" (p. 91), (also in the direct method by attention to the necessary change of accommodation, or by artificial focus-sing), that the exposed sclerotic is some distance behind any retinal vessels which pass in front of it. Occasionally, how-

ever, a white spot in the choroid is due to a recent formation—an inflammatory "exudation," or a growth such as tubercle. This is prominent, and may be difficult to distinguish from a white spot due to change in the nuclear layers of the retina, but there may be adjacent pigmentary disturbance; the whiteness is usually less intense than in purely retinal affections, the edge is very soft, and sometimes it is evidently much behind the inner retinal structures. If the prominence is such as to disturb the course of the retinal vessels, it is easily perceived. It must be remembered that large choroidal exudations may cause opacity of the overlying retina.

White spots due to the persistence of the white substance of the nerve fibres, or to connective tissue at the back of the vitreous, may be mistaken for new formations in the retina. They have been already spoken of (pp. 5 and 6).

Pigmentary deposits may be left after extravasation of blood, but such are always small. More extensive pigmentation is commonly the result of the accumulation in the retina of the disturbed pigment, and is a consequence of choroido-retinitis, or of retinitis pigmentosa.

Retinitis pigmentosa appears sometimes to have an obscure connection with morbid states of the nervous system. More than one case may be met with in the same family. It occurs, as Liebreich first pointed out, not infrequently in the offspring of marriages of consanguinity. It has been thought to be connected with inherited syphilis, but the evidence on the subject scarcely supports the theory. It often occurs, however, in families in which there is a history of nervous disease. This is well illustrated by three out of four cases of the disease narrated by Nettleship.[1] Of the first patient, two cousins were epileptic and two insane. Of the second, the grandfather and great-aunt were insane, and an aunt half imbecile, and a brother paraplegic. Of the third patient, the mother was epileptic, and probably also suffered from retinitis pigmentosa. But it may be met with apart from such associations. It seems some-

[1] "Ophth. Hosp. Rep." ix. 170.

times to be the result of the toxin of an acute specific disease.

THE CHOROID.

Choroidal changes, like those of the retina, are for the most part the result of special diseases, and their characters will be described in greater detail in Part II. Hæmorrhages are rare, although their local effects are sometimes met with. The common changes consist in white spots, and in the disturbance of pigment which so constantly results from any changes in this structure. The white spots are either new formations or patches of atrophy. The differences between them have just been alluded to in describing the distinction from retinal changes. White spots, not atrophic, are the result of inflammation, or growth—tubercle or lymphadenoma. The latter are extremely rare, and only occur when the general lymphatic disease is well marked. Tubercles are isolated and small—rarely large. Pigment may be seen adjacent to the older formations. The evidence of the general disease is almost always so prominent as to prevent error. In acute choroiditis the white patches are large and often numerous: the signs of the dyscrasiæ associated with growths are absent, and there is often a well-marked history of syphilis. In all recent forms, the vitreous is commonly cloudy from the presence of minute dust-like opacities: these are best seen on using a high convex lens behind a plane mirror by the direct method. The results of previous choroiditis are conspicuous atrophic and pigmentary changes, often associated with pigmentary deposits in the retina. It must be remembered that this pigment frequently occupies only, or chiefly, the peripheral portions of the fundus, and an examination, confined to the neighbourhood of the optic disc, may be insufficient to discover it. The changes are very important, on account of the frequency with which the inflammation causing them is the result of syphilis. They are also interesting to the physician as associated, in some other cases, with evidence of a family tendency to nervous disease. It is possible that

inherited syphilis may be the link between these morbid states.

Choroidal exudations (local) sometimes occur about puberty, resembling choroidal tubercles, and it has been suggested that these are really foci of scrofulous or tuberculous inflammation.

Chronic choroidal degenerations sometimes occur as a senile change, possibly in consequence of general arterial degeneration. Circumscribed changes may result from hæmorrhage. Amyloid degeneration of the choroidal arteries was described by Knapp in a case in which hæmorrhage occurred.

Embolism of choroidal vessels was believed by Knapp to be the cause of morbid appearances in two cases of heart disease observed by him.[1] In each there was sudden affection of sight, at first general and then central, accompanied by achromatopsy. Corresponding to the scotoma, there was a localized retinal opacity with hyperæmia. The opacity, ascribed to effusion, extended to the optic disc. Sight, and the appearance of the fundus, ultimately became normal.

[1] "Arch. f. Ophth.," Bd. xiv.

PART II.

OPHTHALMOSCOPIC CHANGES IN SPECIAL DISEASES.

DISEASES AND INJURIES OF THE NERVOUS SYSTEM.

DISEASES OF THE BRAIN.

In diseases of the brain, two forms of ophthalmoscopic change may be met with:—First, those which are a consequence of the general condition by which the cerebral disease is produced—*associated changes*; and, secondly, those which are the consequence of the cerebral disease—*consecutive changes*.

Anæmia and Hyperæmia of the Brain.

It has been supposed that the state of the circulation in the eye and brain correspond, and that the anæmia and hyperæmia of the brain are revealed by similar conditions in the fundus oculi, and especially in the vessels of the retina and optic nerve; the vascularity of the choroid being too great to permit of the recognition of any change in its circulation. But, as already stated (p. 19), this conclusion, if true at all, is true only within narrow limits. Local conditions, chiefly the intra-ocular tension, but also the venous anastomoses, so influence these vessels, that they undergo little alteration when changes occur in the state of the vessels of the brain. The eyeball participates in variations in the blood-supply to the whole head, but it does not share simple vascular states of the brain (in

which the rest of the head does not participate) to a degree that can render it an index to their existence. This statement applies especially to the retinal vessels, in which alterations can be most readily perceived.

Cerebral Hyperæmia.—There is no sufficient evidence to show that the vascularity of the disc or retina is altered by any transient cause of cerebral congestion, unless the whole head suffers. Yet in some cases of long-continued vascular disturbance, and in morbid states which are ascribed, with some probability, to cerebral congestion, ophthalmoscopic changes are sometimes to be seen—a transient increase of colour, sometimes with slight blurring of the edge of the disc. But in most of these cases there is evidence of grave functional disturbance of the brain or prolonged hyperæmia. The bright injection of the discs described by Macnamara as occurring during the headache produced by exposure to the tropical sun, increasing to papillitis when actual meningitis occurs, may be an instance of this.

This conclusion—the absence of any marked vascular alteration in the eye in changes in the cerebral circulation—is at variance with early statements and *a priori* theories; but it is abundantly supported by skilled observers.[1]

Lastly, it is *probable* that when cerebral hyperæmia is due to blood states, the cause may sometimes influence also the optic disc and induce congestion. But this has not yet been proved.

Anæmia of the Brain is rare as a primary vascular condition, except as part of a general cephalic anæmia. It is questionable whether any diminution in the tint of the

[1] See, for instance, the statements of Manz, Schmidt-Rimpler, and others, at the discussion at Heidelberg, reported in the "Ann. d'Oculistique," vol. lxxiv. 1875, p. 262, *et seq.*

It must be remembered that "congestion of the brain" as a name is exceedingly convenient; the condition is invoked with a readiness that cannot but excite surprise in those who know how different is the significance of the symptoms it is considered to cause. Red discs alone prove nothing. To be of significance the redness must lessen in an unequivocal degree as symptoms pass away.

disc has ever been observed to coincide with a diminution in the amount of blood within the brain alone.

When the cerebral anæmia is part of a similar state affecting the whole head, the retina certainly participates, although it is not often that an opportunity is obtained of observing this with the ophthalmoscope. Loss of function of the retina affords evidence of its participation; transient loss of sight, probably from this cause, may attend syncopal seizures. In an instance that came under my observation, a lad engaged in a stooping occupation in a hot crowded room, felt faint, and went out into the cool night-air. On re-entering the room he could not see: the room was absolutely dark to him. After sitting still for a few minutes sight slowly returned. It is hardly conceivable that the loss of sight was the result of anæmia of the brain, because the other cerebral functions were not affected, and the loss of sight persisted after he otherwise felt quite well. Probably the retina shared the cephalic anæmia (due to heart-failure), and suffered in function more and longer than the brain.[1]

Inflammation of the Brain.

Acute general inflammation of the brain is only definitely known in association with meningitis. The latter is the dominant lesion, and to it the symptoms are customarily ascribed. Of ophthalmoscopic changes in acute inflammation of brain without meningitis, we know nothing. Such cases of "active hyperæmia" as those described in the last section, from insolation, may be regarded as encephalitis. There is no sharp line to be drawn between "active congestion" and "inflammation," but there appear to be no pathological facts to warrant us in regarding the morbid process in

[1] This fact is one of some significance. It suggests how extremely sensitive to sudden influences the retina is in its function, and this must entail a similar susceptibility to derangement of nutrition from similar disturbances.

these cases as actual cerebritis. Local acute inflammation is usually secondary to a local cause. But it is possible that any local inflammation of the brain may cause neuritis if it continues for a sufficient time.

There is, however, a class of cases to which the term "chronic encephalitis," or, perhaps, more accurately, "chronic cerebritis," appears fully applicable, and in which there may be very marked ophthalmoscopic changes. These cases present evidence of mental and motor failure, the latter may be local and attended by convulsion. Death may be preceded by coma. Headache is often severe. There are not the tremors or mental peculiarities of general paralysis, the symptoms resembling much more closely those of cerebral tumour. Post-mortem there is no sign of meningitis; the brain may present evidence of degeneration, sometimes of wasting, but no "focal" disease. Such cases may be attended by optic papillitis very similar to that found in cerebral tumour, due most probably to the propagation of an irritative process from the cerebrum along the nerves. A well-marked case of this kind has been described by Hughlings-Jackson.[1] Dr. Sutton's microscopical examination of the convolutions showed only an undue number of the "spherical nuclear bodies," and in places, instead of the normal pyramidal nerve cells, were large numbers of staining nuclei, with unstaining cell-bodies around them. In places these nuclei were aggregated into groups of ten or twenty. The eye, examined by myself, presented the characteristics of moderate papillitis, the swollen papillæ being infiltrated with nuclear bodies similar to those seen in the brain. Similar corpuscles were so abundant throughout the optic nerves as to justify the assumption that the neuritis had been "descending." A case published by in Noyes, 1873, was probably similar. Double optic neuritis, passing into atrophy, was accompanied by severe pain in the head, and paralysis of various cerebral nerves and unsteady gait. After death, no lesion of the brain was discovered. A case of the same kind, also accom-

[1] "Ophth. Hosp. Rep.," viii. 445.

panied by optic neuritis, has been recorded by Stephen Mackenzie.[1]

In the rare cases in which hæmorrhage, or softening from vascular occlusion, causes optic neuritis, this is probably produced through the agency of such secondary inflammation.

Cases are sometimes met with in which we have a difficulty in assigning to inflammation or growth the chief share in the morbid process. Such cases may be accompanied by descending neuritis, and simulate closely the symptoms of cerebral tumour. Pl. IV. 2 shows the optic disc in a case in which local injury, years before, had caused the production of cheesy degenerating tissue beneath the membranes over certain convolutions, and a more widely spread, but irregularly distributed, meningitis had led to vascular disease, from the effects of which the patient died. The optic nerves were infiltrated with nucleated cells, and "miliary abscesses" were found in the optic tracts (Figs. 25 and 35).

Cerebral Hæmorrhage.

Associated Changes.—The common form of cerebral hæmorrhage is due to the rupture of "miliary aneurisms"; that is, minute arteries suffer in the nutrition of their wall, which yields before the blood-pressure, and dilatations are produced. The conditions which give rise to these aneurisms seldom influence the arteries of the retina, but the capillaries may undergo degeneration, and thus small extravasations occur. In Fig. 3, p. 17, are shown capillary aneurisms from a case in which cerebral and retinal hæmorrhages coexisted. Retinal and cerebral aneurisms may occur together in kidney disease, but are rare. The aneurisms are depicted in Pl. X. from a case in which all the conditions for the production of cerebral hæmorrhage were present in extreme degree. The patient was a woman aged only

[1] "Brain," vol. ii. p. 257. See also Elschnig, "Arch. f. Augenheilk.," Bd. 26, p. 56, for a case in which such a change in the brain was accompanied by retro-ocular neuritis, extending to the pial, but not to the dural sheath.

thirty-six, who had advanced kidney disease with great cardiac hypertrophy, and high arterial tension. There was obvious change in the coats of the retinal arteries, with several large hæmorrhages, and in a few places, aneurismal dilatations. She died a few weeks later from cerebral hæmorrhage, a striking instance of coincident states.

Retinal Hæmorrhages are present in a considerable number of cases of cerebral hæmorrhage, and furnish an indication of considerable value. Their most frequent cause is that which is the most frequent cause of cerebral hæmorrhage, chronic renal disease. They may exist, as in Pl. VII. 1, without other purely retinal change, or may form part of albuminuric retinitis (Pl. VIII. 1 and X. 1). In either case they indicate the existence of the conditions which favour vascular degeneration and rupture,[1] and the retinal arteries will usually be found to exhibit those degenerative changes previously described (*see* p. 17). In the retina shown in Pl. VIII. 1, capillary dilatations and other changes were found. The retinal hæmorrhages are often associated with cardiac hypertrophy, and thus accompany all the most potent causes of cerebral hæmorrhage. It must not be concluded, however, that the presence of albuminuric retinitis or even extravasation proves a cerebral lesion to be hæmorrhagic. The disease of the kidneys is associated, not only with the minute aneurisms that lead to hæmorrhage, but also with the atheroma of the larger arteries that leads to thrombosis within them. Hence, softening due to the closure of atheromatous arteries is often associated with retinal changes due to kidney disease, especially after middle life, and have weight in the differential diagnosis, only in confirmation of other evidence.

In other conditions retinal hæmorrhages point to a state in which cerebral hæmorrhage is likely to occur, and have more decided significance, since these other causes of retinal

[1] They have been said to occur chiefly in the eye corresponding to the side of the cerebral lesion, but a large number of cases must be observed to substantiate the fact. *See* R. T. Williamson, "Brit. Med. Jour.," 1898, i. p. 1515.

hæmorrhage do not produce arterial atheroma. They occur, for example, in pernicious anæmia (Pl. IX. 1) and in leucocythæmia (Pl. IX. 2), and in the latter disease the brain stands second in frequency as the seat of internal hæmorrhage.[1]

The significance of retinal extravasations as indications of the probability of the future occurrence of apoplexy may be overrated. They are not uncommon in old and gouty persons, who do not suffer subsequently from cerebral hæmorrhage. Perhaps this is, in part, due to the fact that the conditions in which they arise are such that many other causes of death coexist. Moreover, the existence of the conditions favourable to an event does not necessarily involve a balance of probability in favour of its occurrence.

Among the very rare causes of cerebral hæmorrhage are syphilis and heart disease, by the mechanism of aneurism. Changes in the fundus may conceivably be sometimes of service in the differential diagnosis, but death usually anticipates the need for such assistance.

Consecutive Changes.—Hæmorrhage into the substance of the brain is not usually attended with any secondary ophthalmoscopic changes. Neuritis has been described in a few instances, but so rarely that the question arises, whether, when met with, it has really been due to the cerebral lesion. Kidney disease so often coexists as to explain the occurrence of optic neuritis when it exists alone as a distinct sequel to hæmorrhage. But here, as in so many other conditions, the insufficiency of a single cause does not exclude an influence exerted in association with another cause. Even if secondary inflammation is insufficient alone, it may determine the occurrence of neuritis in conjunction with renal disease. This is probably the explanation of the few cases in which considerable optic neuritis has been observed in pure hæmorrhage. One such case has been described by Hughlings-Jackson; ten weeks after an attack of cerebral hæmorrhage, the discs presented the appearance of the later stage of neuritis. The patient died a week subsequently, and

[1] Retinal hæmorrhages not included. *See* Gowers' "Leucocythæmia," "Reynolds' System of Medicine," vol. v.

the necropsy revealed a large extravasation into the middle cerebral lobe, and a few specks of hæmorrhage into the corpora quadrigemina. Another case is recorded by Bristowe.[1] The hæmorrhage was in the posterior part of the optic thalamus. Robin[2] mentions a case with well-marked neuritis, such as is met with in tumours, in which the autopsy revealed a clot of blood, the size of a walnut, compressing the pons. In this case the neuritis can hardly have been the result of the extravasation. In a case described by Gemuseus,[3] double neuro-retinitis was observed during life, and, after death, numerous hæmorrhages were found in the brain.

In many cases of intense optic neuritis met with in cerebral hæmorrhage, the blood has been extravasated into a soft growth in the brain, to which the neuritis has really been due. In one case, observed during life, the post-mortem revealed a large clot surrounded by secondary softening, but the latter was found to be a very soft grey glioma into which the hæmorrhage had occurred. It is probable that this is the explanation of most cases of considerable optic neuritis associated with hæmorrhage. The nature of soft brain tissue adjacent to the extravasation is easily mistaken.

When, however, the hæmorrhage is into the meninges, slight optic neuritis may exist. The blood may pass into, and distend, the sheath of the nerve, as has been found (in a case of my own) in meningeal hæmorrhage from fracture of the skull; also in rupture of an aneurism of the middle cerebral (Mackenzie), in rupture of an intra-cerebral extravasation into the meninges (Michel), and in hæmorrhagic pachymeningitis (Manz). Opacity and blurring of the outline of the disc, with slight swelling, may be thus produced. Retinal extravasations may co-exist, as in a case figured by Poncet.[4] Early changes in the papilla, in a case of undoubted

[1] "Trans. Ophth. Soc.," vi. 363.

[2] "Des Troubles Oculaires dans les Maladies de l'Encephale," Paris, 1880, p. 284.

[3] "Klin. Monatsbl f. Augenheilk.," 1880, p. 380.

[4] "Atlas" of Perrin and Poncet.

cerebral hæmorrhage, would thus be probable evidence that the blood was effused into the membranes.

In rare cases optic nerve atrophy has been met with in association with cerebral hæmorrhage. Thus a case is recorded by Vulpian [1] in which blindness supervened on an attack of apoplexy. Death occurred fifteen years later, and the remains of an old hæmorrhage were found in the left corpus striatum. Both optic nerves and optic tracts presented grey atrophy. The connection between the two is obscure, but probably indirect. It is important to remember, in medical ophthalmoscopy, as in every branch of medicine, that coexistence does not necessarily mean causation.

Cerebral Softening.

In softening of the brain, marked ophthalmoscopic changes are rare as the result of the cerebral mischief, although they occasionally occur. Most of the cases have been softening from embolism, not thrombosis from primary vascular disease. Changes in the fundus oculi may also result from the same causes as lead to the cerebral mischief.

1. Embolic Softening : (*a*) *Associated Changes.*—Embolism of the trunk or of a branch of the central artery of the retina may occur before or after embolism of a cerebral artery ; very rarely at the same time (*see* p. 32). When the two occur at the same moment, the demonstration of the nature of a cerebral lesion is brought almost to a perfect form. An instance of such coincidence is afforded by the case illustrated in Pl. X. 2. The plug in the retinal artery is shown in Fig. 4, p. 35.[2]

(*b*) *Consecutive Changes.*—When the artery plugged is the middle cerebral, marked disturbance of the circulation might be expected in the eye which derives its blood-supply from the same trunk. Any signs of such disturbance have, however, hitherto escaped attention, and probably the free

[1] Galezowski : "Journal d'Ophthalmologie," Jan. 1872.

[2] For fuller details see description of the plate.

anastomoses of the circle of Willis carry off any excess of pressure.

If the condition of the disc is carefully observed from time to time, I believe that a state of congestion may often be observed a few weeks after the onset of embolic softening, especially in those cases in which the cerebral damage is extensive and leads to mental change.[1] Actual neuritis has been observed in a few cases, distinct, moderate in intensity, coming on some days or weeks after the cerebral lesion, running a subacute course, and slowly subsiding. In a case described by Broadbent,[2] a man, aged nineteen, with mitral disease, was seized with left hemiplegia and impairment of sensation. Nine days after the onset the margins of the optic discs were ill defined, with swelling, the retinal veins were large, dark, and tortuous. By the eighteenth day the paralysis had improved, but the papillitis persisted, with perfect sight. During the second week it slowly lessened. Subsequently symptoms of ulcerative endocarditis came on, and caused death four months after the onset of the hemiplegia. There was softening in the right occipital lobe, and involving the fibres passing to it from the thalamus.

Double neuritis, with slight changes in the contiguous retina, was seen by Stephen Mackenzie[3] in a case of left hemiplegia, clearly due to embolism. Three days after the onset, the discs (previously normal) were swollen, and three weeks later the swelling persisted, but with a good deal of opacity, the vessels being "buried in exudation." One or two hæmorrhages existed close to the discs.

A grey infiltration, incompletely veiling the disc, and extending into the adjacent retina, from a case of hemiplegia

[1] In a young man, with mitral disease and left hemiplegia from embolism, the increased redness of the disc, with slightly softened outline to direct examination, developed in both eyes under observation, and was so marked that I thought neuritis was coming on. It became stationary, however; soon lessened in the left eye, and much more slowly in the right. Coincidently with it there was marked and increasing mental failure, persistence of the complete hemiplegia, and rapid development of the foot-clonus.

[2] "Clin. Trans.," vol. ix. 1876, p. 62.

[3] "Brain," Jan. 1879.

with mitral regurgitation, in a child of seven years, is figured by Bouchut.

Most of the above cases seem to be distinct instances of the association of neuritis and softening. It is important to note that all were cases of softening from embolism, that in most the plug came from valves the seat of actual recent inflammation, and that in some the development of the optic neuritis was accompanied by evidence of wide-spread disturbance of the cerebral functions. There is nothing in the mere process of necrotic softening, the breaking up of the nerve-elements into discontinuous particles separated by liquid, which can cause optic neuritis, according to our present knowledge. But the process is never one of simple necrosis of the tissue-elements. Adjacent inflammation always accompanies it, and emboli from an inflamed cardiac valve have a special power of exciting this process, apparently because they convey infective organisms. The inflammation thus excited or conveyed varies in its intensity and in its tendency to spread, just as the endocarditis varies in its "malignancy." Hence the secondary inflammation has its character modified by the influence of the plug; organisms may readily find their way into the adjacent brain tissue and determine the characters of the inflammation. We can thus understand such differences as we have noticed, and that in some cases the spread of the irritative process should lead to an optic neuritis, slight or severe, which is absent in others.

It may be well, however, again to remark how easily the error may be made of mistaking a soft glioma for a patch of softening.[1] In a case recorded by Leber of supposed neuritis from softening, the fact that the "softening" was a soft glioma was not suspected until discovered on microscopic examination.

Atrophy of one optic nerve is said to succeed softening.

[1] The following case has been recorded by Drs. Darby and Upham ("Boston Med. and Surg. Journal," vol. lxxii.) as one of softening, in which, however, there was no evidence of embolism. A man aged twenty-six had a hemiplegic attack, followed by fits and double "neuro-retinitis" with hæmorrhages. A necropsy some months later revealed a

embolic or other, just as it has been observed to succeed hæmorrhage. This result is supposed to be due to the seat of the lesion being such as to damage the nutrition of some part of the brain to which the optic fibres are related, but the relation is not beyond doubt. Embolism of the middle meningeal artery, which supplies the dura mater near the optic nerve, is said also to cause atrophy of the latter.

2. Softening from Thrombosis.—(1) *Arterial.*—This may be due to syphilitic or degenerative disease of the vessels, or to blood-changes.

Syphilitic Disease.—In softening from syphilitic disease of vessels, associated ophthalmoscopic changes are common; consecutive changes are very rare. The associated conditions are the various changes which are due to syphilis, and which need not be mentioned here. They come practically under the cognisance and teaching of the ophthalmic surgeon. This is because their active stage affects sight and seldom coincides with disease elsewhere. But the changes in the eye in inherited syphilis come often under the notice of the physician, and in both the inherited and acquired disease the signs of previous mischief are of great value.

In cerebral softening from arterial disease, such indications are most significant in persons who have not reached the period of life at which vascular degeneration is common. In the latter condition, the recognition of constitutional syphilis still leaves us in some doubt, and care must be taken to avoid attaching undue weight to its signs. At the same time syphilitic vascular disease does occur in the degenerative period. Syphilitic disease and atheroma have been observed post-mortem in the same individual. In doubtful cases, the recognition of the ocular signs of syphilis should always lead to a trial of specific treatment. Iodide

peculiar softening of the corpus striatum and optic thalamus, grey and white gelatinous soft tissue, to the naked eye very like a glioma, but, on microscopic examination, only the signs of degeneration were visible. It is to be remarked, however, that many parts of these tumours may contain, and even appear to consist only of, products of degeneration. A careful search may be necessary for the very delicate cells of which they consist.

should not, however, be given in very large doses (more than 15 grams) on account of its influence in promoting coagulation of the blood.

Consecutive alterations in the eye are very rare in softening from syphilitic disease of vessels. Only one case has been recorded in which neuritis was apparently due to this cause.[1] In the few other cases, syphilitic growths in the brain were associated with the vascular disease, and the ocular change was due to the former, not to the latter. This was probably true of one case I have seen; a fortnight after the sudden onset of hemiplegia, after constitutional syphilis, there was slight distinct optic neuritis; and preceding pain in the head for six months rendered it probable that there was more than arterial disease. The absence of neuritis is sometimes of some diagnostic significance when there is doubt whether the lesion is a growth or slow effects of arterial disease.[2]

Is papillitis ever a direct effect of the syphilitic poison? This question we cannot at present answer. Syphilis probably can cause a retro-ocular neuritis; it certainly can cause retinitis involving the papilla. Cases are met with in which the neuritis may be syphilitic, and this possibility makes it the more doubtful whether papillitis in softening from syphilitic arterial disease is consecutive. Moreover, a syphilitic growth may coincide with such softening, and on the whole this is the probable explanation of cases in which considerable optic neuritis has been observed to coincide.

Degenerative Disease: "Atheroma."—Cerebral softening from this cause may occur without any similar morbid state of the retinal arteries, which are below the size in which "endarteritis deformans" is common. But thickening of

[1] Leber, "Zeitsch. f. Klin. Med.," 1882, Bd. ii. p. 173. The patient, a man aged eighteen, died from limited softening of the inner part of the right crus and adjacent part of the pons, due to syphilitic disease of the extremity of the basilar artery; and seven days after the onset of the acute symptoms there was found "neuro-retinitis with choked disc as in cerebral tumour" (Dr. Hiller), although no other lesion than syphilitic disease of the vessels could be found. The details from an ophthalmoscopic point of view leave much to be desired.

[2] *See* a Lecture on Syph. Vasc. Disease, "Lancet," Nov. 9, 1901.

the wall, the "silver wire" aspect, and obstruction of the circulation in a vein that is crossed, or undue tortuosity of the retinal arteries, have been often observed. True atheroma is an affection of the inner coat, and such alterations as are met with consist in a thickening of the middle and outer coats. (*See* "Bright's Disease.") The conspicuous change depicted in Pl. X. 1 is manifestly seated in the outer coat of the vessel or in its sheath. (*See* p. 17.)

All forms of albuminuric retinitis may be associated with cerebral softening. They are also associated, in the same manner, with cerebral hæmorrhages, and hence the affection of the retina, and even hæmorrhages in it, are evidence only of probable disease of the arteries of the brain. In the case figured in Pl. VII. 1, for instance, although there was a retinal hæmorrhage due to the effect of chronic renal disease, the cerebral symptoms pointed unmistakably to softening rather than to hæmorrhage.

Consecutive changes are very rare in senile arterial thrombosis. Optic neuritis certainly due to this cause is scarcely ever met with.[1] In the few cases on record it is probable that the papillitis was due to the renal disease—a source of fallacy to be carefully borne in mind.[2] Atrophy of the discs has, in rare cases, been observed to supervene, but there is doubt whether it is ever truly consecutive.

In some cases, however, the obstruction by thrombosis of the internal carotid may give rise to alterations in the eye, which have been hitherto observed only after death, but which must be attended by marked ophthalmoscopic changes. Such a case was described long ago by Virchow.[3] A man aged forty-six who had an attack of apoplexy, leaving right hemiplegia, died from a melanotic cancer of the liver. The

[1] *See* Wilbrand, "Arch. für Ophth.," Bd. xxxi. p. 119, Pl. 3.

[2] Most recorded cases may be thus explained. In the remarkable case figured in Pl. IV. 2, optic neuritis coexisted with softening from extensive arterial disease, the results of old traumatic meningitis, but inflammatory (?) growths existed beneath two old fractures of the skull. The man had had syphilis, but the lesions presented no syphilitic character.

[3] "Arch. für Path. Anat.," Bd. x. 1856, p. 189.

internal carotid was obstructed by a thrombus, probably spontaneous, since no embolus was found, and there was fatty and calcareous degeneration of the wall of the vessel. There was a large area of softening in the left hemisphere. The ophthalmic artery was patent, a collateral circulation having been set up. The vitreous was transparent, the retina thickened, and around the papilla were four opaque white spots, which were, however, found to be due to the persistence of the medullary sheath of the nerve fibres.[1] The ganglion cells were granular. The elements of the nuclear layers showed a tendency to arrange themselves in lobular cylinders. Another case of the same character which came under my observation has been before alluded to (p. 32), and in it the ophthalmoscopic changes would probably have been much more striking. Although the origin of the ophthalmic artery was closed by clot, the central artery of the retina retained a channel, narrowed by clot formed upon its walls. Some retinal branches were pervious, others closed. The retina presented atrophy of all its structures, and was reduced to about two-thirds of its normal thickness.

It is important, therefore, to watch the fundus continuously in cases of thrombosis in the region of the internal carotid. It is probable that the obstruction of the carotid would always be accompanied by a sudden diminution in the size of the retinal artery; the degree of this, and the occurrence of parenchymatous changes in the retina, depending on the anastomoses of the ophthalmic artery. These are usually abundant, chiefly with the facial, but also to a less extent with the middle meningeal.

Softening from Arterial Thrombosis due to Blood States.—In this condition, which is rare except in the puerperal

[1] A curious instance of the pitfalls of which the ophthalmoscopist must beware. Opaque white areas adjacent to the disc in such a case might easily have been thought to be the pathological changes, which the observer expected to find. The author was once asked to see a case in which vague cerebral symptoms were ascribed to syphilis solely on the ground of white areas of opaque nerve fibres, quite characteristic, but misinterpreted.

state, ophthalmoscopic changes have been found only in cases of septicæmia (*q.v.*).

Softening from Venous Thrombosis. — Ophthalmoscopic changes are unknown. In thrombosis of the cavernous sinus, it is said that there may be double optic neuritis and exophthalmos. Slow obliteration of this sinus, however, may cause no ophthalmoscopic changes.

3. Primary Softening.—Primary softening of the brain is still a region of cerebral pathology of which we know little. Apparently three forms occur, acute and subacute inflammatory softening, and a senile form of chronic softening.

Inflammatory Softening.—The acute form is only known in connection with injuries in which meningitis is never absent, and ophthalmoscopic changes must be ascribed to this rather than to the morbid process in the brain.

Subacute Softening is a probable lesion, symptoms suggesting it being met with especially in gouty persons, but no optic neuritis has been seen in connection with it—a fact of much importance, since it is upon this and upon the occasional retrogression of the symptoms that the diagnosis from tumour chiefly rests.

Chronic Softening is a senile lesion, but is extremely rare. A few cases have been described, but it seems not to be attended with ophthalmoscopic changes.

Abscess of Brain.

The only changes known are consecutive. Optic neuritis occurs in many cases, probably in at least two-thirds. It is similar in character to that met with in tumour, but seldom reaches the high degree often met with in intra-cranial growths. The papillæ are swollen, and often opaque, the vessels concealed, and hæmorrhages may be present. But neuritis is absent in some cases, and it is difficult to discern the conditions which determine its

occurrence. One important element in its production seems to be that of time. The more acute the process of suppuration the more likely is optic neuritis to be present. But this element in causation is influenced, on the one hand, by the fact that optic neuritis needs time for its development, and, on the other, that the more septic the character of the suppuration, the more rapidly neuritis seems to develop. In cases in which there is reason to think that the abscess has existed for more than four weeks, neuritis is found in about three-fourths of the cases. Yet cases are met with of long duration with normal discs. Nor can any relation be traced to either seat or extent of the abscess. Neuritis has been absent, or trifling, when the abscess was in the frontal lobe, and even when it has been secondary to suppuration[1] in the frontal sinus and caries of the bone. There was little change in a case in which the abscess was said to have occupied almost the whole of the left cerebral hemisphere.[2] Yet in even small abscesses in any part, optic neuritis often occurs and presents the same features as in cerebral tumour. It sometimes attains the intensity of the "choked disc" (stauungs-papille), with much swelling, dilated veins and hæmorrhages, a condition which must be regarded simply as an indication of degree of inflammation. As a rule, the optic neuritis, if confined to, or greater on, one side, has been on the side corresponding to the abscess.

The changes found in the optic nerves are similar to those met with in cases of tumour. Dropsy of the sheath has seldom been noted, apparently because it has seldom been looked for.

The chief difference between the cases of abscess, with and without neuritis, seems to be that of course. Acute local suppuration is seldom unattended with neuritis if it lasts more than a month. This may be due, as we have seen, to the influence of the acute process, coupled with the time required for the development of the secondary result. An

[1] *E.g.*, Herzfeld, "Berlin. Kl. Wochenschr.," 1901, 43.
[2] K. Muller, "Archiv. f. Ohrenheilk." 1900.

abscess which forms slowly may exist for long, and attain a wide extent, without causing this effect. The influence of increased intra-cranial pressure is probably to augment the degree of neuritis, and thus, by the compression of the veins within the swollen papilla, to cause the aspect of "choked disc" with hæmorrhages, but its influence has not yet been duly ascertained from facts.

For the most part, strange to say, little has been ascertained regarding optic neuritis from abscess beyond the general facts that are true of neuritis from tumour.

Tumours of the Brain.

A.—*GROWTHS.*

Associated Conditions.—Growths may occur in the eye, of the same nature as the growth in the brain, but such cases are not common. The disc shown in Pl. I. 4 is the left disc of a boy, whose right eye was the seat of a tubercular growth, and in whose brain there was another similar tumour, of which vomiting and optic neuritis were the only signs. In such a case the ocular growth becomes an important symptom. Choroidal miliary tubercles might be expected to be found occasionally in cases in which a tubercular mass exists in the brain, but they occur rather in acute general tuberculosis, while tubercular tumours of the brain are rare in that condition. The nature of these is rather that of the tubercular condition that we associate with the word "scrofula." This differs in course and associations from acute tuberculosis, although presenting the same bacilli. Hence choroidal tubercles are met with far more frequently in cases of tubercular meningitis than in tubercular tumours.

Consecutive Changes.—Optic neuritis is *the* ocular lesion of intra-cranial growths, which are, on the other hand, its most frequent causes. It is present, in various degrees, in a large proportion of the cases of intra-cranial tumour; in what proportion cannot be determined by statistics from published

cases, on account of selection for publication on special grounds. From my own experience I should say that neuritis occurs in about four-fifths of the cases. This is a much smaller proportion than has been deduced from published cases. Annuske and Reich, for instance, collected eighty-eight cases with ophthalmoscopic examination and autopsy, and found that there was no ophthalmoscopic change in only five per cent. But these cases were recorded during the period when ophthalmoscopic observation possessed the interest of novelty, and a far larger proportion of cases with neuritis has probably been published than of cases without neuritis.

It does not seem possible at present to say on what the occurrence of optic neuritis depends; why it is present in the majority, absent in the minority. Position of growth has apparently no direct influence on its occurrence, and only an indirect influence, insomuch as secondary meningitis near the nerves is more considerable when the tumour is not far from that part of the base. But the influences through which neuritis is caused seem to be exerted from any situation. It has been met with in tumours of every part of the cerebral hemispheres, of the pons Varolii, the crura cerebri, the cerebellum. Tumours of the medulla below the pons often cause death too quickly for optic neuritis to be developed; but Barlow has met with a case of neuritis from a small tumour in the middle of the medulla oblongata. It has been thought that tumours of the anterior lobes are more uniformly attended with neuritis than those of other parts, but growths in this part, even of large size, may be unattended with optic changes.

Nor does the nature of the tumour apparently influence the development of neuritis. It occurs with every variety—glioma, sarcoma, tubercle, syphiloma. The most frequent forms of tumour are those which are most often associated with optic neuritis; and they are also those in which neuritis is most frequently absent—syphilomata, tubercles, and gliomata. At the same time, growths that infiltrate and only damage the nerve elements late in time, and little

in comparison with the amount of the growth, seem to have less tendency to cause neuritis than those which damage readily. A like difference—perhaps, indeed, related—is seen from the amount of adjacent inflammation that is produced. The greater these secondary processes about the tumour, the more readily does neuritis occur. Hence the nature of a growth has an indirect, though not a direct, effect. A glioma infiltrating almost the whole of the medulla oblongata, which was under my observation for two months before death, caused no optic neuritis. It is often absent in gliomata of the cortex, especially if of slow development.

The size of the tumour also seems to have little influence. I have twice seen syphilomata the size of half an egg without optic neuritis. One of the largest intra-cranial tumours I have met with was a sarcomatous growth, the size of the closed fist, growing from the dura mater, and compressing, not invading, the brain over the posterior portion of the parietal lobe, a tumour which must have increased the intra-cranial pressure as much as it is ever increased directly by a growth. In this case the discs, repeatedly examined from soon after the onset of the symptoms until death, about six months later, were perfectly normal.[1] A similar case is described by Byrom Bramwell.[2] On the other hand, Benedikt has recorded a case of well-marked neuritis with much swelling and hæmorrhages, due to a tubercle of the pons Varolii no larger than a cherry, without meningitis.

The chief facts at present known regarding the mechanism by which optic neuritis is produced have been already discussed (p. 78). Some points having special reference to tumour may be again adverted to. Encephalic tumours seem not to cause neuritis by the simple effect of their bulk on the intra-cranial pressure. Yet the effect of increase of

[1] The case (which occurred long ago) is impressed on my memory as the cause of much apprehension. I last examined the eyes in a small room in a southern suburb, and as the sunshine was streaming in at the window, I used it, and obtained an excellent view of the normal discs. It occurred to me afterwards that I might thereby have destroyed the retina. The patient died a week later, and I found the tumour described.

[2] Bramwell, "Intra-cranial Tumours," pp. 11. 12.

intra-cranial pressure has been revealed by the constant diminution of neuritis after this has been relieved by trephining, whether or not the tumour was removed. A slight neuritis usually passes away; if the inflammation has been so great as to fill the papilla with inflammatory products, these of necessity persist and contract, and the operation has little effect in saving sight. Hence it is probable that the relief afforded is to the lymphatic and vascular channels, and that the hindrance to the return of their contents is a powerful augmenting influence, whatever be the mechanism by which the inflammation is primarily induced.

Rapidity of growth has been thought to influence the occurrence of optic neuritis, but although it may be one factor in determining the rapidity or slowness of the course of the neuritis, it does not seem alone to determine its occurrence.

One mode in which neuritis is produced may sometimes be distinctly traced post mortem, namely, the mechanism of meningitis. The disc shown in Pl. I. 3, for instance, was in a case of tumour originating in the pineal body and invading the anterior corpora quadrigemina. The changes in the disc were very gradual in development, and moderate in degree. There was no general meningitis, but the orbital lobules were gently adherent, and fine shreds of lymph were visible on the dura mater after their separation. The optic nerves in front of the commissure were swollen and reddened. Microscopical evidence of neuritis of the nerve-trunk was very distinct. In another case of tumour (glioma) of the frontal lobe, in which the neuritis was characteristic, with much swelling, the microscopic changes in the nerve-trunk, most intense behind the foramen, indicated a communicated descending neuritis, and old adhesions over the tumour showed that there had been local meningitis. It must be remembered that, in such cases, whatever mechanism leads to the occurrence of neuritis without meningitis may influence the degree and course of that which is excited by meningitis.

In most cases optic neuritis is a transient event in the history of a cerebral tumour, not a continuous accompani-

ment. A tumour may exist, and cause symptoms for a considerable time, without leading to any change in the eyes, and then optic neuritis may be developed, run its course, and pass away, in many cases leaving atrophy of the discs, while the symptoms of the tumour continue or increase for months or years. It is not only that a tumour takes time to cause optic neuritis, but it often exists for a considerable time before the mechanism for the production of neuritis, whatever that may be, is set in operation. A tumour may cause symptoms for years before optic neuritis is produced. The microscopical changes, shown in Fig. 22, p. 61, are from a case, under the care of Dr. Hughlings-Jackson, in which repeated examination during nine months showed the discs normal. Then neuritis came on, but soon subsided, and in six weeks the discs were again normal, and continued so till death. Dr. Jackson has recorded[1] a still more significant case, in which a man had had symptoms of cerebral tumour for nine years: during the last three years his discs had been repeatedly examined, but only six weeks before death, neuritis was first discovered. In many cases in which neuritis occurs long after the symptoms of tumour have existed, its occurrence precedes death by no long interval.

The appearance of the discs in intra-cranial tumour is that of neuritis in its most typical form, as described in a preceding page (p. 49). The neuritis may stop at any one of its stages (*see* p. 88), but, unless arrested by treatment, it usually progresses to a degree considerable enough to entail failure of sight and consecutive atrophy.

The neuritis of tumour is in most cases double, sometimes equally advanced in the two eyes, often more intense in one. Rarely it is unilateral, even though the tumour is such, in seat and nature, as commonly to cause double neuritis. In a few cases the neuritis has been on the side opposite to the tumour, but, as a rule, the eye on the same side is affected first,[2] and sometimes only,[3] and this seems true irrespective of

[1] "Med. Times and Gazette," Sept. 4, 1875.

[2] Hughlings-Jackson, "Ophth. Hosp. Rep.," 1871, and "Brit. Med. Journal," July 20, 1872; and Field, "Brain," July, 1881, p. 247.

[3] Greenfield, "Brit. Med. Journal," 1886, p. 317.

the seat of the growth. It must be remembered, as a source of possible fallacy, that the eye first affected may be in the course of subsidence, when the neuritis in the opposite eye is at its height. Thus one-sided preponderance, while not conclusive, affords a probability that the growth is on the same side. The probability seems great if the growth is in the front of the brain, but considerable even if it is subtentorial.[1]

Symptoms.—The symptoms of the neuritis which accompanies cerebral tumour have been fully described (p. 69). All symptoms may be absent, vision perfect in all respects, even with neuritis considerable in degree. But intense neuritis impairs sight, and the impairment increases, or may first occur, during its subsidence. These effects have been already considered. It must also be remembered that sight may be affected, not from the optic nerve, but from the intra-cranial disease.

Regarding the course of the neuritis in cerebral tumour, it is important to note that the neuritis often coincides at its onset with an obvious increase in the other symptoms of the cerebral tumour. This was pointed out, long ago, by Dr. Hughlings-Jackson. Instances of it are frequent, but at the same time exceptions are not rare. It is probably true, however, that *the development of optic neuritis always indicates progress in the morbid growth or its consequences.*

With regard to the course of the neuritis, it is necessary to distinguish two classes of cases. One of these is where the

[1] For some facts on this subject, *see* Gunn, "Brain," Autumn, 1898, p. 335. The usual correspondence in side, is the strong conviction of Sir Victor Horsley from his personal experience (oral communication). It is strongly supported by a collection of 47 cases at the Queen Square Hospital, in which the position of subtentorial disease, tumour or abscess, was ascertained by operation or autopsy. The facts have been taken from the case books by Dr. Holmes, R.M.O. Most of the cases were tumour of the cerebellum, a few were of abscess, and in a few cases the disease was in the pons. Of the 47 cases in which the observations were precise, the neuritis was equal on the two sides in 6; in 7 it was greater on the side opposite to the disease; it was greater (in a few earlier) on the same side of the disease, in 34, or 72 per cent. The measurement of the swelling was made by one of us (M.G.) in all these cases.

progress of the tumour, either spontaneously, or under the influence of treatment, becomes lessened or arrested after the onset of the neuritis; the other, where the progress of the tumour to which the neuritis is due is uninterrupted.

In the first event, the neuritis commonly subsides. It may pass away completely, even although it has reached the stage of considerable swelling with obscuration of disc and vessels, with distended veins and narrowed arteries, and sight may throughout be little impaired. This occurred, for instance, in the cases shown in Pl. II. 1, 2, 3, 4, III. 3. Less commonly, a slight or moderate damage to sight, from the inflammatory swelling and damage to nerve fibres, passes away; but often, while the neuritis subsides, amblyopia occurs or increases because the nerve fibres suffer compression from the contracting tissue. The last is the more likely to occur the longer the duration of the neuritis, because there is then more tissue formed, which can only undergo cicatricial contraction.

Instances of each course are seen in syphilitic tumours, and also when there is strong reason to believe that a scrofulous tumour exists—a cerebral or cerebellar tubercle. Subsidence of the neuritis may be the first sign of improvement. It was so in the case figured in Pl. III. 4, in which the neuritis passed away before there was any diminution in the symptoms, and then the paralysis slowly lessened, and improved up to a certain point, at which it became stationary; the tumour (probably tubercular) no doubt ceased to grow, and became smaller, permitting compressed tissue near it to recover, although destruction, which had before taken place, necessarily persisted. In syphilitic tumours, arrest can be obtained much more rapidly than in tubercular growths, and a considerable neuritis may pass away without damage to vision (Pl. II. 1 & 2, 3 & 4, IV. 4 & 5). In these cases, however, if a considerable neuritis exists before the treatment influences the tumour, tissue-changes too often progress in the disc to an extent which leads to loss of sight, even though ultimately the cerebral lesion becomes quiescent (Pl. IV. 5, 6). Thus complete and permanent blindness

may result from a syphilitic growth, discerned and treated too late. Occasionally, although very rarely, an analogous arrest seems to occur in other tumours, attended with degeneration and calcification, and the neuritis may subside with the change.

In the cases in which the tumour causing the optic change continues its growth, as do most tumours other than the tubercular and syphilitic growths, the course of the neuritis differs according to its intensity. When this reaches a considerable degree, signs of strangulation are developed, the swelling and hæmorrhage increase, and damage to the adjacent retina occurs. Then it subsides slowly to atrophy. The inflammation, as it were, terminates itself, but its consequences remain. When the neuritis does not reach a high degree, it has often a longer duration. The grey œdematous neuritis, with little sign of strangulation, may persist for months without much change, and then slowly subside; sight perhaps being little impaired until subsidence, and then not often in a grave degree. In a still slighter degree of neuritis the change may persist without alteration for a very long time.

There is at present little direct information regarding the conditions which determine the course and duration of neuritis in the cases in which the cerebral tumour continues its progress. But we have seen that the onset of neuritis may accompany an increase in other symptoms, and also that the early subsidence of neuritis may attend a diminution in the other effects of the tumour that may be taken as indicative of arrest. These facts taken together indicate that the course of the neuritis is, to some extent at least, dependent on, and influenced by, the course of the tumour. This conclusion is confirmed by the fact that in some cases of tumour of very chronic nature the course of the neuritis is equally chronic. The case mentioned above (Pl. III. 1 & 2) illustrates this, since the progress of the symptoms was very slight during the year and a half in which the neuritis was quite stationary. In the cases in which, without retrogression of the tumour, neuritis subsides, the affection of the optic nerve is probably due chiefly to excessive secondary

effects of the growth, such as disproportionate adjacent softening.

Significance.—The value of optic neuritis as an indication of the existence of an intra-cranial tumour is very great. Tumour is the cause of the majority of cases of pronounced neuritis due to intra-cranial disease. On the other hand, neuritis is present, at some period, in at least four-fifths of the cases of tumour, and it may be the only unequivocal sign of the organic intra-cranial disease.

It is important to remember that the neuritis is a transient condition, however long its duration, and that its effects persist after the inflammation has ceased. The atrophy left by neuritis may constitute unequivocal evidence of the antecedent inflammation, and where there is not actual atrophy, the aspect of the disc may show clearly that there has been previous neuritis. Yet it is not always possible, long afterwards, to say, from the aspect of the discs, how the atrophy originated. If the neuritis was moderate, a clean cut disc may be left, and the narrowing of the vessels may not be greater than is sometimes seen in cases of atrophy of other nature. The concealment of the lamina cribrosa is, however, usually complete, and there is frequently some disturbance of the adjacent choroid. Valuable information may also be gained from the circumstances under which the loss of sight came on, and the existence at the time of cerebral symptoms.

It is not only during life that neuritis may assist the diagnosis of tumour. As an instance, I may mention the case of a man with hemiplegia, in whose brain a soft area was found, limited and crossed by trabeculæ of firmer tissue. It was at first thought to be an area of old softening with some connective-tissue formation in and about it. Before it was examined with the microscope, the backs of the eyes were removed, and found to present distinct evidence of neuritis—swollen papillæ with hæmorrhages. The assumption that the disease was a growth was fully confirmed by the microscope.

From the facts given above it is evident that optic neuritis

may, in some cases, afford not only diagnostic, but prognostic indications. A subsidence of neuritis which has not reached any considerable degree of intensity, may be taken as indicating, in most cases, a retrogression of the growth, and a neuritis of very chronic course affords evidence that the progress of the tumour is and will be equally chronic. Yet it must be remembered that an acute neuritis may occur in the course of a chronic tumour, especially in the later stage. In such cases it often occurs not long before death, and therefore it affords some prognostic indication. In more acute cases, or in those in which it develops early, it has not the same significance. Other details regarding this effect of tumour will be found in the preceding chapter on Neuritis.

Simple atrophy of the optic nerves may result from intracranial tumour, but only by the mechanism of compression of the optic nerve of one eye or both. Thus, such atrophy results from pressure on the chiasma, or on one of the nerves in front of the chiasma. The pressure may be that of the growth itself, or (in the case of the chiasma) of a distended third ventricle. Compression of one tract seldom causes more alteration in the disc than is attended with slight pallor, and slight shrinking, in both eyes. Theoretically, pressure on both tracts should cause conspicuous atrophy, but no instance is known; perhaps life, in such a case, is not long enough to permit such visible change. The simple atrophy is thus "secondary" in nature. It is doubtful whether this form of atrophy ever results from the damage to the nerve by inflammation, such as may be produced by a secondary meningitis. In compression by a tumour, the tendency for some inflammation to spread down the nerve is such that the optic neuritis is usually to be seen in the eye. The visible inflammation is often slighter than the failure of sight, and the ultimate atrophy may be in part simple, in part papillitic, the degree in which each character preponderates varying in different cases. Great caution is therefore necessary in inferring, from the appearance of discs long after the onset of the

atrophy, that this was simple and not neuritic, and its significance. It must be remembered also that the chiasma may be compressed by ventricular effusion, or even by a fresh increase in the tumour, *after* neuritis of ordinary character and causation, and even after this has gone on to partial atrophy. Sight, damaged much or little by the neuritic process, may fail rapidly at a subsequent period from secondary pressure effects, without any corresponding change in the aspect of the discs. In a well-marked example of this, in which vision had become almost normal after neuritis from a syphilitic growth, sight failed concurrently with a subjective smell. The temporal half of the right field was lost first, and subsequently the temporal half of the left, with partial paralysis of the left third nerve, and subsequently the nasal half fields also failed. The discs, little changed by the neuritis, became atrophic, and the sense of smell entirely lost.

B.—*CYSTS.*

Cysts of various nature and seat are often attended by optic neuritis. They sometimes develop within a definite morbid growth, and in other cases neoplastic tissue may be found in some part of the cyst wall, as if the morbid growth had been arrested by the pressure of the liquid in the cyst. In other cases, the nature of the cyst is mysterious, but it is always of a size to have caused considerable increase of the intra-cranial pressure. The mechanism of this is usually direct pressure, but it may be ventricular distension, as in a case, with optic neuritis, in which a small cyst of the choroid plexus had obstructed the passage to the fourth ventricle, and had caused great distension of the lateral ventricles.[1] When a cyst is a local change in part of a definite morbid growth, the character of the optic neuritis depends, in general, on the nature of the tumour. In other cases the neuritis is usually slow in development and course, and moderate in degree, the swelling being seldom great, and hæmorrhages few or absent.

[1] Collier and Batten, "Brain," vol. xxii. p. 532.

HYDATID CYSTS.—*Associated Changes.*—A cysticercus has been occasionally observed in the vitreous humour, but the coincidence of a parasite in the eye with symptoms of cerebral tumour due to another in the brain, has not, I believe, hitherto been recorded.

Consecutive Changes.—Optic neuritis is met with in cases of hydatid disease of the brain, and has all the characters of the neuritis which is caused by growths. It has been observed as a result of hydatid cyst in both cerebrum and cerebellum. It may go on to consecutive atrophy, life being prolonged for years.

LABIO-GLOSSAL PARALYSIS.

In chronic bulbar paralysis, due to degeneration, ophthalmoscopic changes are extremely rare. Unilateral atrophy was once seen by Galezowski, and Robin quotes a case of rapid atrophy of both nerves in the course of the affection in a man aged sixty-seven, which was exceptional in some later return of vision. Atrophy may accompany almost any degeneration of the nervous system.

INTRA-CRANIAL ANEURISM.

Miliary aneurisms have been spoken of in connection with cerebral hæmorrhage. Intra-cranial aneurisms of larger size are not, as a rule, accompanied by any *associated* ocular changes; those of the central artery of the retina are too rare to be of significance, and only met with in degeneration of the arteries. Nor do aneurisms often cause *consecutive* changes, unless their position is such as to press upon the optic nerve (causing unilateral amaurosis and secondary atrophy), on the chiasma (lateral) or, very rarely, on the optic tract (temporal hemianopia). An aneurism of the internal carotid may obstruct the cavernous sinus, and cause transient distension of the retinal veins, without papillary changes, but the pressure is relieved by the free communication of the ophthalmic and facial veins;

the enlarged angular vein may be conspicuous beneath the skin. In rare cases, however, an aneurism in this situation has led to optic neuritis, as in a case recorded by Michel;[1] double neuritis, with evidence of obstruction, was the first sign of a cirsoid aneurism of the two internal carotids. It pressed on the optic nerves, which showed evidence of interstitial inflammation. Holmes of Chicago has recorded several cases in which optic neuritis co-existed with intra-cranial bruit, and in the only one on which a post-mortem was obtained, an aneurism of the internal carotid was found, but there was also an adjacent growth in the pituitary body.

In an interesting case[2] (by Jeaffreson of Newcastle-on-Tyne), although there was no post-mortem examination, an aneurism of the internal carotid was probable. There was unilateral papillitis, and a loud intra-cranial murmur could be arrested by compression of the carotid; there was also paralysis of the third nerve, and subsequently aphasia.

The origin of the papillitis in these cases is probably a descending inflammation, extending to the nerve from that which always exists around an aneurism. That the neuritis is not usually the direct effect of compression of the cavernous sinus is shown by the fact that aneurisms which produce the same effect on the sinus have often not been accompanied by papillitis. Moreover, when there is papillitis, the enlarged communications with the facial vein may (as in Jeaffreson's case) afford the same evidence of relief to mechanical obstruction as in cases when it is absent.

Internal Hydrocephalus.

Simple internal hydrocephalus, without a growth, is not at first attended by ophthalmoscopic changes unless the state is due to inherited syphilis. They may be absent throughout, even though the distension of the ventricles is such as to cause a marked increase in the size of the head. Some-

[1] "Arch. f. Ophth.," xxxiii. 2, p. 225.
[2] "The Lancet," March 8, 1879.

times there is slight fulness of the retinal veins. Sight often fails at a later period, and in some cases early, and the signs of simple atrophy of the optic nerve are then present. In several cases the onset of the atrophy has been watched, and the occurrence of any neuritic process excluded. In a few cases the atrophy has been preceded by signs of neuritis similar to that seen in tumour; it is usually slight in degree, but sometimes considerable.[1]

The simple atrophy of the nerves is usually due to the pressure of the distended third ventricle on the optic chiasma. In one adult case, mentioned by Förster, the distended ventricle appeared at the base of the brain as a bladder measuring ten lines by eight.

It has been said by Bouchut that the ophthalmoscopic changes may serve to distinguish chronic hydrocephalus from the large head of rickets; but, owing to the lateness of the optic changes, the cases must be very rare in which the nature of the disease is not distinct long before ophthalmoscopic signs are present.

DISEASES OF THE MEMBRANES OF THE BRAIN.

Meningeal Growths.

Tumours springing from the pia mater always damage the cerebral substance to a greater or less extent either by invasion or compression, and their effects, for the most part, have been included in the account of the cerebral tumours.

Tumours springing from the dura mater differ in their effects according to two characteristics—first, their tendency to invade; secondly, their position, whether at the base of the brain or on the convexity.

Growths springing from the dura mater at the base cause optic neuritis much more frequently than those of the cortex.

[1] Wildbrand, "Centralbl. f. med. Wiss.," 1879, p. 923.

When in the front of the base, the inflammation around the growth may extend directly to the nerve. When more distant, as in the posterior fossa, optic neuritis is still a frequent consequence and may be intense. Those that invade the brain have the same tendency to cause optic neuritis as tumours beginning in the brain substance. The growths that merely compress and do not invade, have this tendency in far slighter degree, and it is less the slower the growth of the tumour. The more rapidly the pressure is induced, the greater and more acute is the secondary inflammatory process which pressure causes, in proportion to its rapidity, manifested by the softening. With very slowly growing tumours such softening may be entirely absent, and the tendency to the occurrence of optic neuritis is much slighter. I have already mentioned a tumour the size of the closed fist, which had compressed one hemisphere behind the mid-parietal region, so as to produce a depression corresponding in size to the growth; there was no optic neuritis up to the end, and no sign that optic neuritis had ever existed. Hence the absence of neuritis is evidence of some value that a tumour at the surface of the brain springs from the membranes and does not invade. Cases are on record, however, in which optic neuritis was for a long time the only symptom of such a growth; in one case, after the neuritis had existed for months, hemiplegia came on, and was found to be due to a sarcoma springing from the periosteal dura mater, and which had compressed the left hemisphere of the cerebellum and the left side of the pons Varolii.[1] In the case figured in Pl. III. 5, optic neuritis, although not the earliest symptom, reached its height before any motor paralysis occurred. The tumour sprang from the dura mater, and had compressed the right side of the pons and right hemisphere of the cerebellum.

In some of these cases secondary meningitis may be traced along the base of the brain. Such inflammation is produced by meningeal growths even more frequently than it is by

[1] Pagenstecher and Genth's "Atlas of the Path. Anat. of the Eyeball," Pl. xxxiv. Fig. 3.

tumours in the substance of the brain, and it may play some part in the production of the changes in the eye.

Meningitis.

The effects of meningitis on the eye vary much according to its seat, being slight and late when the inflammation is at the convexity of the hemisphere, considerable and early when the meningitis is at the base. In some cases, especially of the former class, ophthalmoscopic changes are entirely absent, and when present they attend the stage of developed inflammation rather than the initial vascular disturbance. They thus afford, as Manz and others have pointed out, little support to the doctrine that the intra-ocular circulation shares and reveals intra-cranial vascular disturbance.

Simple and Septic Meningitis.—Acute simple meningitis of the convexity is usually unaccompanied by ophthalmoscopic changes; only when it has lasted for a considerable time is neuritis sometimes developed.

Chronic simple meningitis of the convexity, slight in degree (such as that of which traces are often found in the brains of drunkards), is also commonly unattended by any optic change. The slight œdema and congestion of the disc, sometimes seen in chronic alcoholism, may be the direct result of the toxæmic condition.

Simple meningitis of the base (apart from the posterior form) is rare except from tubercle or syphilis, or in association with tumour or some bone disease. Optic neuritis may occur by direct propagation, and in those cases in which the disease is chronic, the visible changes in the disc may be considerable in degree and duration. Basilar meningitis is, however, in most cases tubercular or syphilitic. In all forms, neuritis is common and of great diagnostic importance.

Posterior Basal Meningitis, the peculiar chronic form that occurs in children, is not often attended by optic neuritis. It was found only in three out of forty-two cases (Barlow

and Lees [1]). This form has little invasive tendency, and to this the rarity of neuritis may be due. But neuritis may also, sometimes, be a coincident effect of a specific cause of the meningitis, since an organism, similar to that of cerebro-spinal meningitis, has been found in the membranes by Still.[2]

Septic Meningitis, whether due to local disease or to a distant cause, is usually attended with optic neuritis if the duration of life is sufficient to permit its production. In a case of purulent meningitis, suppurative inflammation of the eye (chemosis and post-mortem infiltration of the retina with pus) was observed by Berthold,[3] but was probably coincident. Leube[4] has recorded a case of purulent meningitis of the convexity, secondary to septicæmia, in which there was intense inflammation of the optic nerve in front of the commissure. The only changes in the eye were distension of the retinal veins and hæmorrhages. I have seen well-marked neuritis in a case of septic meningitis (post-puerperal) with grave cerebral symptoms. The patient recovered.

In association with subacute septic meningitis in children, and such as may arise by extension from ear disease, a peculiar form of slow inflammation of most of the structures of the eyeball has been described by Nettleship[5] and others under the name of "pseudo-glioma." It is probably due to direct extension by the optic nerve and sheath, such as seems the mode by which the neuritis is produced. In one case, due to purulent disease of the middle ear, there was optic neuritis only in one eye, while the other was the seat of this peculiar ophthalmitis.

Tubercular Meningitis : *Associated Condition.*—Tubercles of the choroid may now and then be found in tubercular meningitis, and furnish valuable diagnostic information. But they are less frequent, as Cohnheim pointed out, in

[1] Art., Clifford Allbutt's "System of Med."

[2] "J. of Path. and Bact.," 1898.

[3] "Arch. f. Ophth.," Bd. xvii. 1874.

[4] "Deut. Arch. f. Klin. Med.," 1878, xxii. 263.

[5] "Trans. Oph. Soc.," vol. iii. *See* also Flemming, *ib.* 1900, Stephenson, *ib.*, and T. Collins, "R.L. Oph. Hosp. Rep.," 1892.

tubercular meningitis than in general tuberculosis without meningitis. Heinzel[1] never saw them in forty-one cases of tubercular meningitis which he examined with the ophthalmoscope, and the case figured (Fig. 49) was the sole instance in which they were found in twenty-six cases repeatedly examined by Garlick at the Hospital for Sick Children.[2] Some cases in which neuritis due to meningitis co-existed with tubercles of the choroid have been collected by Brückner.[3]

Consecutive Changes.—A peculiar marbled reflection from the retina has been described by Leber and Hock, particularly in the neighbourhood of the veins, in conjunction with tubercles of the choroid. Its pathological nature is very doubtful.[4] He associates it with œdema.

Changes in the optic discs of more considerable degree are present in tubercular meningitis so often as to make them an important symptom of the disease. Their frequency has been variously stated. The discs may be normal throughout in the rare cases in which the tubercular inflammation is confined to the convexity of the brain. In some cases of basal meningitis, also, changes are entirely absent. Garlick,[5] of twenty-six cases, found the discs normal throughout in five; distinct swelling was developed in about half the whole number, increased redness only in one quarter, and in a few others only distension of veins. In many of these cases, however, the changes were slight, and their pathological character was recognizable only by their development under observation. It is probable, then, that considerable changes are present in one-half the cases, and that in two-thirds of the remainder slight alterations will be found, if the discs are watched with care from day to day. The occurrence of congestion and œdema of the disc seems to be especially related to the occurrence of inflamma-

[1] "Jahrbuch für Kinderheilkunde," 1875, p. 334.

[2] *See* p. 168.

[3] "Arch. f. Ophthal.," vol. xxvi. pt. 3, 1880, p. 154.

[4] A condition very like it is met with apart from disease, and often called the "watered-silk retina." For an explanation of this appearance, *see* Gunn, "Ophth. Hosp. Rep.," vol. xi. p. 348.

[5] "Med.-Chir. Trans.," 1879, p. 441.

tion, and formation of lymph, in the anterior part of the base, about the chiasma and the optic nerves.

The degree of change is rarely great. The disc becomes full-coloured, and its outlines hazy. Sometimes this and distended veins constitute the only morbid appearance. More often swelling, with undue striation, becomes visible on direct examination, and the edges of the disc gradually cease to be recognizable. The disc has often a reddish-grey aspect. In some early cases the colour of the swollen papillæ is paler, especially on examination by the indirect method, than in the early stage of the acute neuritis of cerebral tumour. The neuritis rarely attains a more intense degree, perhaps because life only lasts sufficiently long in cases in which the inflammation is not intense. The veins are often, though not always, over-distended from the first. In Garlick's careful observations their distension was especially related to excess of subarachnoid fluid; when the quantity of this was normal, there was also no distension of the sheath of the nerve—a fact of much importance. Occasionally white lines along the sides of the vessels are unduly conspicuous. Hæmorrhages are rare.

Sometimes white spots are seen in the neighbourhood of the swollen disc. They are in the substance of the retina, and consist of an accumulation of lymphoid corpuscles in the nuclear and molecular layers, or of degeneration of nerve-fibres. They may readily be mistaken for tubercles of the choroid. It has been thought that they are of the nature of tubercles, and they have accordingly been described as retinal tubercles, but very similar spots are seen in papillitis from other causes.

The changes are always double, though they may begin, and be more advanced, on one side. The excess is generally (but not always) on the side of the chief cerebral change. In most cases the patients have died not long after its development, and sight has suffered little. In the rare cases that recover the inflammation does not become intense, but it is of extreme diagnostic importance. As the cerebral symptoms subside, the neuritis passes away, and sight is preserved or

restored. This has been pointed out by Clifford Allbutt, and two probable instances are described by Garlick. The symptoms were headache, vomiting, constipation, irregular pulse, normal temperature, and the development of ophthalmoscopic changes under observation. In both cases recovery was complete. I have also seen a few cases of recovery from characteristic symptoms, in which the presence of distinct optic neuritis made the existence of meningitis certain, and its tubercular nature was highly probable. Most were in adults.[1]

Cases of .optic nerve atrophy of old-standing are occasionally seen in which sight was lost in early life with acute cerebral symptoms very like those of an attack of tubercular meningitis.[2] In some of the cases of recovery from supposed tubercular meningitis with ophthalmoscopic changes, the symptoms may possibly have been due to a tubercular mass in the brain. The symptoms of such a tumour more often pass away than those of tubercular meningitis.

The neuritis of tubercular meningitis was regarded by v. Graefe as descending neuritis, the inflammation passing directly from the membranes to the optic nerves. This is probable; the existence of inflammation in the trunk of the nerve may be obvious to naked-eye examination. The nerve is swollen, softened, and reddened, and the descent of neuritis may be demonstrated by microscopical examination. Distension of the sheath often, but not always, coincides, and it usually presents, under the microscope, evidence of inflammation. Moreover, Michel,[3] in a case in which there was a cloudy halo around the papilla, found not only effusion into the sheath, but numerous miliary tubercles in both the dural sheath and pial tissue.

In many cases the symptoms of meningitis are distinct

[1] A strong tubercular history and the basal seat of the inflammation, made the diagnosis almost certain. I have not seen tubercles of the choroid in a case that recovered.

[2] Hutchinson, "Ophth. Hosp. Rep.," v. 310 and ix. 124.

[3] "Deutsch. Archiv. f. Klin. Med." xxxii. p. 439.

before the ocular changes are developed. In such cases the ophthalmoscope confirms rather than assists the diagnosis. But in some cases the cerebral symptoms are latent or dubious, and in these the examination of the eyes may afford valuable help, and it is probable that it would do so in at least one-third of the cases. Of the twenty-six cases watched by Garlick, the ophthalmoscope was of real diagnostic assistance in six, and would doubtless have been so in a larger number had earlier examination been practicable. In one case, which lasted twenty-six days, the other symptoms were indefinite until the nineteenth day, but on the fourteenth day the ophthalmoscopic changes were so unmistakable that the diagnosis of meningitis was confidently made. In another case, ophthalmoscopic changes were distinct on the ninth day; other symptoms were conclusive only on the fifteenth day, the patient dying on the twentieth day. In both cases, the morbid process about the optic commissure was very marked.

During the course of meningitis a diminution of the cerebral symptoms may be accompanied by a corresponding lessening of the intra-ocular changes.

Syphilitic Meningitis.—Syphilitic meningitis (1) may be associated with the ocular signs of syphilis, and (2) may cause optic neuritis. When at the base, the ophthalmoscopic signs are similar to those of tubercular meningitis, but more chronic in course and more considerable in degree. The greater degree depends on the longer time the morbid process usually exerts its influence. When localized in the convexity, ocular symptoms are usually absent in the early stage, and may be throughout. If the case is not treated and local chronic meningitis persists, the disc may present neuritis similar to that which is seen in cerebral tumour. But the diagnosis from a gumma is never possible, and a growth cannot be excluded if focal symptoms exist. Moreover, the degree of gummatous tissue present in local inflammation is sometimes such as to make the pathological discrimination far from easy.

Hæmorrhagic Pachymeningitis (Hæmatoma of the Dura Mater).—According to Fürstner,[1] there may be mechanical congestion of the retinal veins, and even papillitis, accompanied by distension of the optic sheath with dark-coloured fluid.

Cerebro-Spinal Meningitis.—In epidemic cerebro-spinal meningitis, optic neuritis may occur, but is rare. Schirmer found it in one only of twenty-seven cases examined. Inflammation of all the structures of one eyeball occurred in a case described by Stephenson[2] in which the inflamed membranes presented the meningococcus described by Weichselbaum as the specific cause of cerebro-spinal meningitis. Cyclitis and retinitis were found by Oeller.[3] Many of the retinal veins contained thrombi and granular plugs; no direct connection with the intra-cranial process could be traced. A purulent irido-choroiditis is the most frequent eye change in this disease.

In the sporadic form of cerebro-spinal meningitis, optic neuritis may occur, and may lead to atrophy. A lad, after a severe wetting, suffered from intense headache, delirium, fever, and retraction of the head. Sight failed ten days after the onset. The symptoms subsided at the end of six weeks, but he remained blind, and six months later there was slight perception of light in one eye only. The optic discs had all the appearance of consecutive atrophy, the centres were filled in with new tissue, the vessels narrowed, and the adjacent choroid disturbed.

Traumatic Meningitis often causes ophthalmoscopic changes, of which an instance is shown in Pl. I. 5, a case in which fever, delirium, and convulsions succeeded a fall on the head. The neuritis subsided with the symptoms. These cases are considered in the section—"Injuries to the Head." Ophthalmoscopic changes are frequent, and are important in the many cases in which other symptoms are subjective only, and when the grave nature of the

[1] "Arch. f. Psychiatrie," vol. viii. pt. 1.
[2] "Trans. Oph. Soc.," xx. 1900, p. 121.
[3] "Arch. f. Augenkrank.," vol. viii. 1878, p. 357.

effects of the injury may be doubted or even denied by those whose interests are concerned, as in railway cases.

Diseases of the Cranial Bones.

Caries.—In caries of the sphenoid bone, or suppuration beneath the periosteum, the inflammation may extend to the optic nerve, damaging it, and causing secondary atrophy, or, descending the nerve, may produce intra-ocular neuritis. The disc shown in Pl. I. 2 is an illustration of this effect. The case was one of caries of the body of the sphenoid bone in a girl aged sixteen. There was well-marked neuritis in the left eye, but for a month afterwards the right eye was normal. Coincidently with an increase of the symptoms of meningitis, this also became inflamed, and she died a few days later. The autopsy showed caries of the sphenoid, chronic meningitis around the left sphenoidal fissure, involving the sheath of the left optic nerve. There was also general acute purulent meningitis, which had, no doubt, been the cause of the neuritis in the right eye. The damage to the left nerve was just in front of the chiasma. When the drawing was made, the neuritis was confined to the left eye. In this case there was no change in the sheath of the nerve. In a case recorded by Horner, of caries of the sphenoid, the sheath of the optic nerve was distended by purulent material as far as the eyeball.

Caries of the bone, at a distance from the optic nerves, does not cause ophthalmoscopic changes unless it excites meningitis or cerebral abscess. To this, however, an exception must be made in regard to disease of the bones of the ear, which there is reason to believe may cause optic neuritis when no abscess or meningitis is to be found. It has been suggested by Barker that the papillitis in these cases may be the result of a septic inflammation in the middle ear, infecting directly the adjacent carotid canal, and extending along the lymphatics of the latter to the sheath of the optic nerve. Cases of neuritis in consequence only of middle ear disease, associated with optic neuritis, with no intra-cranial link,

seem unquestionable. They are important, and deserve careful study by the pathologist.

THICKENING OF THE CRANIAL BONES.—General thickening of the cranial bones may be attended by optic neuritis and consecutive atrophy. Michel has recorded the case of a boy who was blinded by neuritis and consecutive atrophy early in life, and who died at fifteen. The necropsy revealed great hyperostosis of the bones of the skull, by which both optic foramina were considerably narrowed. The optic nerves were atrophied from the chiasma to the eye, but the orbital portion was greatly thickened by hyperplasia of the cellular tissue in the subvaginal space. A similar case has been described by Manz, in which the tissue between the sheath and the nerve had a semi-gelatinous aspect. Michel explains this change, by assuming that the narrowing of the foramen leads to retention within the sheath of lymphatic fluids, which cause irritative over-growth.

In other cases, similar conditions of bone, exostoses, &c., narrowing the optic foramen, have caused only simple atrophy of the optic nerve.

DISEASES OF THE ORBIT.

Inflammatory Processes in the Orbit, e.g., cellulitis (as in facial erysipelas), inflammation at the back of the orbit, or periosteal affections in which the symptoms and their course point clearly to the seat and nature of the lesion (although the pathological inference is still unconfirmed by *post-mortem* evidence), frequently damage the optic nerve. This damage always involves inflammation, which may or may not descend, and be visible in the papilla. The difference depends on the position of the chief inflammation, *i.e.*, on its distance from the eye, and also on its tendency to spread. Whether there is papillitis or not, atrophy is subsequently visible, proportioned in degree to the impairment of sight. It usually has the aspect of "secondary atrophy," already described; signs of preceding inflammation are usually

slight. The affection of sight is due chiefly, not to the visible inflammation, but to the changes behind the eye. Hence care must be taken not to regard the visible papillitis as the chief cause of the failure of sight or to form a forecast from its degree. Sight may be lost from compression of the nerve, which causes no visible effect, except slow secondary atrophy of the disc, sometimes with narrowing of the vessels. When inflammation is communicated to the nerve, and descends to the eye, there may be visible papillitis. In many cases the eyeball becomes prominent, usually only in slight degree. The nerves to the ocular muscles may be gravely damaged when the general orbital inflammation is slight, and when the optic nerve suffers little or not at all.

In one form,[1] loss of sight of one eye comes on simultaneously with paralysis of all the ocular muscles, sometimes with tenderness on pressing the eyeball back. The symptoms have been ascribed to hæmorrhage and to orbital cellulitis. This is certainly an occasional cause, and seems generally to be rheumatic in nature. In one case the symptoms came on with much pain, after exposure to cold, in an intensely rheumatic woman, who had previously had an attack of paralysis of the facial nerve. There was palsy of all the ocular muscles and complete loss of sight. The former slowly passed away, but the blindness endured, and the disc slowly passed into atrophy without the least sign of neuritis. In such a case it is probable that the nerve suffered chiefly from pressure. Similar cases have been met with, in which the affection of the optic nerve has been less, and some recovery has ultimately occurred.

In a case, probably of syphilitic inflammation at the back of the orbit, with intense pain in the eye, orbit, and head, the inflammation descended to the eye, and produced secondary papillitis, ending in atrophy. In this case sight was lost, and the vision of the other eye also became impaired. Hence it is possible that the inflammation extended from one optic

[1] For example, cases recorded by v. Graefe, "Arch. f. Ophth.," vol. i. pt. 1, p. 424, and Baumeister, ibid. vol. xix. pt. 2, p. 264.

nerve to the other, perhaps by the chiasma—or the primary lesion may have been situated in the sphenoidal sinus between the two optic canals.

A very similar state of secondary atrophy of the nerve may result from a blow on the head,[1] or on the eye. Rapid exophthalmos and the appearance of the lids may show that hæmorrhage has occurred into the orbit. (*See* "Injuries to the Head.")

In rare instances, hæmorrhage has occurred, apart from injury, in sufficient quantity to cause prominence of the eyeball, and distension of the eyelids with blood. The exciting cause may be some violent effort. The degree of impairment of sight appears to depend upon the amount of blood effused, and the consequent damage to the optic nerve.

Tumours in the Orbit.—A tumour at the back of the orbit or of the optic nerve, may cause neuritis such as results from intra-cranial tumour, but this is at first limited to the eye in front of the growth; the other optic papilla either escapes or presents only a slighter and later inflammation, communicated to the nerve. There is also distinct and increasing prominence of the eyeball. A simple atrophy of the optic disc is, however, more common as the result of orbital tumour, which often simply compresses the nerve.

Injuries to the Head.

Injuries to the head, blows, falls, &c., frequently cause ocular symptoms and often very marked ophthalmoscopic signs. The forms of ocular affection are of several varieties.

1. Impairment or loss of sight, without ophthalmoscopic changes, or with very slight alterations—simple congestion of the disc, easily overlooked. Such effects may result from blows on the anterior portion of the head. In some cases the mischief is probably direct concussion of the retina, for in slight cases an alteration of vision has been noted, such as must be ascribed to disturbance of the retinal elements. For instance, in a case recorded by Gosetti, after a blow on

[1] Snell: "Ophth, Rev.," i. 402.

one angle of the orbit, near objects appeared unduly large, and there was some colour-blindness, but no ophthalmoscopic change.

2. Optic neuritis has followed injuries to the head in many cases, at an interval of a few days or weeks. It is apparently due to secondary results of injury, especially to meningitis (Pl. I. 5), less commonly to traumatic inflammation of the brain or hernia cerebri. In one case, there was a compound depressed fracture of the left parietal bone. This was elevated five weeks after the injury, but a few days later hernia cerebri occurred. The optic discs were then normal, but five days later there was acute optic neuritis, which persisted until death. In another group of cases optic neuritis develops several weeks or months after the injury, and is then generally the consequence of an abscess of the brain, of which it may be the chief manifestation. (*See* "Abscess of Brain.") In such cases the neuritis may be slight, or so considerable as to entail loss of sight and consecutive atrophy.

3. Simple atrophy, unilateral or bilateral, may result from injuries which damage the optic nerves, directly or by pressure from secondary inflammation. An example of this was presented by a patient in whom a fall on the right side of the head and shoulder, injuring the circumflex nerve, was followed by slow grey atrophy of the right optic disc. In such cases sight may fail some time before the ophthalmoscopic signs of atrophy are apparent. (*See* p. 175, "Fracture of the Skull.")

4. In rare cases an injury to the head may be followed by gradual failure of sight, with very slight and stationary papillitis. In such cases it is probable that a chronic interstitial neuritis has been set up in the nerve trunk.

Concussion of the Brain is attended by no ophthalmoscopic change. Simple concussion of the nerve and retina may possibly, as just stated, cause loss of sight and slow atrophy, but usually the affection of vision is only temporary.

Contusion and Laceration of the Brain may entail optic neuritis, commonly slight in degree, although sometimes

very definite, with some opacity of the adjacent retina. It is apparently due, in some cases, to a secondary meningitis, but may be a direct result. It sometimes constitutes a valuable indication of the occurrence of greater mischief than a mere concussion. In one case,[1] the symptoms of concussion were followed by neuritis and consecutive atrophy, and ten weeks after the injury the necropsy showed two foci of red softening in the right anterior lobe and one in the corpus callosum.

Fracture of the Skull[2] may cause loss of sight in consequence of laceration of the optic nerve. According to the statistics of Hölder, quoted by Berlin, the orbital vault is involved in 90 per cent. of fractures of the base of the skull (80 out of 88 cases), and the optic canal was implicated in 54 (or 60 per cent.) In 42 of these there was hæmorrhage into the sheath of the optic nerve. The most frequent causes are blows, and falls on the frontal bone, less frequently on the temporal or occipital bone. The effect of the laceration of the nerve is usually immediate and permanent loss of sight. It is generally unilateral and on the side of the injury, very rarely on the opposite side; in a case recorded by Leber and Deutschmann, the eye blinded was on the side opposite to the ear from which blood escaped. Both eyes are only affected when both optic canals are fractured. Sometimes the hæmorrhage into the orbit is shown by prominence of the eyeball and effusion of blood into the eyelids. The optic nerve may be torn, compressed, or stretched. Loss of sight, if absolute, is usually permanent. When the lesion, as is commonly the case, is behind the place of entrance of the central vessels, there is at first no ophthalmoscopic change, or only transient hyperæmia, but atrophy gradually sets in. The pallor has been observed to commence three weeks after the injury. The ultimate appearance of the disc is

[1] "L'Union Méd.," 1865, ii. 3, No. 63.

[2] The statements in the text are, in part, derived from important papers by Berlin ("Heidelberg. Ophth. Gesellsch.," 1879, and "Annales d'Oculistique," vol. lxxxiii. 1880, p. 69), and by Leber and Deutschmann, "Arch. f. Ophth.," vol. xxvii. pt. 274. *See* also Graefe and Saemisch's "Handbuch," vol. v. p. 219.

usually that of simple atrophy, the edges sharp, and the vessels of normal size. Sometimes narrowing of the vessels has been observed, and has been ascribed to the extension of inflammation to the tissue around the vessels, or to their direct compression. Ophthalmoscopic signs of inflammation are not common, except as a result of subsequent meningitis, but I have seen œdema of the disc with retinal hæmorrhages, from effusion of blood into the optic sheath. If the injury to the nerve is in front of the place of entrance of the central artery, the appearances are similar to those of embolism. When the injury to the nerve is partial, the loss of sight may be incomplete, and in such cases changes in the field are common. When sight is impaired only by effusion of blood into the sheath, the prognosis is said to be better than when the nerve is injured. Occasionally signs of direct injury to the eye have been observed in these cases, rupture of the choroid, or vitreal opacities.

Compression of the Brain may, it is said, be attended by changes in the fundus oculi—distension of the veins, congestion and œdema of the papilla, but such appearances are rare.

Traumatic Meningitis may entail ophthalmoscopic changes similar, for the most part, to those which are seen in tubercular meningitis. Meningitis often results from fracture of the base of the skull, and may also be attended with neuritis. An instance of traumatic mischief with neuritis is afforded by the case figured in Pl. I. 5. The neuritis came on with mental disturbance and convulsions, following, at an interval of a week, a fall on the head. The change was slight in degree, although very distinct, and passed away soon after the cerebral symptoms subsided, leaving no trace. When the neuritis is more intense, blindness may result. In a child who had symptoms of meningitis five months after a fall on the head,[1] optic neuritis (" descending ") was found with the ophthalmoscope, sight being little impaired. Four years later, however, the child was healthy but blind,

[1] Hock, " Oest. Jahrb. für Pädiatrik," vol. v. 1874, p. 1. " Nagel's Jahrb. f. Ophth.," vol. v. p. 427.

with atrophy of both optic nerves. In other cases of the kind, actual meningitis has been found. The neuritis may be associated with the signs of mischief at the base of the brain, paralysis of ocular muscles, &c. The chronic inflammatory consequences of an injury (chronic meningitis, inflammatory "growths," &c.) may persist, and progress for a long time, even for years. In one case meningeal growths, apparently the result of chronic inflammation, were found beneath two old fractures of the skull, the result of an injury received several years previously. At the base, meningitis had damaged the arteries and caused softening, but no recent process was found. It must be remembered that an injury may be the excitant of tubercular and other growths in predisposed subjects.

Hernia Cerebri, resulting from fracture of the skull, with loss of bone, may be accompanied with neuritis. The mechanism is uncertain.

Necrosis of the Cranial Bones.—The damage to bone by injury may cause necrosis and meningitis, or abscess of the brain, either of which may entail inflammation of the optic nerve. As Hughlings-Jackson has pointed out, the relation of the symptoms to the injury may be obscure and unsuspected by the patient or friends, so that careful attention should be paid to any sign of injury, such as puffy swelling, &c., and a blow or fall should be carefully inquired for. Syphilitic disease of the cranial bones only causes changes when it is adjacent to the optic nerve, or leads to secondary meningitis.

Diseases of the Nose.

Some curious cases have been recorded[1] in which optic neuritis coincided with persistent discharge of watery fluid from one nostril. In most cases there were chronic cerebral symptoms; in some there were polypoid growths in the nose.

[1] *See* Nettleship and Priestley Smith: "Oph. Rev.," 1883, pp. 1, 4, and Emrys Jones: "Oph. Rev.," vii. 97.

Not infrequently the fluid is cerebro-spinal; the most probable explanation is that there is increased intra-cranial pressure and sometimes hydrocephalus, and that the escape of fluid relieves the pressure indirectly. In a case recorded by Baxter[1] the bones of the skull were abnormally thick. St. Clair Thomson[2] has recently given a valuable historical and clinical account of cases of this nature. The mechanism by which the optic neuritis is produced is not known.

Insolation and Heatstroke.

The occurrence of congestion of the optic discs (p. 44), in cases of severe sunstroke, has been described by Macnamara. In America, according to Hotz,[3] it is not uncommon to meet with cases of atrophy of the optic nerves, which are ascribed by the patients to sunstroke. Commonly the arteries are narrowed, as if from preceding inflammation, and in some recent cases he met with actual neuritis. In three the exposure had been to the sun, in three to an intense heat. Severe headache was a prominent early symptom, and it is probable that the neuritis was secondary to cerebral or meningeal inflammation.

Hotz has also seen exudative choroiditis apparently from the same cause, in degree sufficient to cause detachment of the retina. He regards it as due to the extension of inflammation along the sheath of the optic nerve, but the absence of choroiditis in other cases of such extension renders the explanation difficult to accept.

DISEASES OF THE SPINAL CORD.

Inflammation.

Spinal meningitis may be accompanied by ophthalmoscopic changes when the cerebral membranes are also

[1] "Brain," v. 325.

[2] "The Cerebro-Spinal Fluid: its Spontaneous Escape from the Nose." Cassell & Co., 1899.

[3] "American Journal of Medical Science," July, 1879.

affected (*see* "Cerebro-Spinal Meningitis." But neuritis may coincide with any local inflammation that is produced by a blood-state, and the states in which the papilla becomes inflamed are various.

Myelitis is usually unattended by any eye changes, but optic neuritis has been observed to coincide with the inflammation of the spinal cord, transverse or disseminated, to precede it, or to follow it. In most cases it has probably been a coincident effect of the blood-state which caused the myelitis.

Seguin[1] has twice seen transient optic neuritis, coincident with subacute transverse myelitis.

Two cases have been recorded by Dreschfeld. In one, a man of 45, the myelitis, fatal in a month, came on in the course of intense optic neuritis, accompanied by slight mental disturbance. The necropsy revealed disseminated acute inflammation of the spinal cord in the dorsal and lumbar regions. The brain appeared healthy. In the other case, that of a woman aged thirty-eight, who died from respiratory paralysis six weeks after the onset of symptoms of acute myelitis, the double optic neuritis was found soon after the paralysis came on. The brain presented merely signs of congestion, but the upper part of the spinal cord was softened.

Optic neuritis has been thought to have a special relation to myelitis, or other disease, in the cervical or upper dorsal region. Taylor and Collier[2] have recorded a series of cases coincident with spinal cord disease, some of which, they think, support this conclusion, but on examination of the facts, the evidence they afford is scarcely conclusive. In most of the cases, a common cause, or coincidence from simultaneous intra-cranial disease, cannot be excluded.

In some cases (Noyes, Steffen) there has been evidence that the optic neuritis was more intense in the trunk of the nerve than within the eye, and even greatest at the chiasma.

[1] "Journal of Nervous and Mental Diseases," April 1880. *See* also Noyes.

[2] "Brain," Winter 1901.

This also supports the view that, in most cases, the optic neuritis and myelitis have been the result of a common cause. It may be noted that we are led to the same conclusion regarding the atrophy of the optic nerve which is sometimes associated with chronic disease. It coincides with the spinal mischief, but is not its result.

Degeneration.

Tabes: Locomotor Ataxy.

Optic neuritis has been found, very rarely, in cases of tabes.[1] In the few cases in which its origin could be ascertained, it has been found to be due to coincident syphilitic processes in the brain, *e.g.*, chronic syphilitic meningitis. It is most important to remember that the period of active, true syphilitic processes may overlap that of the post-syphilitic degenerations. We can best understand the facts by conceiving that successive broods of organisms arise from latent germs (the amount of which we can never estimate), and that the degenerations due to the toxines of earlier germs may concur with active processes of later development.[2]

Atrophy of the optic nerves is, as is well known, frequent in locomotor ataxy. In what proportion of the cases it occurs is difficult to say. Those who observe cases from the ophthalmic side are naturally impressed with its frequency, since a large majority of cases of simple atrophy have this association. The proportion has much increased since the loss of the knee-jerk has been recognized as a sign of the early stage of tabes.

[1] By Bernhardt, Rendu, F. Pick, Schuster and K. Mendel. The true relation, as stated in the text, is shown clearly by F. Pick's case, in which typical degenerative tabes, was found to be accompanied by chronic syphilitic meningitis, cerebral and spinal, which had caused the optic neuritis. (Festschrift zu F. J. Pick, 1898, Neur. Centrabl., 1899).

[2] If we realize that only the developing or developed organisms, and their effects, can be influenced by treatment, and that the latent germs, as well as the toxines produced by earlier organisms, cannot be influenced, many mysteries become more clear.

But we must not infer from this the converse proposition that a large proportion of cases of tabes present optic nerve atrophy. It has been said that about one ataxic in 14 suffers from optic atrophy.[1] Of the last 400 cases of tabes that I have seen, optic nerve atrophy existed in 26, or 6·5 per cent. This is probably nearer the truth. When it occurs, it is more often an early than a late symptom, occurring before, rather than after, difficulty in walking, and in many cases ataxy does not develop. But the frequency with which tabes does not pass beyond the first stage is great, and it is doubtful whether the frequency with which optic nerve atrophy is associated with stationary pre-ataxic tabes, is greater than corresponds to the proportion of tabetics that do not advance beyond the first stage.[2] But optic atrophy often supervenes after ataxy has developed, and has become great; the loss of visual guidance necessarily increases the inco-ordination. Further, the disorder of movement may come on after the atrophy, and at any interval. I have known atrophic blindness to be complete for twenty years before ataxy of the legs developed, which speedily became such as to prevent standing. In another case, the interval was sixteen years between the blindness and inco-ordination. Of course, in such cases, the knee-jerk was probably lost from the first. Another early symptom is the loss of the light reflex of the iris, with preserved action on accommodation ("Argyll-Robertson pupil," "reflex-iridoplegia"). Loss of sight itself prevents the light-reflex, but the accommodation-contraction can still be obtained by making

[1] Voigt ("Berl. Kl. Wochenschrift," 1881, No. 39), and Erb ("Deut. Arch. f. Kl. Med.," 1879).

[2] The fact of the frequency with which ataxy does not come on when optic nerve atrophy develops, was pointed out in the first edition of this work (1879). A few years later the fact was emphasised by Benedikt, who disregarded the great frequency of stationary early tabes, and enunciated a law that the development of optic atrophy tends to prevent the occurrence of ataxy. Any so-called "law" easily obtains recognition, however doubtful are the facts on which it rests. Those which seem to support it, attract much more notice than those which do not.

the patient strive to look at his own hand placed near the eye.

To have a correct view of the relation between the symptoms, it is necessary to remember that tabes must be ascribed to the action on the nervous system of a post-syphilitic toxine, the chief incidence of which is on the ganglia of the posterior roots of the spinal cord. The cells of these determine the nutrition of the fibres of the posterior roots, and also of the fibres which pass up to the spinal cord, both of which degenerate. The afferent fibres which suffer first and usually most, are those from the muscles (hence the loss of the knee-jerk) and their related fibres in the posterior columns of the spinal cord. The sensory fibres from the skin may suffer little or much. Analgesia may extend even to the region of the fifth nerve. These variations in effect seem due to some minute variations in the character of the toxine; this is perhaps the result of a persistent perversion of the chemical processes in the body, varying from time to time.[1] The differences may be partly related to susceptibility of different tissues. In some cases the optic nerves come within the scope of the toxine, and then atrophy occurs. If the variety which affects the optic nerve has less influence on the muscle nerves, we can understand that ataxy should often be absent when optic atrophy occurs, and if variations in the toxine occur at times, during the course of the malady, we can understand the development of ataxy in previous stationary tabes, even of long duration. Another effect is certainly the isolated loss of the light reflex of the iris, which may occur alone, with no other symptom, or with tabes, or may precede, as well as attend, optic nerve atrophy. Yet another consequence may be the lesions which underlie general paralysis of the insane, and with this also we may have optic nerve atrophy, and also reflex iridoplegia, together or alone.

[1] Compare Martin's discovery that the poison which causes diphtheritic paralysis is due to such a perversion, the result of a ferment produced by the specific organisms. In this also we have varieties of effect, one of which may resemble tabes.

When the atrophy is advanced, the optic discs are usually grey, or greyish white, or white, and often, to direct examination, mottled, the meshes of the lamina cribrosa being visible; the edges are sharp and clear, the sclerotic ring distinct. Sometimes there is a peculiar gelatinous opacity of the substance of the disc. To ordinary daylight (from a sun-lighted cloud) the tint is a peculiar greenish grey. When the discs appear white to the indirect method, a grey mottling can often be seen by the direct method. The vessels are usually of the normal size. The grey disc and normal vessels have been supposed to be peculiar to this form of atrophy, but this is incorrect. The disc in atrophy from post-orbital pressure on the nerve may present the same aspect.

The anatomical characters of the atrophy have been already described (p. 110). The trunk of the optic nerve is commonly nearly normal in size, but is grey and semi-translucent. The degeneration may seem to stop at the chiasma, but often, as Türck pointed out, it involves also the optic tracts, and can be traced to the external corpora geniculata. The microscopical investigations (of Leber especially) have shown that the change in the nerve consists of an increase in the interstitial tissue, and sometimes the formation of translucent colloid tissue around the vessels, as in Fig. 51, p. 111, together with wasting of the nerve fibres. The histological resemblance to the change in the spinal cord is not so close as has been asserted. All evidence suggests that the atrophic change commences in the nerve fibres, but the amount of the interstitial tissue is unlike the condition in similar degenerations elsewhere.

The affection is usually bilateral, although often more advanced in one eye than in the other; one eye is often affected earlier, and in rare cases one eye suffers alone for a long time, a year or more.

Symptoms.—The affection of sight is usually characterized by a progressive defect in vision, which varies in its characters. Often it is a concentric restriction of the field, which increases, narrowing the area of vision until only a small portion is left around the fixing point. With this loss,

which renders sight such as is obtained by looking down a tube, central vision may be little impaired even after the peripheral defect has become very great. When the acuity of vision is thus preserved, patients may, for a long time, be unaware of the affection of sight. In other cases, acuity of central vision fails, with or without restriction of the field. Sometimes there is a sector-like defect of the field, an example of which is figured at p. 118. Rarely one half of a field may be lost (Fig. 56, p. 118), especially when the sight of the other eye has entirely gone. I have known such failure to occur in a few days, beginning as bi-temporal hemianopia —as if from a more acute process at the chiasma, leaving at last only half the field of one eye.

Colour-blindness is frequent, and is almost always an early symptom. The first change is commonly a loss of perception of green, and the later order of failure is generally red, blue, and lastly yellow. In the final stage of atrophy all colour distinctions are lost; but there is no constant relation between the degree of the loss of colour-sense and the amount of central amblyopia.

Pathology.—The pathological relations of tabetic optic nerve atrophy have been already considered. But it is important to note that the precise relation of the changes in the nerve elements is still uncertain. There are reasons, from analogy, to consider that the ganglion cell layer of the retina corresponds to the cells of the posterior spinal ganglia; the fibres from the rods and cones correspond to the peripheral sensory nerve fibres, and that those of the nerve fibre layer and the optic nerves, are analogous to the fibres of the posterior median and other columns in the spinal cord. Thus the variation in the precise elements degenerated, the restriction of the field, or central defect, may be analogous to the loss of pain, or touch or muscle sense, in the limbs.

The course of the optic nerve atrophy is very like that of the cord degeneration. Recovery of sight, if ever observed, is a still rarer event than recovery of co-ordination in the limbs, but arrest of progress is sometimes obtained as it is

in the ataxy. Many years may pass before vision is finally lost, and there is good reason to believe that arrest is, in rare cases, permanent. The treatment has been indicated in the preceding section, "Atrophy."

LATERAL SCLEROSIS.

Ophthalmoscopic changes are very rare in cases which present the symptoms of primary lateral sclerosis of the cord. Rarely grey atrophy slowly develops, similar in character to that met with in locomotor ataxy.

INSULAR (DISSEMINATED) SCLEROSIS.

The optic nerves suffer in two ways in insular sclerosis. In the first form, an islet of sclerosis may occupy one of the optic nerves, very rarely one of the tracts or the chiasma. Such a definite involvement by the morbid process is extremely rare, but when it occurs, it causes symptoms corresponding to its seat. If in the optic tract, or optic path in the hemisphere, the result is lateral hemianopia partial or complete, without marked ophthalmoscopic changes. But, considering the apparently random distribution of the islets of sclerosis, the extreme rarity of this effect is remarkable. Less rare is the involvement of the nerve trunk. Its effects are the symptoms of retro-ocular neuritis with partial damage to the fibres, causing alterations in the field of vision, often irregular in character. Signs of slight inflammation may be visible within the eye. The process of sclerosis is more or less inflammatory, at least in its early stages, and its degree, and proximity to the globe, determine its visibility therein. But the signs of inflammation are never great.

Much more frequent than indications of retro-ocular neuritis is the second form—a simple atrophy of the optic nerves, quite similar to that seen in tabes, but less progressive in course. It is characterized by the same simple pallor and wasting, and by similar changes in sight. Its frequency is difficult to estimate on account of the tendency

to erroneous, or at least unjustified diagnosis. Insular sclerosis of the cord is constantly assumed when all that the symptoms indicate is systemic sclerosis. On the other hand, slight pallor of the discs, with trifling amblyopia, is held to be evidence of optic atrophy. In some cases the signs of atrophy are accompanied by a central or paracentral scotoma, but in many of these there is a history of excessive smoking.[1]

Caries of the Spine.

Changes in the optic disc have been observed only when the caries has been in the cervical region, and then have been chiefly so-called "congestion of the disc." In the cases that have come under my own observation, the pathological character of the condition has been doubtful, so slight has been its degree. In the rare cases in which definite optic neuritis has been present, coincident intra-cranial tubercle was a possible, and even a probable cause.[2]

Multiple Neuritis.

Polyneuritis, or peripheral neuritis, has been perceived in recent years as a definite, although varied malady, depending on changes in the peripheral nerves, which suffer symmetrically, and according to their function. The important fact is the dependence of this affection on toxic material in the blood which reaches all parts, but damages only those that are susceptible to its special influence. The optic nerves seldom suffer, whether the neuritis is due to alcohol or arsenic, or other poisons. The former is by far the most common cause, but next in frequency, more frequent than arsenic, are various little-known endogenous toxines, which also seem to spare the optic nerve. Slight papillitis has indeed been seen when chronic meningitis coexisted as an effect of alcohol, but cannot be regarded as comparable to the general

[1] For some facts bearing on the subject see "Trans. Oph. Soc.," vol. xvii. for the statements of Dr. Buzzard, Dr. Russell and others.

[2] As in a case of caries in the dorsal region described by Taylor and Collier (*loc. cit.*), in which an old choroidal tubercle was also found.

affection of the peripheral nerves. This is, however, less mysterious if we consider the homology of the structures that is suggested by modern knowledge. The nerve endings in the limbs which suffer are far from their nutritional centres in the posterior ganglia or the anterior horns. But if we regard the ganglion cells of the retina as homologous to those of the posterior ganglia of the cord, the proximity of the nutritional centres and the terminal structures leaves less room for the influence of any but toxines of powerful influence. We should expect their action to be manifested rather by the longer fibres which connect the ganglion cells with the cerebral centre, and thus to anticipate retro-ocular neuritis as the effect. This does sometimes occur, and it is important to remember that the interstitial overgrowth, secondary to the degeneration of the nerve elements, seems often to take on an independent activity and to produce its own effects.[1] But, as a rule, the agents that are recognized as affecting the optic nerve, are not those which act on the peripheral nerves in the limbs. These special agents will be mentioned later. Very few cases are recorded in which the optic nerves were affected in association with widespread polyneuritis. Cases in which they apparently suffered, with other nerves, from alcohol, are very rare.[2] The effects of lead are well known; it acts on the peripheral nerves, but its influence seldom corresponds to the range of polyneuritis, as the term is now understood.

Injuries to the Spine.

The subject of the changes in the optic discs in spinal injuries has received a large amount of attention in consequence of the prominence which "railway cases" have given

[1] *See* Gowers' "Abiotrophy"—"Lancet," 1902, April 12; and "Lectures, &c.," Churchill, 1904.

[2] Schuster and K. Mendel record one case in which neuritis coincided with alcoholic neuritis, and another case of very obscure significance. Higier ("Neur. Centralbl.," 1898, p. 390) has striven to show the relation of retro-ocular neuritis to multiple neuritis by mixing up all toxic influences. The subject needs independent study.

to this class of accident. In its scientific relations, the subject has not escaped the sinister influence which litigation exerts on the investigation of facts, and there is no doubt that the pathological nature of many of the appearances, described in these cases, has been the result of an affection of the mind of the observer, rather than of the eye observed. Still, in some cases of spinal injury well-marked ocular changes have been described, especially by Clifford Allbutt, and are said to be more frequent the higher the injury is. Besides "simple congestion," "congestion with œdema" and "slight neuritis" have been described—conditions of doubtful significance. In most recorded cases sight has been slightly affected, and the amblyopia has passed away after a few weeks. But definite optic neuritis of the left eye occurred in a case of fracture (recorded by Taylor and Collier[1]), in which a piece of bone pressing on the lower part of the cervical cord was removed, and the neuritis rapidly subsided.

It has been supposed (first by the late Wharton Jones) that a disturbance of the sympathetic is the cause of the ocular symptoms in spinal injury. In cases of actual disease of the sympathetic, however, no ophthalmoscopic change has been found (Hughlings-Jackson, Riegel, and Jolly). Clifford Allbutt suggests that they may be the result of "meningeal irritation" passing up to the base of the brain, but other evidence of such irritation has not been recognized.

FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM.

Exophthalmic Goître.

The conspicuous ocular symptoms which form part of Graves' disease might lead to the expectation that changes in the fundus oculi would be found in that affection. As a rule, however, it is not so. The prominence of the eyeballs does not lead to any alteration in the optic nerve. The retinal

[1] *Loc. cit.*, p. 544, Case 5.

arteries share to some degree the general arterial dilatation, and are larger than normal; when their course is favourable for their comparison with the veins the two may be observed to be nearly equal in size. The strong pulsation which occurs in the arteries of the head and neck, in consequence of their dilatation and of the excited action of the heart, may be visible in the retina as a spontaneous arterial pulsation, as Becker[1] first pointed out.

Chorea.

Embolism of the central artery of the retina is an extremely rare result of the endocarditis which is generally found (post mortem) to be associated with the disease in severe cases. Only a few instances have been recorded. In one, by Swanzy, of Dublin,[2] the embolism occurred at the time of the commencement of the chorea, and was in the left eye. The chorea was most severe on the left side. The state of the heart is not mentioned. The other, recorded by Förster, was not seen until some time after its occurrence. The patient, a child, had suffered from chorea for some years, and during the chorea had lost the sight of one eye. The disc was atrophied, and the arteries very small.

Slight optic neuritis is not very rare in chorea, and now and then attains a considerable degree, although seldom comparable to that of tumour. I have twice met with considerable neuritis. In each case the patient was a girl of seventeen or eighteen years, and in each the neuritis passed away completely as the chorea subsided. These cases give significance to the slighter forms, in which the edge of the disc is decidedly blurred, although not often to such an extent as to prevent its position being recognized in the indirect method of examination, although it can scarcely be perceived by the direct method. In all the cases I have seen, there has been a recognizable degree of hypermetropia; this fact would lessen the significance of the neuritis in

[1] "Kl. Monatsbl. f. Augenh.," Jan. 1880.
[2] "Ophth. Hosp. Rep.," viii. 181.

regard to the chorea, were it not that the disc became normal when the chorea subsided. The frequency with which such neuritis is met with is difficult to ascertain. The slight form is seen in children—the more severe in girls about puberty. When intense, it may be a coincident effect of some state of the blood, which may vary in degree and character. Acute chorea may be followed by endocarditis of malignant character, and retinal hæmorrhages may occur, with white centres, of the septic embolic type. (*See* "Heart Disease.")

Neuralgia and Migraine.

Occasionally atrophy of the optic nerve has been observed in cases of severe unilateral neuralgia of the fifth nerve. Its origin is obscure. Temporary amaurosis, supposed to be "reflex," is more common. The transient disturbances of sight, temporary amaurosis, hemianopia, &c., which accompany migraine, are well known. In a girl, aged eighteen, blindness of the left eye occurred after some days of migrainous pain. The ophthalmoscopic appearances were normal; vision was qualitative only, and the pupil did not react to light. Treatment was without effect for fifty days; then chloral and quinine were given, and slight improvement took place, probably not due to the treatment. The slow improvement of vision went on, and the sight ultimately became good.

Of great importance, however, are the attacks of loss of sight lasting for a few hours or a day or two, occasionally observed in the subjects of migraine, sometimes apart from attacks of headache, at other times in association with the pain. This failure of sight, usually transient, is sometimes permanent, always in one eye only. The ophthalmoscopic appearances in such cases are those of occlusion of the central artery. The state is usually ascribed to embolism, but it is more like thrombosis. Galezowski[1] has recorded three such

[1] "Rec. d'Oph.," Jan. 1882. *See* also Rampoldi, "Ann. di Ottalmo.," 1882. A case described by Doyne was probably of this nature. There had been two attacks of transient blindness of one eye, in the last

cases without heart disease, also one in which slow atrophy occurred in one eye, and another in which failure of sight after an attack of migraine was attended by signs of neuro-retinitis, with small hæmorrhages and thrombosis in some minute vessels. Now and then atrophy of the optic nerve has been observed to follow repeated attacks, and Hutchinson has associated the three symptoms—migraine, amaurosis and xanthelasma. Glaucoma is also sometimes observed in cases in which there has been long-standing neuralgia of the fifth nerve. It has been proved that irritation of the fifth nerve may increase the intra-ocular tension.[1]

Idiopathic Epilepsy.

Inter-paroxysmal State.—In idiopathic epilepsy the appearance of the fundus oculi between the paroxysms is, as a rule, normal. Some observers have described changes in the optic discs,—increased vascularity, distended retinal vessels, and the like. I have examined very carefully several thousand epileptics, and have found in most cases, every character of the fundus to be such as is presented by persons not epileptic. Now and then an unduly red disc is to be seen, but not more frequently than in persons not epileptic, and in most cases it is explicable by the ocular conditions—a point too much neglected in medical ophthalmoscopy, at least in former years. Spontaneous pulsation in the veins has been said to be especially frequent in epileptics: it is certainly not more frequent in them than in individuals who are not epileptic.

During the paroxysm the appearance of the fundus has been described variously by different observers. For obvious reasons, the difficulties in the examination are great, and

of which the upper half of the retina was found to be œdematous. The ascending arteries ultimately became narrowed on the disc, and the lower part of the field remained defective after several months.—"Trans. Ophth. Soc.," vol. ix. p. 148.

[1] Hippel and Grünhagen: "Arch. f. Ophth.," vols. xiv. and xvi.

opportunities are rare. The only change which seems well established, is that the retinal veins, during the stage of lividity, become much distended. I have had an opportunity of observing the fundus several times during minor attacks, and it has presented no change. In a case of convulsions from meningeal hæmorrhage, and in another case of severe one-sided fits, I have been able to keep a retinal artery and vein under (direct) view through the whole of a severe fit, from before its commencement until after its close. In neither case did the retinal artery present the slightest change in size. During the stage of lividity, the vein became large and dark. In a case of chronic local meningitis of the motor region of the left hemisphere, by applying electricity to the region of the cervical sympathetic, I was able to produce the aura with which the fits commenced, and once watched the retinal vessels by the direct method during the procedure, but no change in their calibre was to be observed, although the aura was so intense as almost to pass into a fit. I have repeatedly examined patients immediately after fits, but without being able to satisfy myself that there was any difference in the appearance of the disc and vessels from their aspect at other times. It is possible that, as Knies[1] has suggested, changes in the size of the vessels sometimes described, may be due to a sudden alteration in the intra-ocular pressure, from changes in the accommodation.

Even when fits have been frequent, as a rule, my own observations have given negative results. In one case, however, I met with marked changes in the discs, developed under observation during a series of exceedingly severe convulsive attacks, recurring at short intervals for several days. The patient was a young man, and the convulsions were of hysteroid type—paroxysms of struggling, arching of back, throwing about of head and limbs, so intense that the united strength of three or four persons was required to keep the man in bed. They were accompanied by loss

[1] "Sitzungsbericht der Heidelberg Ophth. Gesellsch," 1877, p. 61.

of consciousness. Bromide and other remedies produced no effect, and the convulsions continued unabated until ice was applied to the cervical spine, when the attacks at once ceased. The optic discs, after some days of convulsion, became reddened and veiled, so that their edges were quite invisible, and there was distinct swelling. After the cessation of the fits the discs gradually resumed their normal appearance. This patient, about three months later, died, after a series of true epileptiform convulsions beginning in the left hand. Post mortem, no trace of disease was visible in the brain to naked-eye examination.

It might be expected that the retinal vessels would often give way during the violent venous stasis of an epileptic fit, just as do those of the conjunctiva. It is not so; retinal hæmorrhage is rarely observed under the circumstances, no doubt on account of the support afforded to the walls of the vessels by the intra-ocular tension.

It must be remembered that some cases of apparently idiopathic epilepsy present traces of old optic neuritis or choroiditis—indicative, the former certainly, the latter probably, that the convulsions originated in organic brain disease; the choroiditis indicating former syphilis. Traces of old optic neuritis are sometimes seen in cases of epilepsy due to blows on the head. It must also be remembered that chronic convulsions resembling idiopathic epilepsy may occur in the subjects of lead-poisoning and of chronic renal disease, in each of which optic papillitis may be present.

Hysteria.

Although functional disturbances of sight (single or double amblyopia, hemianopia, colour-blindness, often with pain on use of the eyes), occur occasionally in the hysterical, related ophthalmoscopic changes are unknown. Atrophy of the optic nerve has been met with in one or two cases, but was probably an accidental coincidence; or there may have been co-existent organic disease, such as insular sclerosis, underlying the manifestations of hysteria. When there is extreme

amblyopia, dilatation of vessels and serous transudation into the retina have been seen by Landolt. These, and the chronic "perineuritis" described by Galezowski in one case, must be regarded as without significance.

Insanity.

The frequency with which pathological appearances are to be recognized with the ophthalmoscope in cases of insanity has been very variously stated. The discrepancy between observers is so great as to make it certain that undue weight has often been given to appearances which are not uncommon in normal conditions. In fact, the ophthalmoscopic appearances in the insane seem, for some reason, to be a favourite subject for observers whose experience of normal eyes is insufficient to enable them to estimate the significance of the appearances seen. The observations in which changes were found in a large proportion of the cases examined must therefore be received with more than reserve. As an instance of the different conclusions which have been reached may be cited the observations of Tebaldi,[1] who found changes in three-fourths of the cases examined; and of Schmidt-Rimpler,[2] who found changes only in thirteen out of 128 cases, and some of the thirteen he considered as doubtful. An even more striking instance of this discrepancy is afforded by two observers of the appearance in general paralysis, one of whom described atrophy as existing in eight out of every nine cases examined, while the other found hyperæmia in about the same proportion.

It must be remembered, in estimating the significance of the considerable changes sometimes found, that cases of organic brain disease, tumour, softening, chronic meningitis, and the like, in which mental disturbance is prominent, occasionally find their way into asylums.

General Paralysis of the Insane.—This disease is more

[1] *See* Nagel's "Jahresbericht," 1870, p. 374, from the "Rivista Clinica," 1870.

[2] "Ann. d'Oculist.," vol. lxxiv., 1875, p. 267.

closely allied to tabes than to other forms of mental derangement. It is, on the one hand, often associated with symptoms of tabes, or, on the other, with those of lateral sclerosis. It resembles tabes, moreover, in being in the majority of cases a late sequel to syphilis, at least in its well-marked forms. Unequivocal changes in the eye have been found much more frequently than in any other form of insanity. Loss of sight has been known since the time of Calmeil as an occasional complication; but in a considerable degree it is rare. Billod noted complete blindness in only three out of 400 cases.[1] The loss of sight has been proved to depend on grey atrophy of the optic nerves, similar to that which occurs in tabes. The retinal vessels have been normal in size or narrowed. In slight degree, it may affect one eye more than the other.

As in locomotor ataxy, it may be an early event, and may even precede the other symptoms of the disease. Its general features closely resemble those of the atrophy met with in association with tabes, so that a separate description is superfluous. The changes in the optic nerves found after death are also similar.[2]

A stage of hyperæmia of the discs, preceding the atrophy, has been described by a few, but for the most part the atrophy is simple from the first. Any degree of papillitis is exceptional, and due to some special cause, such as an active syphilitic process, which as we have seen, may co-exist with degeneration. (*See* Tabes.)

The only other appearance observed on which any reliance can be placed is a slight degree of congestion during the paroxysmal disturbance, common in this disease. Even this can only be regarded as important when it has definitely cleared away after the attack has passed.

Melancholia.—Most observers have reported the ophthalmoscopic appearances in melancholia to be normal, and with this my own observations entirely agree. Even in the

[1] "Ann. Med.-Psychologiques," 1863.

[2] Quoted by Robin: "Des Troubles Oculaires dans les Maladies de l'Encephale," p. 330, 1880.

acute form, any morbid appearances that have been described are of no significance, with the exception of associated changes connected with the cause of the disease.

Indeed, it may be said that, in the past, the various forms of insanity have constituted a happy hunting-ground for the ophthalmoscopic neophyte. But the ground has been deprived of most of its attractions by the more adequate ophthalmoscopic training of recent years, which has so seriously deprived the observer of the aid of imagination.

DISEASES OF THE URINARY SYSTEM.

Bright's Disease.

Uræmic Amaurosis.—In all forms of renal disease, if severe, loss of sight from uræmic poisoning may occur.[1] Its characteristics are the sudden onset, completeness, the usual absence of ophthalmoscopic changes (excepting such as may have before existed), the preservation of the reaction of the pupil, and the quick disappearance of the symptom when the blood-state is relieved by purgation or diaphoresis.

To the almost invariable rule that the ophthalmoscopic appearances are unaffected by uræmia, a few exceptions have been recorded. Thus, slight and transient œdema of the papilla has been noted to coincide with the acute symptoms, and subside when they had passed away, with and without chronic retinal change.[2]

Retinal Changes.—In all diseases of the kidney of consid-

[1] The association of transient amaurosis with dropsy after scarlet fever was noted in 1812 by Wells ("Transactions of a Society for the Improvement of Medical and Chirurgical Knowledge," vol. iii.). The first observation of actual changes in the retina was made (post-mortem) by Türck in 1750 ("Zeitschrift der Wiener Aerzte," No. 4, 1850). The microscopical changes were first carefully studied by Zencker ("Arch. für Ophth.," ii. 142) and Virchow ("Arch. fur Path. Anat.," x. 1856, p. 178).

[2] Dobrowolsky, "Klin. Monatsbl. für Augenheilk.," March, 1881, p. 121; and Letten, "Charité Annalen," 1879, p. 169.

erable duration, the vessels of the retina may present changes in common with those elsewhere. This degeneration may lead to simple retinal extravasations. But considerable changes often occur in the retina, which vary in different cases, and are commonly described by the general, but not very accurate, term of "retinitis albuminurica."

Vessels.—In some cases of chronic renal disease, especially of the granular form, there is to be seen a notable diminution in size of the retinal arteries, independently of the existence of any special retinal disease.[1] The veins may not be larger than the normal, but the arteries are not more than one-half or one-third the diameter of the veins (Pl. VII. 2), instead of being two-thirds or three-quarters. The comparison can only be made, as already stated (p. 9), between arteries and veins which correspond in distribution. When there is obstruction from papillitis, the arteries may be seen, even by the direct examination, as lines only (Pl. VII. 4), smaller than is ever seen in simple papillitic obstruction without Bright's disease. When this reduction in size exists the pulse presents marked incompressibility. In one case of acute Bright's disease becoming chronic, a diminution in size was observed to coincide with a very marked increase in the tension of the pulse. The contraction is not visible, however, in all cases in which the arteries are tense. In the absence of any cause for the reduction, it must be ascribed to arteriole contraction, and supports the view of the late Dr. Johnson, that such contraction exists, and causes the hypertrophy of the muscular coat of the arteries.

The walls of the arteries are frequently altered in chronic Bright's disease, especially in cases of chronic granular kidney, but also in the later stages of disease which began as acute nephritis. The common change is a uniform thickening by the development of hyaline tissue, which seems to occupy the middle coat, but has been said to begin in the inner layer of the intima.[2] Its first effect is to increase the brightness of the central reflection from the artery, giving

[1] *See* Gowers, "British Medical Journal," December 9, 1876.

[2] Brailey and Edmunds: "Trans. Oph. Soc.," vol. i. p. 44.

rise to the appearance known as "silver wire arteries" (*see* p. 14). Ultimately the thickening becomes great, and may cause a white line to be seen on each side of the vessel. (This appearance, however, is also seen in cases of a natural excess of connective tissue about the vessels on and near the disc, especially conspicuous when this is unduly red.) A more obtrusive proof of the thickening is afforded by the compression of a vein when it passes under such a thickened artery.[1] Such obstruction of the veins was conspicuous on the retina, from which the section of an artery is shown in Fig. 67, in which the hyaline thickening of the wall is strikingly shown, and the disturbance the enlargement of the vessel has caused in the whole thickness of the retina. The venous obstruction thus produced is also conspicuous in the vein and artery passing upwards in Fig. 68 from a case of

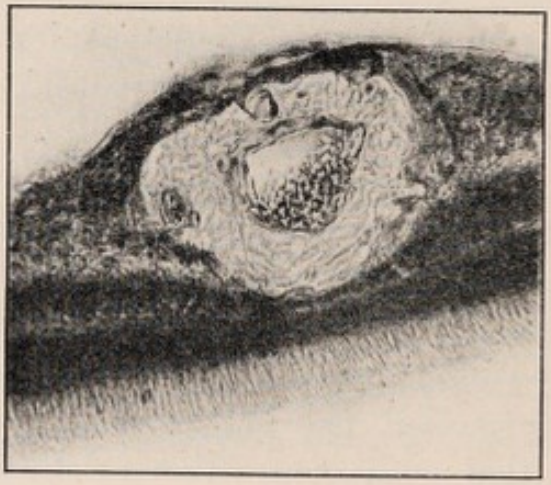


FIG. 67.

ARTERIAL CHANGE IN CHRONIC ALBUMINURIC RETINITIS.

(From a micro-photograph kindly supplied by Dr. Percy Flemming).

The wall of the artery is the seat of great hyaline thickening. During life the arteries presented the characteristic "silver wire" central reflection, and where an artery crossed a vein, its thickening caused obvious obstruction to the venous flow. Old changes existed at the macular region, numerous scattered white spots with extensive diffuse finely granular pigmentation, while elsewhere were a few larger white areas, soft-edged, and evidently more recent.

[1] Gunn: "Trans. Oph. Soc.," xii. p. 124, and xviii. p. 356.

old kidney disease, extreme atheroma of the cerebral arteries, and thrombotic hemiplegia.

The thickening of the wall necessarily diminishes the calibre of the vessel, and the change may lead to its occlusion. This has occurred in the artery shown passing downwards in the figure, which has the aspect of a white band, ending in a tapering extremity, beyond which it can be traced as a fine red line, where peripheral circulation has apparently been maintained by anastomoses. A section of this artery is shown in Fig. 69, in which clot within it has apparently become organized into connective tissue, and the contraction of the lumen has caused the wall to present a

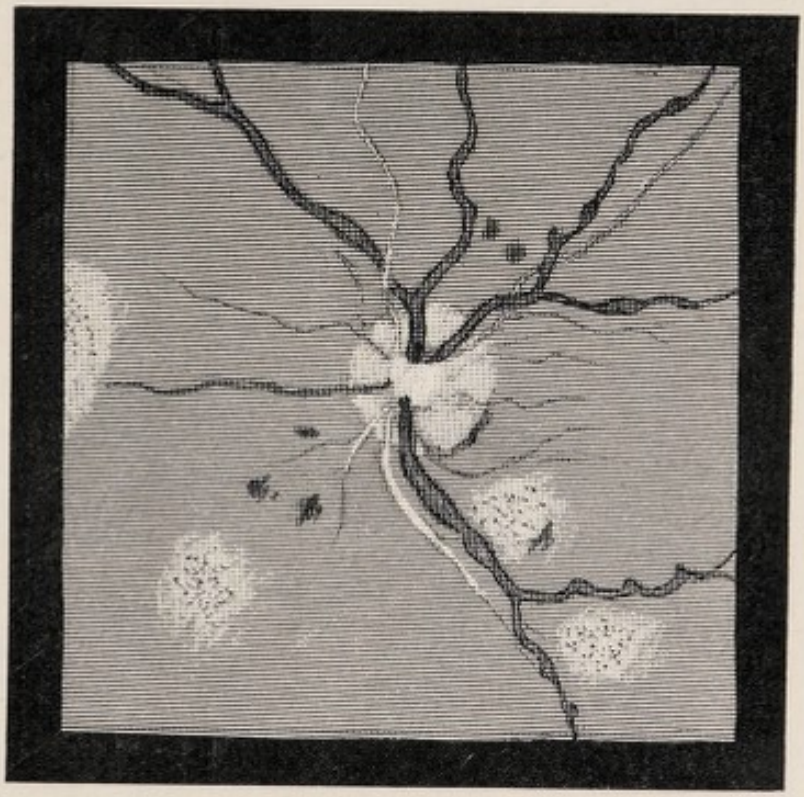
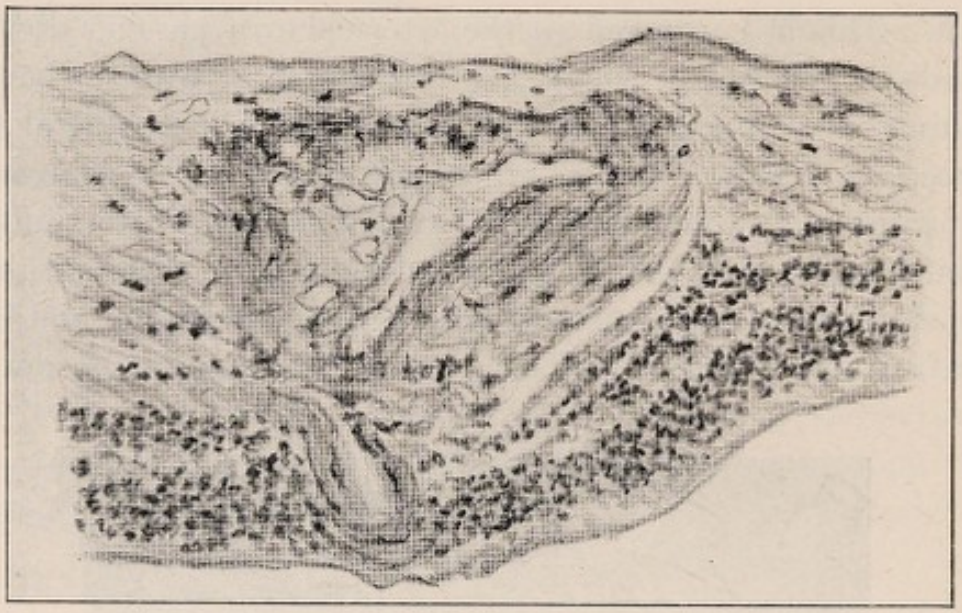


FIG. 68

Changes in retina and vessels in a man with chronic renal disease and extensive atheroma of the arteries of the brain, which had caused hemiplegia from thrombotic softening The chief features are mentioned in the text. Besides the chronic pigmented areas in the retina, and the small hæmorrhages, the veins present remarkable dilatations and are compressed by the thickened arteries. A microscopical examination showed the artery passing down, which appears as a broad white band, to be completely obliterated *See* next figure.

triangular section. The great disturbance of the retinal elements is conspicuous, a small thickened vessel beneath having interrupted the molecular layer.



IG. 69.

The closed artery shown in the last figure is seen to be much reduced in calibre and triangular in section. Its cavity is partly occupied by an organized clot, evidently of considerable duration, since it is becoming fused with the wall by nutritional changes, and the wall itself appears to be blending with the connective tissue of the adjacent retina. A small thickened vessel lies below the artery, and the two occupy almost the whole thickness of the retina, having interrupted the nuclear layers. The rods and cones had disappeared from the preparations. (From a section by Dr. F. Buzzard.)

An even more remarkable example of the effects of thickening of the arterial wall is seen in Pl. X. Fig. 1, in which the increased tissue of the wall is apparently in the outer coat, and ceases suddenly, so that the column of blood continues in apparently undiminished volume. The patient, in middle life, was suffering from advanced renal disease. One artery presents two small aneurismal dilatations, a striking feature in view of the fact that the patient died, a few weeks later, from cerebral hæmorrhage. In the retinal capillaries irregular dilatations may be found, especially in cases of retinal degeneration, as in Fig. 71, p. 207. In this figure an increase of the nuclei of the capillary wall is seen in places, thickening it. It is probable that the degenera-

tion of such nuclei, and the formation of such aneurismal dilatations, are the conditions which lead to hæmorrhages, which were numerous in this case (Pl. VIII. 1).

Hæmorrhages form a conspicuous feature of most cases of retinal disease in albuminuria. Their common seat is the nerve-fibre layer, in which they are striated and flame-shaped, and may follow the course of the vessels. Less commonly they may occur in other layers, and are then rounded and irregular. They may detach the retina from the choroid or burst through into the vitreous. They sometimes occur, however, apart from other retinal changes, as isolated evidence of the hæmorrhagic tendency. An instance of this is shown in Pl. VII. 1. The retina which presented this extravasation, even up to the time of the patient's death, several months later, showed no sign of other changes. The hæmorrhages are probably due to the weakening of the wall of the minute vessels (by such changes as have been just described), and to the increased intra-vascular tension, causes which also give rise to the extravasations into the brain, so common in the same cases.

"Albuminuric Retinitis."—The special retinal alterations are met with chiefly in chronic forms of renal disease—those which are chronic from the beginning, or which become chronic after an acute attack. They have been met with in most forms of kidney disease—granular kidney especially; large white kidney, sequential to an acute attack; and lardaceous kidney. They are by far the most common in the granular form, and least common in the lardaceous kidney.[1] The tendency to their occurrence is said to bear some relation to the amount of albumen in the urine.

Both eyes are almost invariably affected. Yvert, however, records a characteristic case,[2] with numerous hæmorrhages, in which the left eye only was affected. Recovery occurred

[1] It has been said that retinal changes do not occur with lardaceous disease of the kidney. Cases have, however, been recorded by Beckmann, Traube, Alexander, Argyll Robertson, and Bull, and one case has come under my observation.

[2] "Rec. d'Ophthal.," 1883, p. 145.

in the retina, but the patient died, and was found to have only the left kidney, and that was diseased. Yvert assumes a reflex influence as well as of blood-state.

The frequency of retinal changes has been variously stated. Published statistics vary between 7 and 33 per cent. Eales,[1] in 100 cases of chronic disease, found retinal changes in 28, or one in $3\frac{1}{2}$, and this probably represents the maximum. The variation is doubtless due to the relation of retinal changes to the duration of the disease. They occur only after the disease has exerted its influence for a considerable time. They commonly succeed the development of cardiac hypertrophy, but may occur without it. It is not probable that there is any connection between the retinal and the cardiac change, other than that both indicate a pronounced and prolonged effect of the renal disease upon the system. Renal disease may be first ascertained by the discovery of the ocular change, when it has been insidious in onset. Retinal changes occur without albuminuria in the very rare examples of granular kidney, in which albumen may be absent from the urine for a long time. A lady, aged fifty-seven, suffering from hemiplegia, presented characteristic degenerative albuminuric retinitis in each eye. She had hypertrophy of the heart, with strong aortic second sound, but without albumen in the urine. Similar cases have been recorded. In some of them there was polyuria.[2]

The retinal changes, as a rule, are found only in cases of organic disease. But in the mysterious albuminuria of adolescence Eales has described retinal alterations. Of 14 cases of young men between eleven and twenty-eight suffering from what was believed to be temporary slight albuminuria, he found retinal changes in 5 (white specks in 4, white patches in 1). In such cases it is difficult to believe that the kidneys were healthy.

The retinal disease presents certain elements which are variously combined in different cases. These are—(1) diffuse slight opacity and swelling of the retina, due to œdema of

[1] "Birmingham Medical Review," Jan. 1880, p. 34.

[2] Abadie, "La Union Médicale," 1882, p. 627.

its substance; (2) white spots and patches of various size, character and distribution, due partly to inflammatory, but for the most part, to degenerative processes; (3) hæmorrhages; (4) inflammation of the papilla; (5) atrophy of the retina and nerve may succeed inflammatory changes.

In most cases one or other of these predominates, especially in the early stage, and, according to the element most conspicuous, four types of disease may be distinguished. These are—(1) the degenerative; (2) the hæmorrhagic; (3) the inflammatory, and (4) the neuritic, according as white spots of degeneration, extravasations of blood, parenchymatous retinal inflammation, or inflammation limited to the optic nerve, predominate. It is, however, to be observed that degeneration and hæmorrhage commonly accompany or succeed the inflammatory changes, and that forms are often seen combining the characters of these varieties. In the typical degenerative and hæmorrhagic forms, the signs of inflammation are inconspicuous or subordinate.

The *degenerative form* (Pl. VII. 2) is the most common. It usually commences without signs of inflammation, by the appearance of small whitish spots on the substance of the retina, sometimes near the optic nerve entrance, sometimes at a distance. They are commonly at first soft-edged and rounded, and as they get larger become irregular. Generally, small very white spots, often punctiform or elongated, make their appearance around the macula lutea, arranged in a radiating manner, although seldom forming a complete circle. These are sometimes so minute as to be only visible on careful direct examination; sometimes they are large and very conspicuous, and then may be arranged irregularly, end to end, so as to form radiating streaks, beyond which dots are scattered (Fig. 70, Pl. VIII. 2). Often a less intense and diffuse opacity is visible in tracts here and there. Sometimes the larger spots coalesce into white areas, which may surround the disc.

Hæmorrhages, almost constant in all varieties, are least in the most chronic degenerative forms. Often they are adjacent to the white spots due to the changes in the

nerve fibres, and, lying for the most part in the nerve-fibre layer, they have a more or less striated arrangement, determined by the nerve-fibres, the direction of which the striæ follow. Sometimes linear hæmorrhages are seen. When larger, the extravasations are more or less flame-shaped. When small, they often lie adjacent and parallel

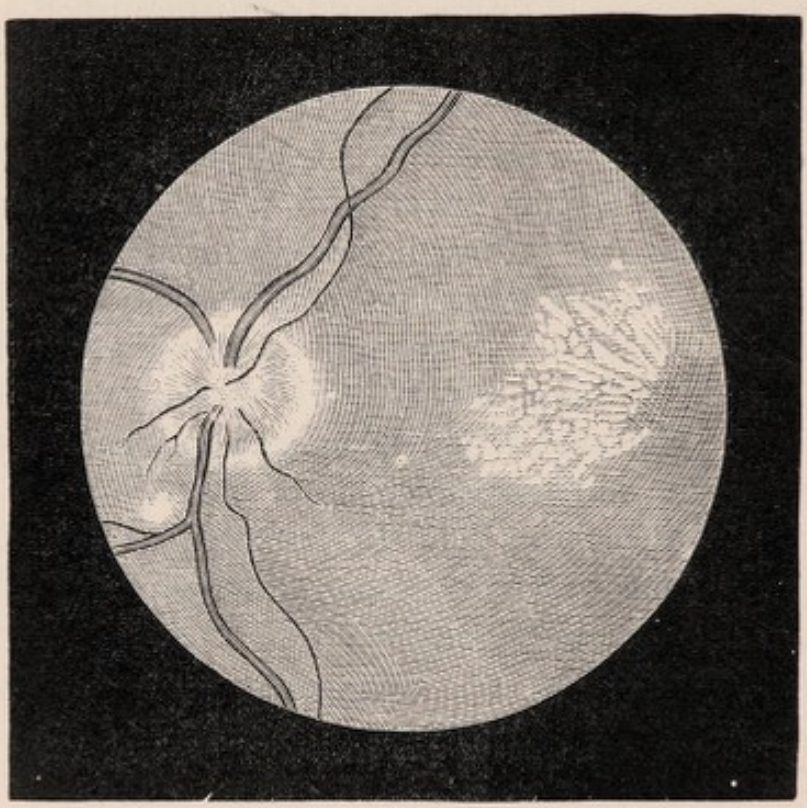


FIG. 70.—THE RETINAL CHANGES IN ALBUMINURIA.

A fan-shaped group of white spots radiating from the macula lutea; small arteries; slight papillitis.

to vessels, but it is not often that the vessel from which they originate can be traced. When large they may be irregular in shape and occupy the deeper layers of the retina.

The diffuse opacity already described is sometimes considerable and accompanied by a little swelling in places. Such a change is, however, rarely well marked in the form which begins with simple degeneration.

The retinal changes in this form may be considerable without any alteration in the optic disc. Often, however, its edges become blurred, the physiological cup indistinct and the tint abnormal, reddish-grey.

In two patients suffering from lardaceous degeneration Bull[1] observed the whole retina to present a uniform whitish infiltration, with numerous hæmorrhages. He suggests that the appearance may have been due to lardaceous degeneration of the retina.

In the *hæmorrhagic* form, the conspicuous change is the occurrence of a large number of hæmorrhages, with but little degenerative change and but slight signs of inflammation of disc or retina. Commonly, especially after a time, there is more or less degeneration adjacent to the hæmorrhages, and traces of the halo of spots around the macula are rarely absent. The hæmorrhages, for the most part, resemble those just described, differing only in their number, size, and predominance. Indeed, they are so prominent in the next form as often to make the distinction of the two difficult.

In the *inflammatory* form (Pl. VIII. 1) there is a general parenchymatous swelling of the retina with complete obscuration of the disc. The vessels are concealed, the arteries especially. The veins are distended, and sometimes have an extremely irregular and tortuous course over the fundus; the arteries are narrow. Hæmorrhages occur in considerable number, and are often large and striated. White spots are commonly numerous, and more or less uniform in character, especially in the acute cases, in which they are large, rounded (as in the figure), and soft-edged. In these cases there is rapid degeneration of the tissue elements, and abundant infiltration with lymphoid cells. If the inflammation subsides, the signs of degeneration may become more predominant, and the optic nerve may present evidence of secondary atrophy. This form is chiefly met with in cases in which the effect of the renal disease on the system is intense, and usually soon leads to death.

[1] "American Journal of Med. Science," Oct. 1879.

Neuritic Form (Pl. VII. 2, 3, 4).—In some cases the inflammation of the optic nerve predominates over the other retinal changes to such an extent that it may appear to be the only alteration, and may present nearly the aspect which is common in intra-cranial disease. The edges of the disc are veiled under a greyish-red swelling, of moderate prominence, which may extend a little distance beyond the normal edge. The prominence may be slight, or such that the veins form conspicuous curves over the sides. The arteries are usually narrow, and often concealed in the swelling; even the veins may be concealed. On direct examination it is generally conspicuously striated. Frequently, on its surface, or apparently beneath this, there is a conspicuous white reflection in certain spots (Pl. VII. 2), most distinct on oblique ophthalmoscopic illumination. Occasionally on the surface of the swollen papilla may be very minute white dots (just recognizable in Pl. VII. 3).

A careful examination will show, in almost all cases, signs of slight retinal degeneration, sometimes so slight as to be discerned only by careful focussing by the direct method. Sometimes, as in Pl. VII. 2, there are one or two white spots in the retina, near the neuritic swelling. At others, as in Pl. VII. 3, 4, minute white spots are to be detected near the macula lutea. Frequently small hæmorrhages are to be seen somewhere about the fundus (Pl. VII. 4). It is remarkable that there is little tendency for hæmorrhages to occur in the swollen papilla in this form. If the neuritis subsides, a condition of consecutive atrophy may be left—a filled-in disc, greyish, with paler lines along the vessels, and often extremely small arteries. Such a condition is shown in Pl. VII. 4.

Anatomical Changes.—The scattered white spots usually depend on degeneration of the layer of nerve fibres, which is thickened. The fibres often present varicosities, which may attain a large size and become crammed with fat-like globules. These ultimately become isolated as large fat-containing spheres, which, with free globules of fatty

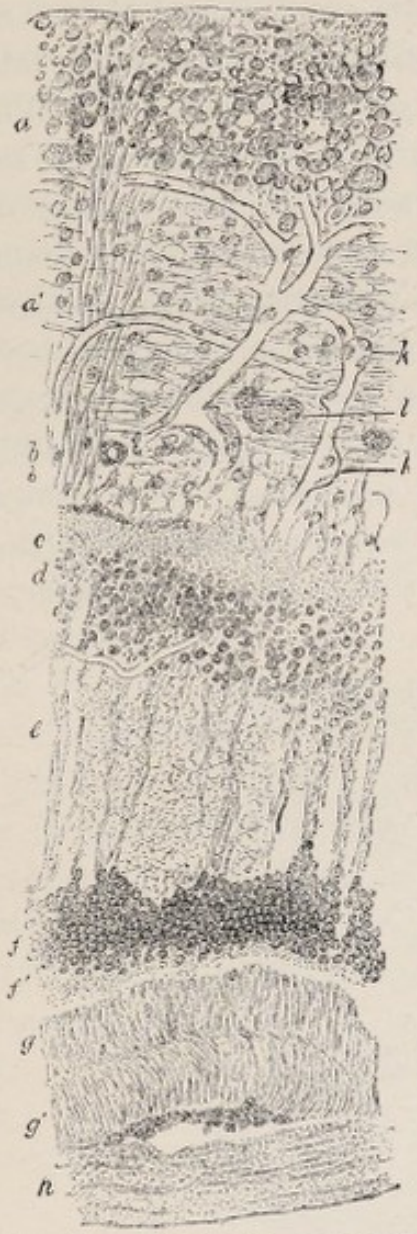


FIG. 71. — SECTION THROUGH RETINA IN A CASE OF ACUTE ALBUMINURIC RETINITIS.

The section passes through one of the white spots near the disc, shown in Pl. X. 1. The retina is greatly thickened, mainly from changes in the nerve-fibre layer (*a a'*), where numerous granular bodies are seen (such as are shown more magnified in Fig. 72). Capillaries are dilated, with conspicuous alterations in their walls; one of them (near right edge of figure) presents a series of aneurismal dilatations). (× 180.)

matters, are found abundantly on microscopical examination of recent specimens (Figs. 71, 72), and are very conspicuous on a surface view (Fig. 74). The degeneration occurs also, and sometimes chiefly, in the deeper layers, which may also be infiltrated with the "compound granule cells." Dege-

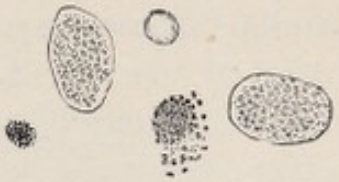


FIG. 72.

PRODUCTS OF DEGENERATION FROM A WHITE PATCH IN A CASE OF ALBUMINURIC RETINITIS. (× 250.)

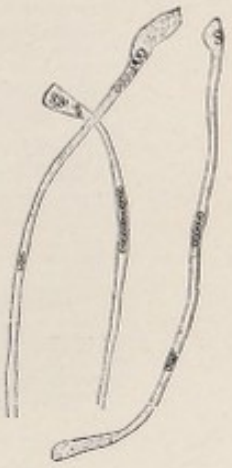


FIG. 73.

DEGENERATED FIBRES OF MÜLLER FROM A CASE OF ALBUMINURIC RETINITIS.

Swelling of the ends of the fibres, and rows of fatty granules due to degeneration. (× 250.)

neration of other retinal elements, round corpuscles, and vertical fibres of Müller may sometimes be found. The latter are swollen and contain minute oil globules (Fig. 74). When swollen they have an undue refraction, and have been said, rather unnecessarily, to be "sclerosed." It is to the position of these that the stellate zone of spots around the macula has been attributed. It is more probable, however, that a radial puckering exists around the fovea in all cases of retinal œdema, and that the stellate figure is due to a deposit of fibrinous material in the folds so produced.[1] The diffuse opacity of the retina is in part due to œdema. The elements of the nerve-fibre layer may be separated by clear spaces, and similar spaces may form in the ganglion-cell layer, in

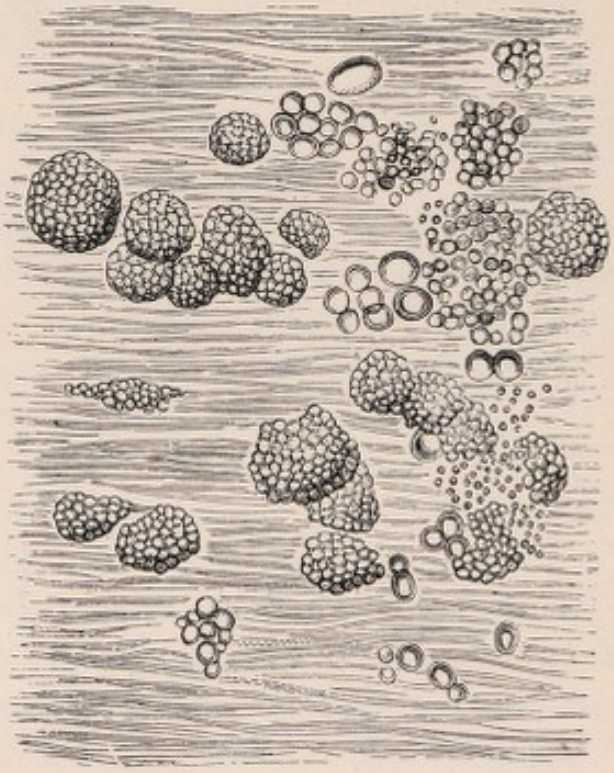


FIG. 74.—SURFACE VIEW OF A WHITE SPOT ON THE RETINA IN ALBUMINURIC RETINITIS.

The transverse lines indicate the nerve fibres. Among these are large and small oil globules and spherules consisting of similar still smaller globules. (After Pagenstecher and Genth.)

[1] *See* Gunn, "On the changes in the Macula associated with Retinal Inflammation and Œdema." "Trans. 8th Intern. Ophth. Congress," 1894.

the molecular and even in the nuclear layers. In this condition the ganglion cells often fall out of the section (Fig. 75). The diffuse opacity is also partly due to an infiltration of the retinal interspaces with a coagulable fluid, which, after hardening processes, presents an appearance of interlacing fibrillæ with granules at their points of intersection. This change may occupy large areas, as in Fig. 71, especially in the outer molecular layer, where cavities, containing this substance and separated by the remains of the vertical fibres, may chiefly be conspicuous. A similar effusion may also separate the "membrana limitans interna" and the bases of Müller's fibres from the rest of the nerve-fibre layer. Occasionally the layer of rods and cones presents remarkable thickening, such as is shown in Fig. 71, and is sometimes seen in other morbid states of the retina. Liebreich has called attention to the occurrence of small angular grey spots of pigment, often arranged in groups, and appearing first in the periphery. They are due to changes in the pigment-epithelium, and are seen especially in cases in which a parenchymatous inflammation has passed away.

Choroidal Changes.—Occasionally, although rarely, choroidal hæmorrhage may occur in Bright's disease, and may lead to

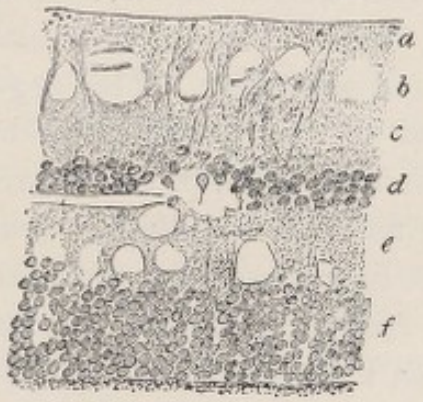


FIG. 75.—SECTION THROUGH THE RETINA, SOME DISTANCE FROM THE DISC, IN A CASE OF ALBUMINURIC RETINITIS, SHOWING ŒDEMA.

The nerve-fibre layer (*a*) is normal, but in the nerve-cell layer (*b*) the ganglion-cells have fallen out, owing to the formation of spaces round them in consequence of the œdema. The other layers show a tendency to dissociation of their constituents, and to the formation of spaces here and there. (× 150.)

circumscribed atrophy of the choroid with adjacent pigmentary disturbance. A peculiar "colloid" degeneration of the vessels of the choroid in old cases of albuminuric retinitis has been figured by Poncet. It leads to a thickening of the tissue of the choroid.

Symptoms.—In the slighter forms of the degenerative, hæmorrhagic, and neuritic varieties, vision may be unaffected. More considerable alteration, and even slight parenchymatous inflammation, commonly entails amblyopia, without limitation of the field or changes in colour-vision. In rare cases colour-vision may be affected. As the changes progress, the interference with vision increases. When the macular region is damaged, central vision is impaired, but this is not common. Degenerative changes rarely reach the centre of this region, no doubt because the structures in which the degeneration occurs do not extend to the fovea centralis itself. Hæmorrhages are also rare in this situation, from the paucity of large vessels. Extravasations may, however, encircle the macula, and cause an annular defect in the field. With a central loss of sight, there may be adjacent colour-blindness. Sight is rarely altogether lost. Attacks of uræmic amaurosis often accompany and complicate the amblyopia due to the retinal disease.

Pathology.—We know little of the relation between the renal and the retinal affections. The degenerative changes have been ascribed to the tendency to fatty degeneration which renal disease entails; but this scarcely explains their localization in the retina. In chronic granular kidney, the retinal changes have been ascribed to the local vascular degeneration that occurs independently of and may precede similar degeneration in the kidneys. But this opinion is rendered doubtful by the fact that the kidney disease may attain a considerable degree before it is manifested by albuminuria, and by the fact that similar changes result from primary kidney disease. It has been thought that a careful recent microscopic examination of the nervous tissues elsewhere reveals the occurrence of similar changes in them. Gull and Sutton state that an extensive increase in the supporting

tissue of the nerve centres may be found in chronic Bright's disease, and the thickening in the supporting tissue of the retina may be part of this change.

The facts stated on p. 202 render it probable that one mechanism at least by which renal disease excites the retinal changes is the altered state of the blood. The hæmorrhages have been ascribed, with reason, to the double effect of the degeneration in the minute vessels and the increased arterial pressure from the cardiac hypertrophy. It must not be overlooked, however, that in some cases the hæmorrhages seem to be venous in origin, occurring from such veins as are distended in consequence of local pressure of arteries crossing over them. Preponderant neuritis must be ascribed to some special morbid state of the blood. The cerebral symptoms common in these neuritic cases—intense headache, delirium, convulsions—are due apparently to the same cause, the effects of the blood-state.

Complications.—Detachment of the retina is an occasional accident, although not frequent. It may be double and extensive, as in one case under my own observation. The whole of one retina was detached in a case recorded by Davidson.[1] It is apparently due to serous effusion between the retina and choroid. An example of it in slight degree is figured in Fig. 71, which shows that the pigment-epithelium may be detached with the retina.

Hæmorrhage into the vitreous occasionally occurs from the rupture of an extensive extravasation in the superficial layers of the retina. It is always single, and may occur without the patient's knowledge. I have observed the fundus to be distinct one day, and vision good; the next nothing but a black reflection from the interior of the eyeball could be seen, and sight was lost. The damage to vision is usually permanent. It seems sometimes to determine glaucoma.

[1] "Trans. Ophth. Soc.," vol. i. p. 57; *see* also vol. viii. p. 141, where Dr. Anderson relates a case in which very extensive retinal detachment occurred in both eyes of a child with chronic interstitial nephritis, and at p. 128 Sir W. J. Collins mentions this condition in a woman of fifty-three suffering from chronic nephritis.

Embolism has been supposed to occur in albuminuric retinitis (Voelcker), but elsewhere it is extremely rare. Thrombosis occurs, and the signs of embolism may have been due to that cause; on the other hand the contraction of the retinal arteries may simulate that in embolism (*see* p. 197); but there is no corresponding defect of the field of vision in these cases, such as would certainly have been present if embolism or thrombosis existed.

Course.—In most cases the retinal changes persist, some lessening, others increasing, until the patient's death. But sometimes they diminish notably, and the changes may even disappear. This is especially the case when the affection comes on in the course of the chronic disease which results from acute nephritis; in this there may be considerable improvement in the renal affection. In other chronic forms there may be improvement from early and adequate treatment. The effect of purgation in lessening the retinal changes has been often observed, and Eales has remarked that constipation appears to increase the tendency to recurrence or relapse. Improvement is often observed in the albuminuria of pregnancy, a form strangely prone to lead to retinal changes; they improve or disappear when the pregnancy is over. The greatest improvement is in the cases of slight papillitis. Hæmorrhages often disappear, and, if the formation of fresh ones can be prevented, considerable diminution in the retinal disease may result. Even the degenerative changes may pass away, especially those which depend on the presence of the granular bodies in the layer of nerve fibres; most of the white spots shown in Pl. VIII. 2 disappeared. The white specks around the macula lutea nearly always remain. Occasionally, remissions in the retinal affection are observed, although the kidney disease progresses.

Diagnosis.—The recognition of the degenerative changes in the retina is only a matter of difficulty when the changes are slight and limited to the region of the macula. The strong contraction of the pupil, when this part is examined, very often renders the use of a mydriatic indispensable for a thorough exploration.

The aspect of the degenerative form is most closely simulated by the retinal degeneration which results from a neuro-retinitis of wide extent (Pl. VI. 2). It is probable, indeed, that the changes are, to a considerable extent, identical. Œdema of the macula with fibrinous exudation may produce a stellate group of shining spots, quite indistinguishable from those which occur in renal disease, and the diffuse white areas, nearer the disc, may also resemble those seen in the latter form. If the patient has come under observation during the acute period of the inflammation, there will be no question as to the nature of the retinitic change. It will be seen that, as in Pl. VI. 1, the neuritic swelling reaches as far as the neighbourhood of the macula, and that the development of the white spots around the latter is part of the changes in the retina occurring near to, and evidently excited by, the inflammation. If, however, the patient comes under observation at a later stage, the distinction may be less easy, especially if a neuritis from cerebral tumour has been unnoticed until loss of sight occurs during its subsidence.

The signs of one cause or the other are usually, however, sufficiently clear to leave little doubt, after a general survey of the symptoms. Yet this does not always afford so clear a guide as might be expected. A cerebral tumour may be accompanied by a trace of albumen in the urine, as in a child in whom the only symptoms were headache, the retinal changes, and a trace of albumen. On the other hand there may be no symptoms of intra-cranial disease, except headache; this can, alone, hardly be regarded as such, and may accompany the neuritis of albuminuria, as in the case of the patient whose eye is shown in Pl. VII. 3. Lastly, a primary neuritis may occur after diseases which are sometimes attended with albuminuria, notably after scarlet fever.

But attention to the following points will generally suffice for the diagnosis. In the first place, the signs of a considerable preceding neuritis are always present in purely neuritic cases. When failure of sight calls attention to the eye, and the white spots are discovered, there is a prominent

pale swelling over the disc, as in Pl. IV. 3. It is very rare for albuminuric neuritis to leave a swelling of this prominence and pallor. If atrophy results from an albuminuric neuritis, the disc, by the time it becomes pale, is very little above the retinal level, as in Pl. VII. 4. In chronic renal disease, the retinal arteries usually show signs of degeneration. Moreover, renal neuritis never occurs, at least as far as recorded facts and my own observation have gone, except in cases of advanced chronic disease, commonly of contracting kidney, in which the signs of Bright's disease are clear. (*See* also pp. 92—94.)

Hence, in the degenerative changes of neuro-retinitis, of such an extent as to simulate closely the appearance of the albuminuric form, as in Pl. VI. 2, all the features of the change are usually those of past, retrogressive mischief. The disc is atrophied, the arteries evidently compressed, and there are, as a rule, no hæmorrhages. If the papilla

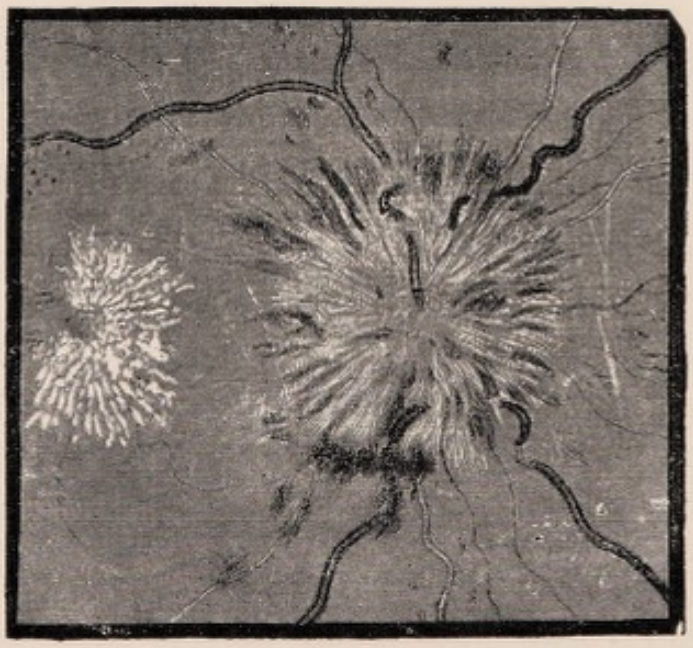


FIG. 76.—PAPILLO-RETINITIS.

From case of cerebral tumour, with appearances at macula closely resembling those common in albuminuric retinitis. (After Edmunds.)[1]

[1] *See* "Trans. Ophth. Soc.," vol. iv. p. 291 and pl. 7.

still presents active inflammation, the degree of this is very great. In the renal form, of corresponding extent, there are signs elsewhere of active processes. The disc may be still inflamed, but only in slight degree, and there are usually hæmorrhages. Lastly, when the retinal degeneration is present as a consequence of neuritis, at the time any difficulty in diagnosis can arise, sight is commonly gravely impaired or even lost. Grave failure of sight is rare in the albuminuric form.

The form in which hæmorrhages and spots of degeneration are combined, may resemble closely the changes in the retina in pernicious anæmia. But in the latter the macular stellate figure is not commonly present, and the degeneration is for the most part connected with, and secondary to, the retinal hæmorrhages. The degeneration does not attain the same extent, and the disc is usually unaffected. The same remarks apply, in the main, also to leucocythæmic retinitis. In the latter, the white spots are much more common in the peripheral portions of the retina than they are in the renal form, and in the latter it is rare to see the pale circular spots surrounded by a halo of hæmorrhage, which are so frequent in leucocythæmia. In the latter the tint of the fundus is commonly very different from that in albuminuria. In both pernicious anæmia and leucocythæmia the independent symptoms of the malady usually leave little room for doubt as to the nature of the retinal changes. The distinction from diabetic changes is described in the following section.

Prognosis.—The presence of advanced retinal change involves an unfavourable prognosis as regards life in cases of chronic renal disease. Such patients seldom live two years, and a large percentage of them die within a few months after the retinal affection is established. The prognosis of the retinal changes themselves has been sufficiently indicated above.

Treatment.—Local treatment is of doubtful value. Good can only be effected by improvement in the blood-state, especially that which is produced by purgation and dia-

phoresis. By this means considerable benefit may often be produced. But this is unfortunately found to be without much influence on the causal disease.

Diabetes.

Diabetes Mellitus.—Defects of sight are common in diabetes (as Bouchardat pointed out many years ago), but changes in the fundus oculi are comparatively rare. The most frequent cause for the visual defect is cataract. Occasionally, considerable amblyopia occurs without ophthalmoscopic changes, probably due to the blood-state and comparable to uræmic amaurosis, although probably the result of a different condition of blood.

Simple atrophy of the optic nerve has been observed in a few cases.

A central scotoma for white and colours has been observed, peripheral vision being normal. The symptom resembles that which results from tobacco, but in some of the cases this cause could be excluded. Examples of this affection have been recorded by several observers.[1] The optic nerves have been examined microscopically by Nettleship and Edmunds, and by others.[2] The changes consist of atrophy of nerve-fibres, with increase of nuclei and connective tissue, in a tract which, at the back of the orbit, occupies the axis of the nerve, and near the eye, the outer portion.

Occasionally retinal changes are visible, first observed by Ed. Jäger[3] and afterwards by Desmarres and Galezowski. A careful study of them has been made by Leber,[4] by

[1] Bresgen, "Centralbl. für prakt. Augenheilk.," Feb. 1881, p. 33; Samelsohn, "Cent. f. prakt. Augenh.," 1882, p. 202; Nettleship and Edmunds, "Trans. Ophthalmological Society," vol. i. p. 124; Moore, "N. Y. Med. Jour." 1888; Schmidt-Rimpler, "Ber. des Ophthal. Ges.," Heidelberg, 1896.

[2] Nettleship and Edmunds, *loc. cit.*; Edmunds and Lawford, "Trans. Ophth. Soc.," vol. iii. p. 160; Fraser and Bruce, "Edin. Med. Journ.," 1896; Schmidt-Rimpler, *loc. cit.*

[3] "Beiträge zur Pathol. des Auges." Wien, 1855, taf. xii.

[4] "Arch. f. Ophth.," xxi. 306.

James Anderson[1] and by Nettleship.[2] They are only seen when the disease is advanced. In such cases of diabetes, albumen is often present in the urine as well as sugar, but the occurrence of these retinal changes is not related to the albuminuria, since they have been observed in many cases in which no albumen was present.

The changes in the retina (Fig. 77) bear considerable resemblance to those of albuminuria, and also to those seen in some cases of pernicious anæmia. Hæmorrhages are conspicuous in many cases, but may be entirely absent, as in the case shown in Fig. 77. They are often situated behind the vessels, and are sometimes of considerable size. They may exist alone or may lead to a secondary parenchymatous retinitis. In one case, figured by Jäger, such retinitis existed in the posterior segment of the eyeball, with obscuration of the disc, concealment of the veins in

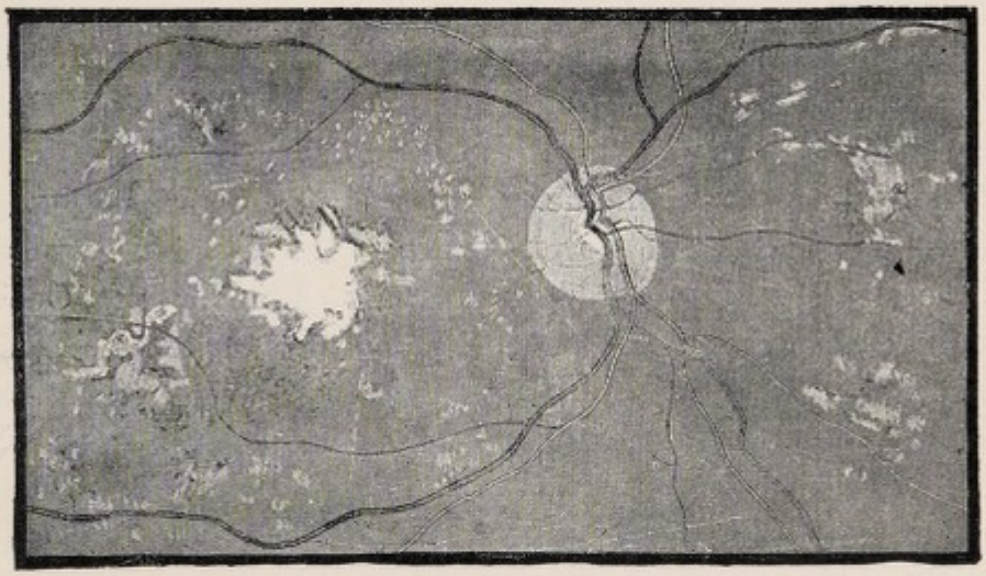


FIG. 77.—OPHTHALMOSCOPIC APPEARANCE IN A CASE OF RETINITIS IN DIABETES. (Nettleship.)

The disc is free from swelling. Scattered about the fundus, especially in yellow spot region, are numerous ill-defined whitish patches (*see* text). In this case there were no hæmorrhages.

[1] "Ophth. Rev.," viii. 1.
[2] "Trans. Ophth. Soc.," vi. 331.

places, a few large whitish spots, and a few striated hæmorrhages, the arteries being unconcealed. White spots of degeneration are frequently present, commonly of moderate size, scattered over the fundus. They are situated in the deeper layers of the retina. They differ from the patches of the albuminuric retinitis in shape, having less tendency to assume a circular form; in colour, having a more dingy shade of white; and in grouping, the stellate arrangement at the macula being rare, although there is a tendency for spots of a rounded or irregular form to occur in this area. Sometimes, however, although rarely, there may be a macular circle of spots, and this in cases in which there is no albumen in the urine. Occasionally a preponderant papillitis may be present (as in albuminuria), and may cause consecutive atrophy and permanent impairment of vision, after the neuritis passed away. The simple atrophy of the optic nerve, which occasionally exists alone, may, in rare cases, accompany the retinal changes (Galezowski).

A marked difference from the forms of retinitis which it most resembles is the frequent association of opacities in the vitreous. They appear to be produced by the escape of blood in small quantities from the retinal hæmorrhages. Leber has traced the development of a complete opacity of the vitreous by repeated hæmorrhagic infiltration. Occasionally, hæmorrhagic glaucoma is the result. In one curious case recorded by Nettleship there were, in several parts of the fundus, capillary loops, apparently from the choroid, perforating the retina, and projecting for several millimetres into the vitreous. In another case he found by the ophthalmoscope numerous small dilatations on a large vein near the disc.[1]

Few microscopical examinations have been made. The chief change found, beyond œdema, was a peculiar hyaloid degeneration of the interna of the arteries, and numerous capillary aneurisms, some of which are shown in Fig. 78.[2]

[1] "Trans. Ophth. Soc.," vol. viii. p. 159; *ib.*, vol. viii. p. 161.

[2] Nettleship and Mackenzie, "Ophth. Hosp. Rep.," ix. p. 150.

The vascular changes afford an explanation of the tendency to hæmorrhage. In this case the vessels of the brain (and of the kidneys and spleen) were similarly affected, and a small cerebral hæmorrhage had occurred.

Both eyes are commonly affected. The disturbance of sight may be slight or considerable. Blindness is usually the result of the extravasations, or of the secondary changes in the vitreous. The retinal changes are never absolutely distinctive from the albuminuric affection, but suggestive indications are the combination with opacity of the vitreous, and sometimes with an apparently simple atrophy of the optic nerve. In albuminuria, such atrophy is very rare.

The retinal affection is apt to relapse, even though temporary improvement be obtained under the influence of dietetic treatment. The advanced stage of the disease at which it occurs, also renders the prognosis unfavourable. The treatment is that for the general disease.

In very rare cases optic neuritis and glycosuria may both be consequences of an organic cerebral disease. The two

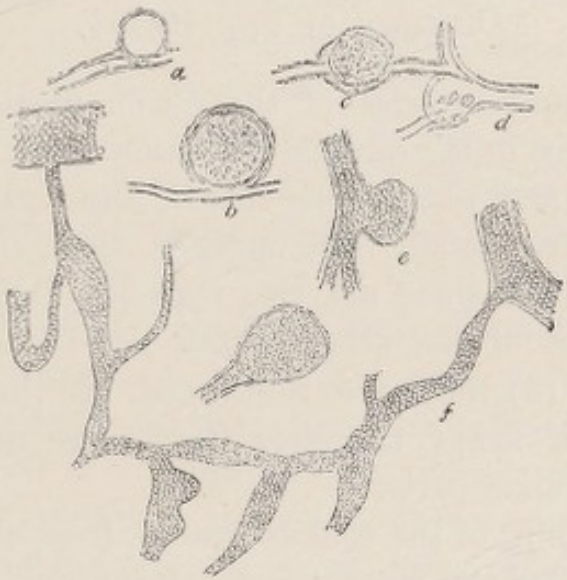


FIG. 78.—CAPILLARY ANEURISMS, AND VARICOSE CAPILLARIES FROM RETINA, IN A CASE OF DIABETES WITH RETINAL HÆMORRHAGES.

They are seen in the course of the vessels (*c*) at their bifurcation (*d*), and also situated laterally (*b*). (× 150.)

symptoms, for instance, existed in a case recorded by Grossmann;[1] the optic neuritis was thought to be due to the diabetes, until other indications of a cerebral tumour developed. After death a tumour was found in the anterior part of the base of the brain. Regarding such rare coincidences it is well to remember that few diseases are mutually exclusive.

Diabetes Insipidus.—In a very few cases of diabetes insipidus, ophthalmoscopic changes have been observed, which have not, however, much analogy with those observed in diabetes mellitus. Atrophy of one optic nerve was observed by Laycock,[2] and double optic neuritis was present in a case described by Van der Heyden.[3] The connection of these changes is probably with the cause, rather than with the condition, of polyuria. It must also be remembered that the polyuria of contracted kidney is sometimes mistaken for diabetes insipidus.

DISEASES OF THE CIRCULATORY SYSTEM.

Diseases of the Heart.

The veins and arteries of the retina participate in any general changes in the circulation which result from diseases of the valves and walls of the heart, although the changes in them are commonly less marked than those in other vessels. For this there are two reasons—(1) Their size is far below that of the other vessels accessible to physical examination; (2) they are influenced by the intra-ocular tension, which keeps the circulation more uniform in the eye than in other parts.

The over-filling of the venous system, from over-distension and dilatation of the right heart, consequent on congenital disease of the pulmonary orifice, on emphysema, and other causes of pulmonary obstruction, and on disease of the

[1] "Berl. Klin. Wochenschrift," 1879, p. 138.
[2] "Lancet," 1875, ii. 242. [3] "Leyden Thesis," 1875.

mitral orifice, may be revealed by an over-distension of the retinal veins, the chief trunks being large, and the smaller veins unduly visible, and therefore apparently more numerous. It seldom affects sight, although transient attacks of amblyopia have been ascribed to it by Galezowski. This condition is most marked in congenital cyanosis. In that disease the retinal veins may be enormously dilated (as in a case figured in the first edition of Liebreich's Atlas), and they afford proof of the degree to which the distension of the venous radicles contributes to the cyanotic tint. Retinal hæmorrhages occurred shortly before death in a case of congenital cyanosis recorded by Stangloneier.[1] In acute venous over-distension, such as occurs during effort, during severe cough, or during an epileptic fit, the venous congestion may also be very marked.

Under-filling of the arterial system, if chronic, such as occurs in aortic obstruction and in mitral disease, is rarely evidenced by a corresponding state of the retinal vessels, no doubt on account of the local influence just mentioned.

Nor is chronic over-action of the left ventricle, if sustained, manifested, as a rule, in the retinal arteries, probably because the most frequent cause of such over-action lies between these minute vessels and the heart, in the small arteries. Exceptions are met with. In exophthalmic goître, in which the over-action of the heart depends on a primary nervous disturbance, and not on an obstruction to be overcome, distension (and even pulsation) of the arteries may be visible.

Sudden over-action of the heart, as from emotion or violent exertion, may also show itself in visible pulsation of the retinal vessels; rarely in the arteries, more frequently in the veins, to which it is transmitted from the arteries.

In aortic regurgitation, pulsation in the veins is common, and pulsation in the arteries is not rare. This depends on the fact that the force of the pulse-wave becomes increased out of proportion to the actual movement of the blood, and

[1] "Inaug. Dissert. Wurzburg, 1878; Nägel's "Jahrbuch für Ophth.," 1878, p. 261.

the conditions which obtain in the larger arteries pass on into the smaller vessels in greater degree than in health, and may overcome the regulating influences of the eye (*see* p. 20). The existence of the valvular lesion has been first suspected from this pulsation.

For the above-mentioned reasons, neither simple dilatation nor simple hypertrophy of the left side of the heart usually affects the size of, or circulation within, the retinal vessels. Dilatation only acts when it involves the right side of the heart in an extreme degree, so as to cause considerable venous congestion. But hypertrophy, when its cause is such as permits it to act on the smaller vessels, may be accompanied by retinal hæmorrhages. In such cases vascular degeneration doubtless precedes rupture and permits it. The hæmorrhages which result may lead to white spots, due to degeneration of the retinal elements, which may persist after the disappearance of the effused blood.

Thrombosis of the central vein occurs in rare cases of heart disease, mitral and aortic (*see* p. 29).

Embolism of the central artery of the retina is an occasional consequence of valvular disease of the heart, and is probably the most common cause of amaurosis associated with cardiac disease—a coincidence which was first noted by Seidl and Kanka in 1846.[1] Its occurrence is governed by the same conditions as those which determine the accident elsewhere. It is most common in mitral disease, especially, like cerebral embolism, in mitral constriction. Its signs have been already described (p. 33).

Transient failure of sight, without ophthalmoscopic changes, sometimes occurs in grave heart disease, and may be unilateral and considerable. To the latter form attention has been called by Nettleship.[2]

Malignant Endocarditis.—In this form, called also "ulcerative endocarditis," attended with fever and septicæmic symptoms, retinal hæmorrhages are frequent and of con-

[1] Canstatt's "Jahresb.," 1846, iii. 115.

[2] "British Medical Journal," Jan. 14, 1879.

siderable diagnostic importance. Choroidal[1] and even conjunctival[2] extravasations have been very rarely seen. The characters of the retinal hæmorrhages are shown in the adjacent figure, which presents the aspect of the fundus in a case of ulcerative endocarditis after chorea. The optic disc is blurred. There are both flame-shaped and striated hæmorrhages in the nerve-fibre layer, and also the very characteristic rounded

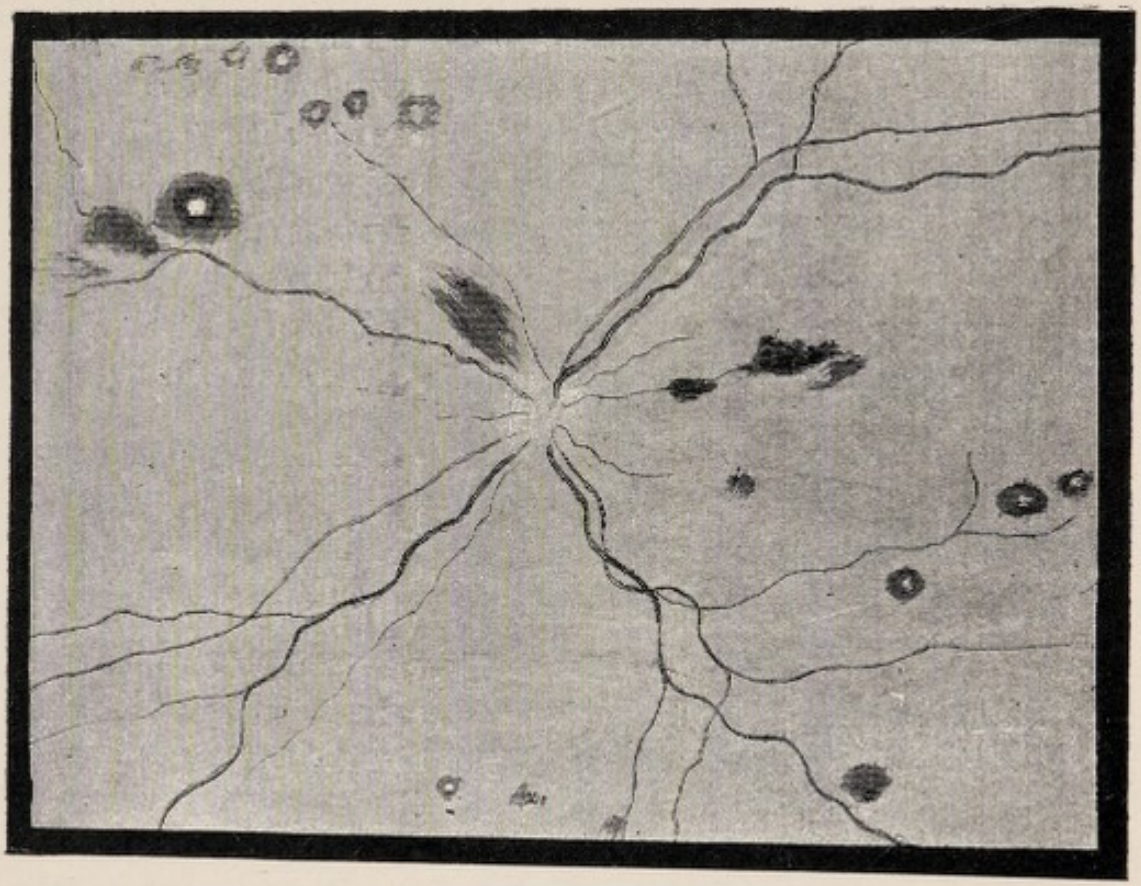


FIG. 79.—ULCERATIVE ENDOCARDITIS AFTER CHOREA. RETINAL HÆMORRHAGES OF VARIOUS SIZES, MANY WITH WHITE CENTRES. SLIGHT PAPILLITIS.

extravasations, with white or pale centres. (*See* also "Septicæmia.") It is probable that organisms pass into the blood and obstruct the minute vessels. They have been observed as the result of endocarditis, produced experimentally, probably without antiseptic precautions. Thus, Rosenbach,[3] in two cases in which endocarditis resulted

[1] Westphal: "Arch. f. Psychiatrie," vol. ix. pt. 3, p. 389.
[2] Michel: "Arch. f. Ophth.," vol. xxiii. p. 113.
[3] "Arch. für Exp. Path. u. Therapie," 1878

from experimental damage to the valves of the heart of dogs, found retinal hæmorrhages, minute, in streaks and dots. In these cases, hæmorrhagic infarcts, with abundant micrococci, were found in various organs. Hyperæmia of the papilla was associated with the retinal hæmorrhages in a case described by Michel. In the optic nerves were found, after death, many dark points, due to capillary embolism and "miliary abscesses"; extravasations into the kidney were associated with bacterial masses. Even panophthalmitis has been met with as a result of embolism from ulcerative endocarditis by Virchow, who has also proved by experiment that embolic obstruction of the minute vessels may cause punctiform extravasations in the retina.[1]

Diseases of the Vessels.

Chronic changes in the vessels may reveal themselves by retinal signs. Those which do occur, the rare coincidence of aneurisms or signs of degeneration of the retinal vessels, with a similar change elsewhere, have been already sufficiently considered in the general account of the changes in the retinal vessels, and of the alterations in albuminuria and diabetes.

DISEASES OF THE BLOOD.

Plethora.

In states of plethora it is said by Jäger that the vessels are large, and the blood-column dark. The changes are not, however, sufficiently marked to be of practical importance.

Anæmia.

Acute Anæmia from Hæmorrhage.—Loss of blood is occasionally followed by affection of vision; the loss of sight may be trifling or complete, transient or permanent, and may come on at the time of the hæmorrhage or not until after several days.

[1] "Arch für Path. Anat.," Bd. x. 1856, p. 179.

It is remarkable that sight is affected much more frequently by spontaneous than by traumatic hæmorrhage. Of the latter, venesection is a more frequent cause of visual loss than accidental wounds or surgical operations. This may be related to the circumstance that in traumatic and surgical cases, the health is less frequently impaired before the loss of blood than in the cases in which spontaneous hæmorrhage occurs, or in cases in which venesection is performed. I am not aware that it has ever been noted in cases of the hæmorrhagic diathesis.

For a valuable compilation of the statistics of these cases we are indebted to Fries.[1] Of 96 cases in which the form of the hæmorrhage was noted, in 34 (35 per cent.) the hæmorrhage was from the gastro-intestinal tract; in 24 (25 per cent.) it was from the uterus, in most cases after childbirth, in a few from menorrhagia; 24 (25 per cent.) were due to the artificial abstraction of blood (21 by venesection, 2 by leeching, 1 by cupping); in 7 cases it was due to epistaxis; in 5 to wounds; in 1 case to hæmoptysis; and in 1 to urethral hæmorrhage.

The loss of sight commonly follows a large hæmorrhage, and especially repeated hæmorrhages, but sometimes occurs after a small one. Now and then it follows immediately on the loss of blood; in 26 per cent. of the cases, the patient wakes from the faint to find himself blind. In 19 per cent. it occurs during the first twelve hours after the hæmorrhage. More frequently there is an interval of two, three, or four days; 33 per cent. occur after the first twelve hours and before the eighteenth day. Fries found that the prompt onset is most common in the cases which occur after venesection, the tardy onset after spontaneous hæmorrhage. Commonly the loss of sight is sudden; rarely, it is preceded by photopsy and pain in the head and back. In one case under my observation, neuralgic pain above the eyes occurred after the (post-partum) hæmorrhage, and lasted for several days after the onset of the blindness. In this case each

[1] "Inaug. Dissert.," Tubingen, "Beilageheft zu den Klin. Monatsbl. f. Augenheilk," 1876. Little has since been added to our knowledge.

previous confinement had been followed by a similar pain, without affection of sight. Occasionally the same individual has suffered from transient affection of sight after hæmorrhage on more than one occasion, as in a case recorded by Samelsohn, in which temporary blindness occurred after each of several attacks of hæmatemesis.

The blindness is commonly double (in 90 per cent.—Fries), rarely one eye being much more affected than the other. In 10 per cent. of the cases one eye is affected exclusively; in 5 per cent. one eye becomes blind and the other is but slightly affected. The loss is often permanent and complete (in 65 per cent.), the pupils being dilated and not acting to light. Partial or complete recovery takes place in about half the cases (partial, 30 per cent.; complete, 20 per cent.). Leber[1] thinks the loss is commonly more complete when the hæmorrhage is from the stomach than when from the intestines or uterus; and this agrees with the conclusions of Fries, that improvement, in spontaneous hæmorrhage, is most common after hæmorrhage from the bowels. Complete restoration of sight has been observed after hæmorrhage from the uterus, from the intestines, from the nose, traumatic hæmorrhage, and venesection; never after hæmorrhage from the stomach. Recovery may be much greater in degree in one eye than in the other. It may be complete in both eyes. When the recovery is partial, the field may be limited, but the limitation varies much in different cases. In the case after childbirth, above mentioned, although the sight of both eyes was lost at first, the right recovered with a normal field; in the left, vision was only $\frac{1}{8}$, and the right half of the field was lost. In one case on record, the permanent loss was in the lower half of each field, and was greater in the left than in the right. In another case (Uhthoff[2]), the right field was restricted above, and the left field was restricted on the temporal side, while the nasal half was lost except in two small islets. The colour-fields were diminished out of proportion to that for white. In a case of

[1] In Graefe u. Saemisch's "Handbuch," vol. v.

[2] "Arch. f. Ophth.," vol. xxvi. pt. i. p. 274.

Samelsohn's, in one eye central, in the other peripheral, vision was left. A central scotoma was also observed by Mandelstamm.[1] Recurrent transient amaurosis marked one case (Leber).

The ophthalmoscopic appearances some time after the onset, have, in rare cases, been normal. In most cases the disc is atrophied with small vessels, as in the case I have mentioned, in which the disc was greyish-white, the arteries much narrowed, the veins small also, and much new tissue about the vessels in the disc. The degree of narrowing of the vessels, and the time at which pallor appears, have varied in different cases.

In the few ophthalmoscopic examinations which have been made early in the history of the cases, there have commonly been signs of inflammation, usually slight, sometimes intense. The slight changes consist in diffuse opacity of the retina with some œdema of the disc, the more intense in a neuro-retinitis with hæmorrhages. The larger the number of early observations the more does it appear that the permanent damage to sight is related in degree to the intensity of the intra-ocular inflammation.

In one case recorded by Hirschberg,[2] three days after the hæmorrhage there was slight opacity of the left papilla; distinct neuritis in the right eye with little swelling; sight nearly normal. Five days later—R., intense neuro-retinitis, V. $\frac{1}{30}$; L., commencing neuritis, V. $\frac{1}{6}$. The sight of the right eye was lost next day. Three weeks later—R. disc as in neuritic atrophy, V. 0; L. disc merely reddish and indistinct, V. $\frac{1}{4}$. Three years later the patient died of cancer of the stomach: the fibres of the optic nerve were found to be replaced by nucleated connective tissue—there was no evidence of hæmorrhage into the nerve sheath. A very similar case has been recorded by Landesberg. The day after a hæmorrhage from the nose, dimness of sight of one eye was complained of, and the ophthalmoscope showed,

[1] "Centralbl. f. prakt. Augenh.," 1879, p. 175.

[2] Hirschberg: "Kl. Monatsbl. f. Augenheilk.," 1877. Supplement, 53—85.

in both eyes, diffuse opacity of the retina with some swelling of the papilla. In one eye the appearances soon lessened, and sight was normal; in the other, a neuro-retinitis with hæmorrhages developed, with ultimate amaurosis. Retinal hæmorrhages and neuro-retinitis were observed by Woinow after the application of four leeches to the uterus. In a case published by Ulrich,[1] the condition was noted a few minutes after an attack of hæmatemesis. The optic discs were pale, and the vessels on its surface presented a normal appearance. At its edge, however, the veins suddenly lost their dark red colour, and became bright red, like arteries. There were numerous hæmorrhages and white spots along the course of the vessels. This condition of the veins gradually disappeared, and in two months the fundi and vision were normal. The same observer has recorded three additional cases with similar ophthalmoscopic appearances.[2]

Förster has recorded a case, in which, twelve days after a hæmorrhage, there was a peculiar white opacity of the retina, with small hæmorrhages around the disc; vessels small, but not as in embolism. There was no affection of sight. The opacity slowly disappeared without neuritis. So in a case seen by Horstmann, three days after hæmatemesis, disturbance of sight occurred (to $\frac{1}{2}$), with slight opacity of the optic nerves and adjacent retina. The changes gradually lessened, and sight became normal. On the other hand, ten days after loss of sight, which occurred seven days after an abortion, Herter[3] found neuro-retinitis with hæmorrhages, quickly subsiding to atrophy; loss of sight permanent in both eyes. Colsmann, however, four days after onset, found only pallor of disc, small arteries, large veins, and no neuritis.

Many theories have been framed to account for the phenomena, but the variety in the changes renders the

[1] "Klin. Monatsbl. f. Augenh.," 1883, p. 183.

[2] "Graefe's Arch. f. Ophth.," 1887, p. 1. *See* "Ophth. Rev.," vol. vii. p. 16.

[3] "Charité Annalen," 1887, p. 525.

appearances very difficult to explain. The theory which has obtained most acceptance is that of v. Graefe, that there is a retro-ocular hæmorrhage situated sometimes near, sometimes far from, the eye. The evidence in favour of this theory is that small retinal hæmorrhages have been seen, that there are sometimes hæmorrhages into other organs in cases of loss of blood, and that in one case there were simultaneous symptoms of a cerebral lesion. But this affords a very inadequate explanation for the cases in which both eyes suffer. Moreover, in those cases which have been examined post mortem, there has been no evidence of such hæmorrhage. To assume, as has been done, that the mischief is at the chiasma, seems unjustifiable, in the entire absence, in all such cases, of other symptoms of mischief at the base of the brain. Förster attributed the slight changes in his case to serous effusion. Horstmann [1] ascribes the symptoms to inflammation in the optic nerves. Ulrich regards papillo-retinitis found after great loss of blood as due to disturbances in circulation in the papilla, and attributes these latter changes to the establishment of an abnormal relation between the blood pressure and that of the vitreous.[2] The appearances noted by him in the veins immediately after severe hæmorrhage (*see* p. 228), he adduces as a proof of these circulatory disturbances. He believes that there is always a slight hindrance to the circulation in the retinal veins, where the latter bend in passing over the edge of the disc, that the influence of the intra-ocular pressure is to increase this hindrance, and that it is still further augmented by the occurrence of any reduction in the blood-pressure. Severe loss of blood, then, produces a condition favourable to venous stagnation by reducing the blood-pressure markedly, while the intra-ocular pressure is not affected or only slightly diminished. Samelsohn thinks that the nervous connection between the stomach and the corpora quadrigemina (lesions of which are *said* to cause gastric hæmorrhage) affords the

[1] "Kl. Monatsbl.," 1878, p. 147.
[2] "Graefe's Archiv," xxvi. 3, p. 80.

best explanation, while von Oettingen[1] believes that he has proved that fatty degeneration of the retinal vessels, quickly following the loss of blood, is the cause of the extravasations sometimes seen.

It is evident, however, that in the majority of cases there are the signs of inflammation, and there is at present no evidence to show that this is not of intra-ocular origin. It seems probable that the mechanism may vary in different cases, and one effect of loss of blood may be upon the retinal elements themselves. The shock to the nervous structures from the anæmia may, in some cases, cause transient loss of function, of sudden or slow onset, and recovery may take place without ophthalmoscopic changes. In other cases no recovery takes place, and atrophy supervenes. In some cases the damage to the nutrition of the retina may lead to a primary inflammation, on the restoration of the normal blood-supply, variable in degree, sometimes slight and general, sometimes most intense in the papilla, where, it may be remembered, inflammation occasionally occurs in chlorosis.

It is probable that more light will be thrown on the pathology of this mysterious accident when physicians are more generally aware of the ocular symptoms which may accompany hæmorrhage, and use the ophthalmoscope in all cases in which the phenomena are likely to occur, since only too many of such cases afford opportunity for post-mortem investigation.

Simple Chronic Anæmia.—Chlorosis.

The colour of the fundus is pale in proportion to the anæmia, but the normal differences are such as to deprive of significance any variations that do not develop under observation. In extreme cases, however, the choroidal pallor may be striking, as in one case that I have seen, in which the red corpuscles were only 26 per cent. of the normal.

[1] "Dorpat Med. Zeitschrift," 1877, Nos. 3 and 4, and Nagel's "Jahresbericht," 1877, p. 239.

The blood in the retinal vessels often presents distinct pallor. The veins are especially pale, often only a little darker than the arteries. When the choroidal pigment is abundant it may be noted that its influence on the apparent tint of the veins is greater than in health; they undergo a greater change in passing from the dark choroid to the pale disc. The veins are also often broad, probably in consequence of the defective distension and consequent flattening in their atonic state by the intra-ocular pressure (*see* p. 10). The reflection from them varies, commonly being broader than normal, no doubt in consequence of the diminished convexity. The arteries are usually narrow, not merely in comparison with the veins, but absolutely; their pallor is much less noticeable than is that of the veins. The reflection from them may also be broad. Spontaneous pulsation in the retinal arteries has been occasionally observed by Becker[1] in chlorotic girls. Schmall[2] found it in 20 out of 55 cases of chlorosis. Hæmorrhages are said to occur, but are certainly very rare, in simple anæmia, and probably only take place where there is a great absolute deficiency in the number of red corpuscles.

Optic neuritis occasionally occurs in chlorotic girls, apparently as a result of the anæmia. I have recorded two cases[3] (one is figured in Pl. V. Fig. 5), and many others have been described. In each case the anæmia was very great, the hæmoglobin being reduced, out of proportion to the corpuscles, in one to 30, and in the other to 38 per cent. One case suffered from a relapse of neuritis on a recurrence of anæmia. In each case the improvement was most rapid under the influence of iron, and physical rest. The degree of neuritis was slight in the first case, but very considerable in the second. In the case figured in Pl. VI. 1 and 2, the neuritis must probably be ascribed to the same cause. The patient was watched for two years after the subsidence of the neuritis, and, except for an occasional headache, there

[1] "Klin. Monatsbl.," Jan. 1880, p. 1.

[2] "Graefe's Archiv," xxxiv. i. p. 37; "Ophth. Rev.," 1888, p. 268.

[3] Gowers, "Brit. Med. Journ.," 1881, i. 793.

was no other symptom to suggest intra-cranial disease. Neuritis from cerebral disease hardly ever develops with such extreme rapidity as in this case; there was an entire absence of cerebral symptoms. Iodide of potassium was first given, but no improvement occurred until iron was substituted, too late, unfortunately, to prevent partial atrophy. This is a common history in such cases. Hirschberg first noted the occurrence of optic neuritis in chlorosis, and an instance, in a girl of sixteen, quickly cured by the administration of iron, was recorded (in 1879) by Bitsch.[1] Several others have been recorded.[2] In one there was double papillo-retinitis, with a marked stellate figure at each macula.[3]

It is worthy of note that all the patients presented a slight degree of hypermetropia. It is generally admitted that this condition is capable of causing slight congestion of the disc, and if so, it is possible that, in these cases of chlorotic neuritis, the hypermetropia may help in setting up the changes in the papilla which, in the special blood-state, progress to a much more intense degree than they would otherwise attain.

In the early treatment of such cases, it is essential to combine physical rest with the administration of iron. The deficiency in the oxygen-carriers is such that the vital organs are imperfectly nourished if the oxygen is used for muscular exertion. Gentle massage may be safely employed, and the addition to the air breathed of a small quantity of oxygen for five minutes every hour is a useful adjunct.

Progressive Pernicious Anæmia.

In pernicious anæmia the tint of the fundus and the appearance of the arteries and veins are such as are seen in the most intense cases of simple anæmia. The rather narrow arteries and broad pale veins are seen in Pl. IX. 1,

[1] Bitsch, "Klin. Monatsbl.," April, 1879, p. 144.
[2] R. Williams, "Brit. Med. Journ.," 1884, i. 10.
[3] Beaumont, "Trans. Ophth. Soc.," vol. x. p. 149.

from a case published by Stephen Mackenzie.[1] The figure shows also that which is a characteristic feature in pernicious anæmia, the tendency to hæmorrhage. Common in other situations, it is far more frequent in the retina than elsewhere. Of sixteen cases examined by Quincke, retinal hæmorrhages were absent in one only. In thirty cases examined by Horner, extravasations were present "almost without exception." The extravasations are often, as in the figure, numerous, and more or less striated or flame-shaped, from their situation in the layer of nerve-fibres. They are usually most abundant around the optic nerve entrance. They are frequently asscciated with white spots and areas, due in part to leucocyte-like cells, in part to degeneration in the disturbed retinal tissues, varicose enlargement of the nerve-fibres, giving rise to finely granular, spherical, and fusiform bodies. Homogeneous ("colloid") and finely granular masses have also been found in the inter-granule layer.[2] Occasionally a pale spot may occupy the centre of a small hæmorrhage. In such a case Manz[3] found the pale centre to consist of round colourless cells, some enclosed in a capsule. He found also ampulliform and sacculated dilatation of the capillaries (no doubt similar to those seen in Fig. 78, p. 219, from diabetes). Some of these were empty, others contained a granular material, others red blood corpuscles or colourless cells. He supposes that the capsule found to enclose the pale cells within the hæmorrhages was really the wall of such a capillary dilatation. A stellate arrangement of white specks around the macula lutea was seen by Quincke in one case, but is certainly rare in this disease. He also once observed œdema of the retina, the vessels being dimmed by a bluish-white cloud. The optic disc is usually normal, but its edges may be blurred, and optic neuritis may, in rare cases, be present in excess of the other retinal changes.[4]

[1] "Lancet," Dec. 7, 1878.

[2] Uhthoff: "Klin. Monatsbl.," Dec., 1880.

[3] "Centralbl. für d. Med. Wiss.," 1875, p. 675.

[4] S. Mackenzie: "Lancet," Dec. 7, 1878.

The hæmorrhages are often quickly absorbed, lasting only a few weeks. They cause no disturbance of ordinary vision except when located near the macula lutea. It is probable that a relation exists between a certain degree of diminution of corpuscles and the occurrence of these hæmorrhages. In one case the hæmorrhages appeared when the corpuscles fell to 27 per cent. of the normal, and increased with the progressive fall of the corpuscles, which before death were only 12 per cent., and the hæmoglobin 8 or 9 per cent. of the normal.[1] They are rarely absent when the corpuscles fall below 25 or 20 per cent.

Scorbutic Anæmia.—A form of anæmia which may be thus distinguished, appears to be a distinct variety of pernicious anæmia. It is characterized by the same progressive pallor and other effects of the deficiency of blood corpuscles, but differs in the occurrence of an affection of the gums resembling that met with in scurvy, and in extravasations into the skin. There may also be other cutaneous rashes, such as are met with in cachectic conditions. Hæmorrhages may occur into the retina just as in the ordinary form of pernicious anæmia. It appears not to be in any degree due to deficiency in vegetable food, but to be occasionally produced by abstinence from meat.

An example of this form has been described by Dr. S. Mackenzie.[2] The patient was a lad, aged eighteen, who had had syphilis, but no cause for the blood-disease could be traced. The symptoms were swelling of the gums, enlargement of the glands beneath the jaw, petechial hæmorrhages in the skin, hæmorrhage from the gums, and profound anæmia. Vomiting preceded death. In the retina were abundant fusiform hæmorrhages, gradually increasing in number until there were twenty or thirty in each retina, some as large as the papilla. Ultimately general retinal œdema occurred. The corpuscular richness of the blood gradually decreased, during two months the patient was

[1] S. Mackenzie: "Trans. Ophth. Soc.," vol. i. p. 48.

[2] Ibid., p. 51.

under observation, from 51 to 13 per cent. of the normal. The coloured corpuscles varied in size, some presenting a fissured appearance. The colourless corpuscles were not in excess, except to a slight degree towards the close. They were small and spherical. The hæmoglobin was reduced out of proportion to the corpuscles. No treatment, dietetic or medicinal, appeared to influence the course of the disease. After death, hæmorrhages were found in the lungs and on the surface of the heart.

In a case of my own, the symptoms were very similar. There were the same progressive anæmia, swelling of the gums, cutaneous and retinal extravasations, and hæmorrhages found after death in the lungs and heart. In this case, also, the patient had for a long time abstained almost entirely from animal food, taking plenty of vegetables. There were also, in addition to, and accompanying the extravasations, papules with infiltration of the adjacent skin. After death a peculiar change was found in the periosteum of some of the bones.

Leucocythæmia.

In all cases of leucocythæmia in which the change in the blood is considerable, the retinal and choroidal vessels are remarkably pale. An orange tint of the choroid has been described. The retinal veins appear broad as well as pale. This apparent increase in width is sometimes very great (Fig. 80), and is probably due to atony and flattening rather than to passive distension. They are often tortuous. The central reflection may at first be broad and indistinct; ultimately, in the large tortuous vessels, a very narrow, almost white, reflection appears. The retinal arteries have a colour inclining to orange, and in extreme cases they become small.

Besides these appearances, there are, in a considerable number of cases, actual changes in the retina. These vary greatly in different cases, and rarely present the appearance described by their discoverer, Liebreich, as "leukæmic retinitis." They are almost confined to the splenic variety,

and are usually double, one eye being often more affected than the other.

The commonest change is the occurrence of retinal hæmorrhages. The tendency to hæmorrhage in this disease is very marked, and statistics[1] show that the most common recorded seat of extravasation into the tissues is the subcutaneous cellular tissue. The constant use of the ophthalmoscope shows that hæmorrhage into the retina is as frequent, if not more so. The tendency to retinal hæmorrhage is apparently greater in leucocythæmia than in simple anæmia, for it occurs with a higher percentage of red corpuscles. I have twice met with it when the blood contained 50 per cent. of red corpuscles. One of these cases is figured in Pl. IX. Fig. 2. The hæmorrhage encircles the fovea centralis in a

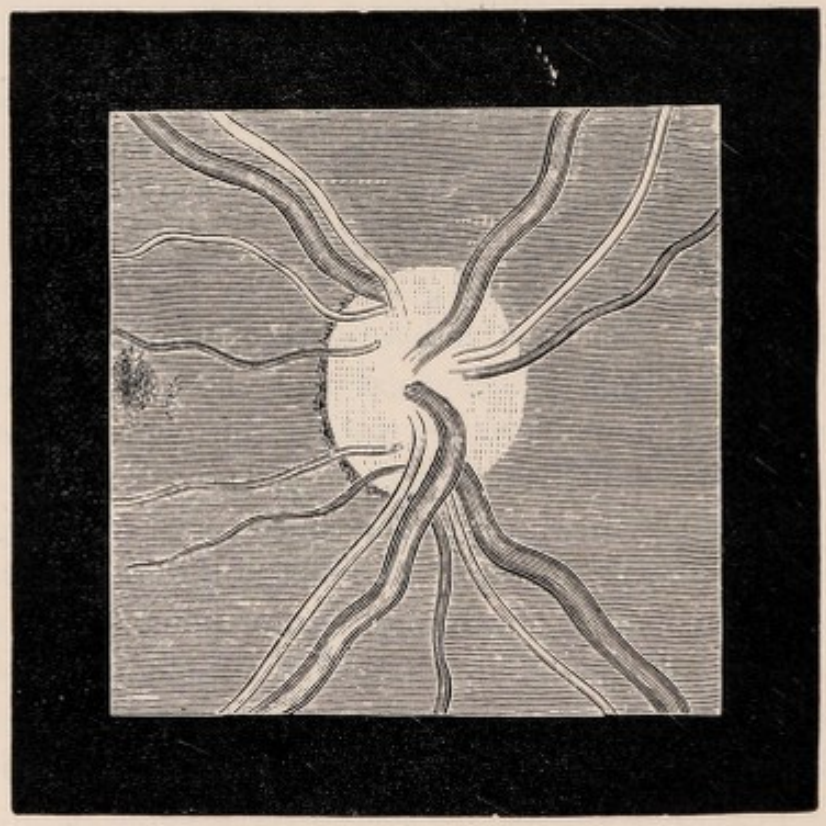


FIG. 80.—BROAD RETINAL VEINS AND NARROW ARTERIES.
From a case of leucocythæmia.

[1] Gowers: Art. Leucocythæmia, "Reynolds' System of Medicine," vol.v. p. 257.

curious series of extravasations, and several smaller ones lie adjacent. Often the hæmorrhages are widely scattered, and generally striated, but sometimes rounded. The hæmorrhages are in these cases usually in the nerve-fibre layer, but a large extravasation may infiltrate the whole thickness of the retina. They are often most numerous in the periphery of the retina, and these are generally rounded in form, sometimes with pale centres. When the excess of white corpuscles is considerable, the effused blood has a pale chocolate tint as in other situations. Extravasation may take place into the substance of the papilla, or into the vitreous.[1]

Besides the hæmorrhages, white or yellowish spots are commonly present, often most abundant in the periphery, or near the macula lutea. These are sometimes irregular, but often rounded, and edged by a halo of extravasation. When large, they are sometimes distinctly prominent, and may be as much as 2 mm. in diameter (Reincke). They consist commonly of leucocytes, similar to the leucocytes of the blood, but they have been regarded as lymphoid growths such as occur in other organs. In rare cases, actual growths of some size have been met with, but it is doubtful whether the smaller spots are of this nature. It is common for the pale corpuscles to be aggregated in the middle of an extravasation. In some cases the white spots are due to degeneration of the retinal elements. The capillaries are full of white corpuscles, and it seems probable that these spots arise by the escape of the corpuscles by diapedesis or by rupture. Larger vessels have been found almost filled with leucocytes, and in places surrounded by them in an annular arrangement. In places varicosities are thus filled, and leucocytes massed around them so abundantly as to constitute visible white spots.[2] In one case Saemisch found an irregular thickening of the inner granule layer, in some places extending into the ganglion-cell layer. He attributes the thickening to escaped leucocytes, which are indistinguishable from the corpuscles of the granule layer. Poncet

[1] *Vide* Perrin and Poncet's "Atlas," pl. 65.

[2] Murakami, Bericht 29 Vers., "Ophth. Ges.," 1901, p. 257.

has found a similar infiltration extending, not only into all the layers of the retina, but also into the substance of the optic nerve. Swelling of the nerve-fibres was the cause of small white spots in a case described by Deutschmann.[1] The capillaries of the retina are often dilated and varicose (as Fig. 78, p. 219). The lymphatic sheaths of the vessels may be filled with white blood corpuscles, and may be visible as a white border along them; towards the periphery some ended in fine white lines in a case described by Grunert.[2]

Occasionally a diffuse opacity of the retina is met with, said by Roth to be due to a thickening of the vertical fibres of the retina, but probably sometimes due to œdema—of the ganglion-cell layer in the case described by Deutschmann,[1] of both ganglion-cell and nerve-fibre layers in a case examined by Oeller,[3] in which these layers were twice the normal thickness. When considerable, it is in part due to a diffuse infiltration of leucocytes, as described by Poncet and Oeller. This opacity, with some swelling and great tortuosity of the veins, was the chief appearance in a woman, age thirty-five, under my own observation. The discs were clear, but a slight diffuse opacity of the retina existed, chiefly near the disc and somewhat striated on direct examination. Towards the periphery of the retina, a few small white spots were seen, and one small hæmorrhage.

In a case recorded by Hartridge, the retinal veins were greatly engorged and the disc swollen from œdema, but no retinal hæmorrhages were observed.[4] An enormous swelling of both papillæ, was observed by Heinzel[5] in a child, completely concealing the discs. The retina presented here and there a little opacity. The vessels were

[1] "Kl. Monatsbl. für Augenheilk.," 1887, p. 231. The microscopical appearances have also been well described (with drawings) by Edmunds, "Trans. Ophth. Soc.," vol. x. p. 157.

[2] "Centralbl. für Augenkr.," 1901, p. 225.

[3] "Arch. f. Ophth.," xxiv. 1878, pt. iii. 241.

[4] "Trans. Ophth. Soc.," vol. xiii. p. 72 (with ophthalmoscopic drawing and also sections of the retina).

[5] "Jahrbuch für Kinderheilk.," 1875, p. 346.

bordered by pale lines of variable width. Numerous hæmorrhages appeared and disappeared in each retina during the time the case was under observation, but the pathological condition passed away in four weeks, and the fundus remained normal until death. The aspect was that of mechanical congestion with consequent œdema and ecchymoses, and a similar condition was observed in the conjunctiva and the skin.[1]

In cases of general changes in the retina, the optic papilla has been found swollen (to ·9 mm.—Oeller) in consequence of leucocytal infiltration and œdema. In other cases papillitis has been found, sometimes great, with hæmorrhages on it.[2]

In some cases of leucocythæmia, the hæmorrhages may be so numerous as to give to the changes the aspect of a hæmorrhagic retinitis. A large extravasation behind the retina may burst through into the retina, and an extravasation in this may even escape into the vitreous, and has been said to cause secondary glaucoma.

Besides the changes which appear related to the blood-state, the complication of kidney disease may lead to changes, identical in appearance and structure with those met with in cases of primary renal disease.[3]

The degree to which the changes interfere with sight depends on their extent and position. If abundant they cause considerable amblyopia; if slight the vision may be unimpaired, and the retinal changes may easily be overlooked, unless systematic examination is made with the ophthalmoscope. In one case[4] the patient sought advice in consequence of seeing a red balloon constantly before

[1] The ophthalmoscopic appearances may have been due to partial thrombosis in the orbital vein, the anastomoses with the facial ultimately sufficing to restore the normal circulation. Venous thrombosis is common in this disease.

[2] Murakami, *loc. cit.*, Bondi, "Prag. Med. Wochenschr.," 1901, p. 311; Grunert, *loc. cit.*

[3] *See* Poncet: Perrin and Poncet: "Atlas," pl. 66.

[4] Hirschberg: "Centr. f. prakt. Augenh.," 1887, p. 97; "Ophth. Rev., 1888, p. 12.

his right eye, and the ophthalmoscope showed a large hæmorrhage of corresponding shape in the macular region with numerous small ones scattered over the fundus. A month later, an exactly similar hæmorrhage occurred in the left eye, giving rise to a similar subjective appearance. When situated near the macula, central vision is much impaired; in the case figured (for instance, Pl. X. 2), sight was very dim, but not lost. Occasionally the disturbance of the retinal elements leads to curious changes in vision. Double exophthalmos, from a lymphoid growth in both orbits, has been described by Leber[1] and Chauvel.[2]

Sometimes the choroid is found infiltrated with leucocyte-like cells, and its vessels may be, at the same time, greatly dilated. From these two changes, in Oeller's case, the choroid, near the outer side of the disc, was swollen to eight times the normal thickness. It was difficult to say whether the leucocytes were free or were contained in enormously dilated vessels. Poncet has figured an infiltration of the iris with leucocytes, supposed to indicate a leucocythæmic iritis.

Purpura.

The tendency to rupture of vessels in purpura leads to retinal as well as to subcutaneous extravasation. Retinal hæmorrhages are certainly common, perhaps invariable, in the severer forms of the affection. Cases have been recorded by Ruc,[3] Stephen Mackenzie,[4] and others. The extravasations are for the most part striated, adjacent to vessels, and most abundant in the neighbourhood of the optic disc. The occurrence of extravasations into the retina therefore indicates a severe, but not necessarily fatal, degree of the disease. The hæmorrhages may disappear, and be replaced by others, and yet the patient may ultimately recover. Hæmorrhage

[1] "Arch. f. Ophth.," vol. xxiv. 1878, p. 295.
[2] "Gaz. Hebd.," 1877, No. 23.
[3] "L'Union Méd.," 1870.
[4] "Med. Times and Gaz.," 1877, 292.

into the choroid has been also met with. Those in the retina, if numerous, may cause considerable amblyopia, and, if near the macula lutea, may gravely damage central vision.

Scurvy.

Retinal hæmorrhages have been found in scurvy, but less commonly than in purpura; perhaps because they have seldom been looked for. They are, as in purpura, commonly in the neighbourhood of the optic nerve. In one case recorded by Wegscheider,[1] numerous small extravasations into the brain co-existed. The signs of thrombosis in the vein have also been met with.[2]

DISEASES OF THE LUNGS.

Pulmonary affections rarely cause ocular troubles. Emphysema may lead to mechanical congestion of the venous system generally, which may be conspicuous in the eye. The same influence has been ascribed, but on very doubtful grounds, to phthisis, in which amblyopia occasionally occurs. Tubercles in the choroid may be met with in cases of general tuberculosis, but not when the tubercular affection is confined to the lungs. Acute pneumonia is said, in one case, to have been associated with neuro-retinitis, but meningitis is sometimes coincident, as an effect of the same organisms, and these may possibly cause obstruction in the smaller retinal vessels. Fraenkel,[3] in one case of pneumonia, found four or five round whitish spots, one-third the diameter of the papilla, which he regarded as due to embolism by micro-organisms. The eyes are seldom carefully observed in cases of pneumonia. Febrile bronchial catarrh, with cyanosis, was observed by Litten[4] to be accompanied with neuro-retinitis,

[1] "Deutsche Med. Wochenschr.," Nos. 17 and 18, 1877.
[2] Deneg, "Munich Med. Wochenschr.," 1895 p. 893.
[3] "Arch. f. Oph.," Bd. 48.
[4] "Charité Annalen" for 1876. Berlin, 1878.

in and around the papilla, of gradual development, and with numerous extravasations, some with white centres, near the equator of the eye. Many of the extravasations were regularly arranged, and situated upon small veins; and he suggests that the changes were probably set up by the great distension of the veins, but both were more likely the effect of micro-organisms. The retinal affection subsided with the bronchitis.

DISEASES OF THE DIGESTIVE ORGANS.

The occasional effect of hæmorrhage from the stomach and intestine has been already described. Galezowski[1] associated atrophy of the optic nerve, in some cases, with gastric troubles. But the significance of such an association has been altogether changed by our knowledge of the frequency of gastric crises in tabes, to which such cases were probably related.

Constipation is regarded by Eales[2] as having been influential in causing retinal hæmorrhage in a series of cases observed by him. All were young men, with slow pulse and high arterial tension, and two had a slight trace of albumen in the urine. The extravasations were chiefly in the left retina, roundish in form as if in the deeper layers. He speculates that the constipation may have been due to, or accompanied by, vaso-motor spasm in the abdominal vessels, sufficient to cause a general increase of arterial tension. But the facts still need confirmation.

Jaundice.—The changes in the blood from jaundice, from any cause, may occasion retinal hæmorrhage.[3] Jäger says that the blood in the vessels may have a yellowish tint, but the appearance is probably due to a tint in the media, such as in rare cases causes yellow vision.

[1] "Journ. d'Ophtalmologie," March, 1872. "L'Union Méd.," 1876, i. p. 368.

[2] "Birmingham Medical Review," July, 1880, p. 262.

[3] Litten: "Zeitsch. f. Klin. Med.," v. i. p. 319.

DISORDERS OF MENSTRUATION.

Sudden suppression of the menses has been observed to be followed by acute optic neuritis, such as accompanies meningitis, and often attended with unpleasant sensations in the head. The occurrence of the neuritis is probably analogous to other changes in the nervous system, such as acute myelitis, met with in the same association, the nature of which is quite uncertain.

In chronic menstrual irregularities, optic neuritis, of chronic course, has been found, and occasionally other disturbances, such as retinal hæmorrhages. It is probable that in most cases of this character the two conditions—the ocular and menstrual disturbance—are related to some common cause.

Loss of sight, sometimes with neuritis, after uterine hæmorrhage, has been already mentioned (p. 225).

The albuminuric retinitis of pregnancy has been already mentioned. Its presence or absence in cases of albuminuria during pregnancy has been said by Silex[1] to furnish important prognostic indications. He states that he has found this to be true even of the altered reflection from the retinal arteries. Under the title "amaurosis by reflex irritation," Landesberg[2] has related two cases of amblyopia, with limitation of field, coming on in pregnancy. In one case the affection of sight quickly passed away; in the other it was accompanied by hemianæsthesia, and cyclitis developed, which necessitated enucleation. A similar condition has been observed in association with menorrhagia. The nature of these cases is obscure. Although most cases of affection of sight from pregnancy are produced through the agency of albuminuric retinitis, it would seem that a more direct influence is sometimes exerted. Loring[3] has described a mysterious case in which each of three pregnancies was

[1] "Berlin Kl. Wochenschr.," 1895, No. 18.
[2] "Arch. f. Ophth.," xxiv. pt. i. p. 161.
[3] "New York Med. Jour.," 1883, p. 59.

accompanied with failure of sight, the first two in the outer half of one field, the last in the outer half of each, with general impairment of vision.

DISEASES OF THE SKIN.

It has been said by some, especially by Mooren, that general skin diseases may be accompanied by inflammation of the retina and papilla; that eczema of the head may be accompanied by optic neuritis, ending in atrophy. The statement has, however, received no confirmation, and the relation of the two conditions is exceedingly doubtful. If such a sequence occurs, it is possible by a local orbital cellulitis. The suppression of a customary cutaneous discharge has also been said to cause neuritis, but a relation is improbable. The coincidence of effects of constitutional states, such as gout, should be borne in mind.

CHRONIC GENERAL DISEASES.

Tuberculosis.

The grey granulations which constitute the anatomical lesion in tuberculosis may form in the vascular structures of the eye, chiefly in the choroid, rarely in the iris and retina. When present in the fundus, they may readily be seen with the ophthalmoscope.[1]

Tubercles in the choroid (Fig. 81) appear as white, yellowish-white, or reddish-yellow spots, usually isolated, and more or less round in form. They are palest in the centre, and often reddish in their outer portions, and the peripheral redness passes gradually into that of the adjacent choroid. They commonly develop in the substance of the choroid,

[1] That tubercles occurred in the choroid as a post-mortem observation has long been known. They were described by Autenrieth in 1808. They were first observed with the ophthalmoscope by Ed. Jäger in 1855.

and the pigment and vessels atrophy before the growing nodule, first and more completely, at the centre, so that the diameter of the tubercle, on section, may be found to be twice or three times as great as that of its exposed portion (Fig. 82). In size they vary from one-third of a millimetre to two or two and a half millimetres, and appear, to the ophthalmoscope, from about a fourth to a tenth to half the diameter of the optic disc, or even more. The larger sizes are rare. Occasionally several are aggregated together to form a mass which may exceed the size of the disc—seven or eight millimetres in diameter. These larger masses project considerably into the eye. Slight prominence may commonly be recognized in all the larger tubercles, and assists the diagnosis. The smaller ones may resemble spots of choroidal exudation or atrophy. From the former, their rounded shape and yellowish tint help to distinguish them. From atrophy, the tint, regular form, concealment of the choroidal vessels, and the (common) absence of any adjacent pigmentary

FIG. 81.

FIGS. 81—83.—TUBERCLES OF THE CHOROID FROM A CASE OF ACUTE MILIARY TUBERCULOSIS IN A CHILD.

FIG. 81.—The front of the eye has been removed, and the retina is drawn over to the left. Six tubercles are seen, varying in diameter from 1 to 4 mm. (× 2). They are prominent in the darker periphery, when the pigment-epithelium over the choroid is intact, and where it has disappeared the tubercle appears white.

disturbance are sufficient distinctions. They are plainly behind the retinal vessels. In structure they consist of the same lymphoid cells as constitute such tubercles elsewhere.

The cells are distinct in the periphery, degenerated in the centre (Fig. 83). I have found extravasations of blood in the substance of one. The tubercles are situated chiefly in the posterior part of the globe, not far from the optic nerve entrance. Usually only three or four are present; sometimes, however, as many as twelve or twenty or even fifty (Cohnheim). They may form rapidly, and, according to Stricker, may become recognizable in from twelve to twenty-four hours. But it must be remembered that they attain some size before they disturb the epithelium, and the partial removal of this may rapidly increase their apparent size.

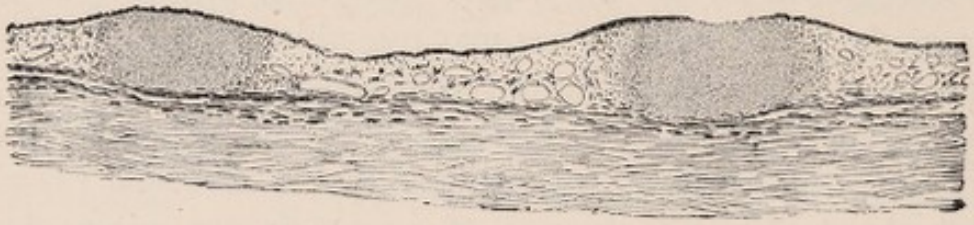


FIG. 82.

FIG. 82.—Section of two of the smaller of these tubercles. They occupy the whole thickness of the vascular layer, pushing forward the pigment-epithelium, and, in the case of the right hand one, breaking through it. (× 30.)

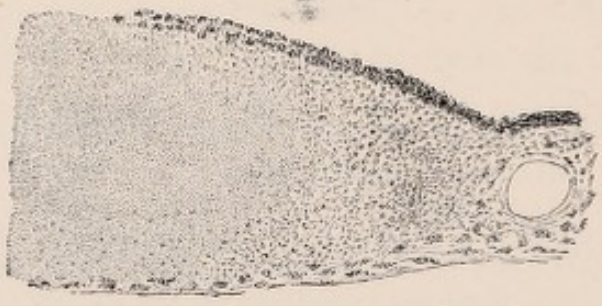


FIG. 83.

FIG. 83.—One half of a tubercle, which has caseated. Above it, in the centre, where it has a granular aspect, the pigment-epithelium has disappeared towards the centre, and below are the deeper pigment cells of the choroid. (× 100.)

The tubercles begin as minute points, "masses of lymphoid cells," and develop in the substance of the choroid, advancing towards the retina until they cause atrophy of the structures above them, and become ophthalmoscopically visible. When the tubercles are large, and grow rapidly, some retinal pigment frequently remains on their surface.

Choroidal tubercles occur in both children and adults, and in the chronic as well as in the acute forms of tuberculosis, but are most frequent in the acute forms. They are practically confined to the cases in which tubercle is widely distributed. Their actual frequency in these cases cannot yet be stated. It is evident, from the rapidity of their appearance, that repeated ophthalmoscopic examination is necessary to exclude their occurrence. Cohnheim described them as very commonly to be found after death, and Litten found them (post-mortem) in thirty-nine out of fifty-two cases. According to most observers they are much less frequently to be seen during life than these figures would suggest. In this country, at any rate, they have not very often been seen. As a rule, they have been observed only when the disease has reached an advanced stage. Exceptions to this have been recorded; in one case they were present before fever or other symptoms of the disease were present;[1] in another, six weeks before the commencement of tubercular meningitis.[2] In such a case they may afford great assistance to diagnosis. Their presence, thus, is of value as evidence of general tuberculosis, especially when the diagnosis of acute tuberculosis from other acute febrile conditions is difficult; their absence is of no significance. Tubercles of the choroid often coincide with tubercular meningitis, but they have also been met with when the membranes were free from tubercle. It is remarkable that the characteristic bacilli have not been found in them, in some cases in which they were readily detected in the

[1] Fraenkel: "Berl. Kl. Wochenschr.," 1872; "Jahr. f. Kinderheilk.," Bd. ii.

[2] Steffen: "Jahrbuch für Kinderheilk.," 1870. Little attention seems to have been paid to them during recent years.

meningeal tubercles.[1] The fact is probably without significance. In a case of choroidal tubercle, where bacilli could not be demonstrated, the inoculation of a guinea-pig with the crushed tubercle produced general tuberculosis.[2]

Frequently choroidal tubercles cause no symptoms. Disturbances of sight are, however, sometimes found, and these may be marked and permanent when the macular region is involved. In one case (Manz[3]) tubercular growths perforated the sclerotic and appeared on the exterior of the eye.

Occasionally, although rarely, a tubercular mass develops in the deeper structures of the eye, quite similar to the masses of the same nature which are found in the brain. I have seen one case in which such growths were associated in these two situations; optic neuritis was also present. Chiari[4] has described a tubercular mass, with granulations in the neighbourhood, which infiltrated the greater part of one optic nerve and invaded the eye, appearing as a white prominence in the position of the papilla, five disc-diameters in width.

Choroidal tubercles were found by Cohnheim in a guinea-pig rendered tubercular by inoculation.

Retina.—The occurrence of tubercles of the retina has been recorded in very rare instances. The aggregations of lymphoid cells which may occur in the nuclear and molecular layers, adjacent to an inflamed disc in tubercular meningitis, have been regarded as such, but their tubercular nature is doubtful. Unequivocal tubercles in the retina (even containing giant cells) have usually been associated with tubercular growths in almost all the structures of the eye (Perls, Manfredi), in rare cases with a tubercular papillitis only (Weiss, Sattler). In the case of

[1] Lawford ("Trans. Ophth. Soc.," vi. p. 348) failed in four out of six cases. He gives a summary of the results obtained by various observers; but Haab ("Klin. Monatsbl. für Augen.," 1884, p. 391) found them almost invariably.

[2] Alexander: "Centralb. f. Augenh.," 1884, p. 161.

[3] "Klin. Monatsbl.," Jan. 1881, p. 26.

[4] "Wien. Med. Jahrbuch," 1877, p. 559. Sattler: "Arch. f. Ophth.," Bd. xxiv. pt. iii. p. 127. *See* also Gunn, "Trans. Ophth. Soc.," vol. xxi. p. 87.

tubercle of the optic nerve referred to above, the optic papilla was the seat of a large mass of caseating tubercle, and miliary tubercles were scattered through all the layers of the adjacent retina.[1]

Tubercles in the eye are, as already stated, almost invariably part of general tuberculosis. In one case, however, they were found in all parts of the eye, although not found elsewhere.[2]

Local tubercular tumours within the cranium may give rise to ophthalmoscopic changes, producing optic neuritis, as do other cerebral tumours. In the rare cases in which tubercular masses are situated in the intra-cranial portion of the optic nerves,[3] or in the chiasma,[4] there has been a corresponding affection of sight (with or without signs of descending neuritis). The inflammation which accompanies the formation of tubercle in the membranes may also be accompanied by optic neuritis (*see* p. 164).

Morbid Growths.

Morbid growths other than tubercular are seldom present at the same time in the eye and brain. Cancer of both choroids has, however, been observed by Puts, secondary to a primary epithelioma of the lung.

Syphilis.

Acquired Syphilis.—The syphilitic diseases of the eye, during their active stage, commonly come under the care of the ophthalmic surgeon. Their effects on the fundus oculi are often encountered by the physician in his own work, and furnish him with useful information. A knowledge of these changes is, therefore, of great importance.

Iris.—Although not strictly an ophthalmoscopic sign, the

[1] "Arch. f. Ophth.," xxiv. pt. iii. p. 150.
[2] Weiss: "Arch. f. Ophth.," xxiii. pt. iv. p. 57.
[3] Cruveilhier: "Anat. Path. Gén.," 1862, Bd. 4.
[4] Hjort: "Kl. Monatsbl.," 1867, p. 166.

evidence of a past attack of iritis is often first discovered by the ophthalmoscope revealing the presence of uveal pigment on the anterior surface of the lens. In a large proportion of cases iritis is due to, and its traces are signs of, constitutional syphilis; and the importance of the evidence thus afforded, from its frequency and easy recognition, is very great. But it must be remembered that rheumatic iritis may leave similar adhesions. These, whatever their cause, may explain irregularity of the pupil, which otherwise would be of uncertain significance.

Choroid.—Next to the iris, the choroid is affected by syphilitic disease more frequently than any other part of the eye. In spite of the opinions which have been expressed by some authorities, there is strong reason to believe that disseminated choroiditis is, in the majority of cases, syphilitic. In its active stage, in which extensive areas of wide exudation, comparable, in Hutchinson's opinion, to gummata, are the conspicuous features, it rarely comes under the notice of the physician. In its later stage, in which extensive regions of atrophy alternate with scattered stellate and crater-like pigmentary deposits, it is often seen. It is to be remembered that pigment is deposited in the retina also, and care must be taken to avoid confounding the change with primary retinitis pigmentosa. The choroiditis of acquired syphilis may be either unilateral or bilateral. When slight, the traces of it may be detected only in the peripheral part of the retina towards the ora serrata.

Retina.—Isolated syphilitic retinitis is less common than syphilitic choroiditis. It is characterized by areas of diffuse opacity, parenchymatous swelling, tortuous vessels, and a blurred disc. The vitreous frequently shows fine dust-like opacities. Sight is considerably impaired. This form also comes chiefly under the ophthalmic surgeon's notice.

Optic Nerve.—*Neuritis*, limited to the papilla, is common in syphilis, as secondary to brain disease, but is very rare as a primary affection, except as the effect of a gumma in the nerve. This is rare, but has been met with, increasing the thickness of the nerve at the spot to that of the finger, with

intense papillitis and opacity of the adjacent vitreous.[1] Small spots of like nature are said sometimes to form in the choroid, may press the retina forward, necessarily damaging its structure, and may even cause opacity of the vitreous.

Simple atrophy of the nerve is occasionally met with as the consequence of syphilis, double, unaccompanied by spinal symptoms. It is similar to that which occurs in tabes, of which syphilis is the common antecedent. Moreover, the loss of reflex action of the iris, which so commonly accompanies ataxy, may occur without spinal symptoms in cases of constitutional syphilis (as I pointed out long ago), and it may accompany optic nerve atrophy before the loss of sight is sufficient to explain it. The symptoms met with in most cases of tabetic optic nerve atrophy, may occur in isolation or in any partial combination, as the effect of the post-syphilitic toxine. The recognition of this makes the effects intelligible, and is certainly justified by the analogy of the post-diphtheritic toxine. (See Tabes.)

Inherited Syphilis. — The characteristic indication of inherited syphilis, interstitial inflammation of the cornea, does not come within the scope of the present work.

Of the deeper structures of the eye, the one most liable to be affected is the choroid, which is often the seat of disseminated inflammation, in infancy or later. Scattered areas of atrophy may be left, associated with accumulations of pigment, just as in the form which results from the acquired disease. When slight, they are small and round; this form is very characteristic, although somewhat rare. When the pigmentary disturbance is slight, it is sometimes mistaken for tubercle of the choroid. In two cases described by Sir T. Barlow,[2] in one of which the autopsy showed also chronic syphilitic disease of the cerebral membranes, the choroid presented, in each case, brownish flecks of exudation without disturbance of pigment or atrophy. The microscopical examination (by Nettleship)

[1] Hirschberg: "Beitrag. Dermatol. u. Syph.," 1895.

[2] "Trans. Path. Soc.," 1877, p. 287.

showed the chorio-capillaris beneath these flecks to be infiltrated with pus-like cells, and in several instances there was a layer of flattened cells on the surface next the retina. In the other case, Barlow traced the progress of similar flecks to a stage of atrophy, such as is met with in disseminated choroiditis, later in life. Hirschberg[1] has given a careful description of this form of choroiditis, which, occurring as it does in the first eighteen months of life, often escapes observation in its early stage. The ultimate results are the pale areas and pigmentation, which may end in an areolar or serpiginous arrangement of white spots and black margins.

A peculiar form of atrophy of the disc has several times come under my notice in children the subjects of this disease. The disc has a uniform reddish tint, the edges are not well defined, and the vessels are small. There has not commonly been any atrophy of the choroid or pigmentary accumulation. It is probably secondary to retinitis or widespread capillary choroiditis. Several times since first observing the connection between the two conditions, this form of atrophy has drawn my attention to the existence of inherited syphilis, which had otherwise escaped notice. Sight is usually impaired, sometimes considerably.

Retinitis sometimes occurs in the inherited, just as in the acquired disease, and often leads to permanent changes in the vessels, as has been pointed out by Spicer.[2] The ophthalmoscopic appearances left behind vary greatly in degree, from mere faint lines along the smaller (peripheral) vessels, to complete obliteration of the vessels everywhere except near the disc.

Retinitis pigmentosa is, by some, believed to be connected with inherited syphilis, and an instance of the association of the two diseases has been described by Swanzy. Deposits of pigment in the retina frequently accompany the atrophic

[1] "Deut. Med. Wochenschr.," 1895, No. 26. *See* also Hutchinson, "Ophth. Hosp. Rep.," vii. Pl. 4, Fig. 3.

[2] Spicer: "Trans. Ophth. Soc.," vol. xii. p. 116 (with microscopical drawings).

changes in disseminated choroiditis, but the connection of true retinitis pigmentosa with syphilis is very doubtful.

Chronic Rheumatism.

Chronic rheumatism has only accidental associations with changes in the fundus oculi. Neuro-retinitis has been ascribed to "rheumatism." Probably some of the cases were instances of gout, with albuminuric retinitis due to granular kidneys. It must be remembered also that the optic nerve, like the other orbital nerves, may be damaged by rheumatic inflammation at the back of the orbit (*q.v.*), and that the retro-ocular neuritis, due to a gouty diathesis, may be associated with chronic rheumatism, as effects of the same cause.

Gout.

The influence of gout in producing kidney disease renders it a powerful indirect cause of the retinal affection which accompanies albuminuria. There are, however, other ophthalmoscopic changes which are to be ascribed, with more or less probability, to the same cause, since they are seen chiefly in persons who are the subjects of gout, inherited or acquired.

(1.) *Hæmorrhagic Retinitis.*—The frequency with which the subjects of this affection present a history of gout was first pointed out by Hutchinson,[1] and the relation seems well established. It has been met with in young persons with a strong gouty inheritance. The extravasations may be small, flame-shaped, and scattered over the whole fundus. They are usually present in one eye only. Hæmorrhages may recur during a long period. The immediate cause has been thought to be thrombosis in the central retinal vein, from which Michel has shown that extensive hæmorrhages may result (*see* p. 30). The obstruction in the vein must be incomplete in many of the cases, since there is no such

[1] "Trans. Clin. Soc.," vol. xi. p. 132. *See* also "Trans. Ophth. Soc.," vol. i. 1881, p. 26.

intense venous engorgement as has been found when the thrombosis has been complete. The tendency to recurrence of the extravasations is in favour of the cause being a partial obstruction, such as would result from the gradual formation of a parietal thrombus at one or more points in the vein, in connection with changes in or around its walls.

In many cases of hæmorrhagic retinitis, however, the hæmorrhages are dependent upon local venous obstruction in the retina itself, due to the pressure exerted by an overlying thickened artery. The association with gout is then indirect, through vascular degeneration.

(2.) *Retro-ocular Neuritis.*—We have learned that spontaneous inflammation of nerve trunks and plexuses, on one side only, and occurring after the age of thirty or forty, is seldom due to any other cause than gout. Such neuritis means always primary perineuritis. This would lead us to expect that such an influence would be exerted frequently on a nerve so prone to suffer from inflammation as the optic, but inflammation of its sheath behind the eye is less common in ordinary gout than might be anticipated. Acute or subacute inflammation is, however, met with in younger adults in whom no other cause can be traced, thus tending to confirm the relation to inherited gout long maintained by Hutchinson. The characteristics of these cases are the greater degree of affection of sight than corresponds to the visible changes in the optic disc; the tendency to irregular defects in the field of vision, and particularly to central colour scotoma; and the strong tendency to the affection of the other nerve, not apparently by extension through the commissure, but by an independent symmetrical morbid process. Such symmetry always proves a general cause. The part of the nerve diseased differs, and the affection of sight varies accordingly. In some cases, the affection seems to commence at the chiasma, as described in the following paragraph. When the process is far back, no signs of inflammation may be seen within the eye, or only such as are slight and equivocal, but pallor of the disc slowly supervenes, though sight may be regained.

There is frequently considerable pain. As a rule, other orbital nerves escape—a contrast to the rheumatic cellulitis and perineuritis mentioned above (p. 172).

(3.) *Chiasmal Neuritis.*[1]—In a group of cases, rare but well defined, there is failure of sight in the temporal half of each field of vision, indicative of damage to the fibres that cross at the chiasma, probably at the seat of their decussation. The temporal hemianopia may be irregular in form and incomplete in extent, but often extends up to the middle line. There may be complete loss of the half fields for colour when perception of white is only lessened, or when there is a paracentral scotoma, on the defective side. The optic nerves within the eye gradually become pale, presenting the aspect of simple atrophy.

The symptoms are like those that are caused by pressure on the chiasma, as by a growth from the pituitary body.[2] Many, perhaps most, of the cases have been assumed to be due to a basal tumour. The opinion that they are neuritic is at present only an inference from the symptoms, positive and negative. No pathological evidence has yet been obtained, but this fact may be of some significance, and justifies the consideration of the indirect evidence as to their nature. They are not accompanied by other symptoms of organic intra-cranial disease. Under treatment, the loss of sight, in many cases, ceases to increase, and does not progress to complete blindness. Most of the patients present a strong history of ancestral gout, which we are compelled to regard as one cause of simple retro-ocular neuritis in the form just described. Lastly, the treatment which seems effective is that which is so in other chronic inflammations of the same class —mercury, to arrest the inflammatory process, followed by strychnia to influence the damaged nerve elements.

In ordinary retro-ocular neuritis, apparently dependent on

[1] It is right to state that I am alone responsible for this section.—W.R.G.

[2] A series (some of which may have been of this character) is described by Nettleship, "Trans. Ophth. Soc.," vol. xvii. 1897, p. 277. In one only was an organic cause proved (an extensive basal cyst), but in some of the others there were symptoms indicative of organic disease.

inherited gout, the fibres in the nerve from the macular region of the retina suffer most. It has been suggested that their susceptibility is connected with their higher function, which renders them, and their interstitial tissue, more readily affected. It is important to recognize that there seems to be a solidarity in nutritional tendency between the nerve elements and the neuroglia (the residue of the embryonal tissue from which both are developed), by which they seem to undergo opposite changes. Wasting of the nerve elements is attended with overgrowth of the neuroglia, which seems readily to become excessive. In gouty inflammation, this interstitial tissue may be assumed to lead the way. We can conceive that it possesses greatest susceptibility in the region of the nerve in which the macular fibres are situated, and that thus their special affection in ordinary retro-ocular neuritis may be explained.[1] These considerations may prevent surprise, should the crossing place in the centre of the chiasma present a special proclivity to suffer from the causes of retro-ocular neuritis, with temporal hemianopia as the effect, incomplete, or extending from affection of the adjacent nerve trunks. The interference with function will necessarily be similar to that caused by the pressure of a growth. Of necessity also the latter pathological cause is occasionally revealed by death. But the large proportion of cases of bi-temporal hemianopia, in which this symptom exists alone, with no evidence of organic disease, and in which the course is so prolonged that the ultimate result is unknown, is far greater than in any known form of intra-cranial growth. It seems to warrant the provisional diagnosis of chiasmal neuritis.

Lead Poisoning.

The eye is occasionally affected in lead poisoning, apart from the effects of induced kidney disease. It may suffer in

1 *See* Gunn, Ophth. Soc. Discussion, "Trans.," vol. xvii., and Gowers, "Abiotrophy," *loc. cit.*

three ways. There may be (1) amblyopia, usually transient, without ophthalmoscopic changes; (2) atrophy of the optic nerve; (3) optic neuritis.

The occurrence of blindness in lead poisoning has long been known. Some well-marked cases were published by Duplay in 1834.[1] In what v. Graefe called "the pre-ophthalmoscopic period," the transient amblyopia attracted, however, more attention than the enduring affection, so that Tanquerel des Planches spoke of failure of sight as almost invariably passing away. Optic nerve atrophy in lead poisoning was first described by Hirschler in 1866,[2] and optic neuritis by Meyer in 1868.[3] Attention was, however, especially called to the affection by Hutchinson in 1871.[4]

(1.) The transient amblyopia, without ophthalmoscopic changes, is usually sudden in onset, and may be complete. It has been observed in some cases of acute saturnism after but short exposure to the exciting cause. It commonly soon passes away, and is probably due to a direct effect of the lead on the nerve centres, analogous to the temporary amaurosis of uræmia and diabetes. In one case recorded by Fano there was, for some months, a periodical transient failure of sight at the same hour each day.

Hemianæsthesia has been observed (chiefly on the Continent) in consequence of lead poisoning. It is apparently of functional origin, and may be due to the same mechanism as the transient amblyopia. The two coincided in a case recorded by Landolt,[5] although in this case it is very doubtful whether the symptoms were due entirely to functional disturbance. Hemiplegic weakness, hemianæsthesia and amblyopia came on together, and there were peculiar scotomata which endured.

[1] "Arch. Gén. de Méd.," 1834.

[2] "Wien. Med. Wochenschr.," 1866, Nos. 6 and 7. It is not easy to say, from the account of the case, whether neuritis was present or not. The disc is spoken of as being grey and having lost its transparency.

[3] "L'Union Méd.," No. 78.

[4] "Ophth. Hosp. Rep.," vol. vii. p. 6.

[5] "Ann. d'Oculistique," vol. lxxxiii. March, 1880, p. 165.

Stood [1] has published several cases where there was progressive concentric contraction of both fields, both for white and for colours. In some there was slight neuritis, and a central scotoma in a few.

(2.) In amblyopia of long duration it is common to find the signs of atrophy of the optic nerves. The discs are sharp-edged, pale, and often greyish, the arteries small. It is said that the atrophy may be from the first unattended by vascular changes (Horner); in some cases, an early stage of simple congestion of the discs has been described, with softened edge, but little swelling. Gradually the redness fades, and a reddish-grey atrophy results, often with distinct white lines along the narrowed vessels. Sight has been much affected in all the recorded cases, the acuity of vision impaired, the field presenting a central or peripheral defect. The loss often progresses until even quantitative perception of light may be lost.

(3.) Occasionally cases of lead poisoning present much more pronounced inflammatory changes—considerable papillitis with swelling, and hæmorrhages. An instance of this form of neuritis is shown at Pl. V. 6. The affection is almost always double, and necessarily impairs vision. It leaves "consecutive atrophy," a dull-white, full-looking disc, with narrow arteries. On the other hand, the neuritis may clear away, and useful sight return, as in a case recorded by Schrœder. It is important to remember that the affection of sight in these cases may be in part due to the direct effect of the lead on the nerve elements, which has been already mentioned. This is the more likely when, as in many recorded cases, loss of sight comes on suddenly.[2]

These ocular changes commonly occur in chronic cases of lead poisoning, which have presented toxic symptoms for some time, often for years, and may coincide with an increase of the other symptoms. It must be remembered, however, that the manifestations of lead are very irregular,

[1] "Arch. f. Ophth.," 1884, iii. p. 215.

[2] *See* a case reported by Stricker from Traube's Clinique, and quoted by Abadie ("Mal. des Yeux").

and any one may be absent, or may alone be present. In cases of very recent intense lead poisoning, toxic amblyopia and neuritis are apparently more common than atrophy. In many cases the occurrence of neuritis coincides with symptoms of cerebral disturbance, headache, convulsions, delirium,[1] &c. The case figured in Pl. V. 6 presented, at the same time as the neuritis, much mental disturbance, and both symptoms passed away together. In a fatal case of this description, recorded by R. Atkinson,[2] there were no naked-eye changes in the meninges or brain, but lead was found in it in quantity equivalent to five grains in the whole brain. The association of cerebral disturbance with optic neuritis in these cases is probably more than a coincidence, and the analogous fact as regards albuminuric neuritis (p. 213) may be borne in mind.

The diagnosis of saturnine atrophy and neuritis rests especially on the recognition of the signs of lead poisoning, the line on the gums, the occurrence of gout, of colic, of wrist-drop, and the presence of anæmia. It is only by these symptoms that the cause of the neuritis can be discerned. The possibility of a renal neuritis must be borne in mind. It is highly probable that in at least one published case the retinal change was due to this, and not directly to the lead. Albuminuric retinitis is not uncommon in cases of chronic lead poisoning of long duration. It must not be forgotten that an intra-cranial growth of any nature may coexist with lead poisoning. Coincidence suggests a causal relation, but does not involve it. An important element in diagnosis is to remember the possibility of the improbable.

The prognosis in all forms of saturnine changes is uncertain. It is least grave in the case of simple toxic amblyopia, next in cases of pronounced neuritis, especially of acute course,

[1] This is an old observation. Tanquerel des Planches described saturnine amaurosis as the accompaniment of encephalopathia and colica saturnina, and stated that in cases of this kind no material change is to be found in the brain ("Traité des Mal. de Plomb.," 1839, tom. ii. pp. 211 and 235).

[2] "Lancet," 1878, i. p. 784.

and moderate degree. In pronounced atrophy it is very unfavourable. Of fourteen cases of various forms, collected by Lespille-Moutard,[1] nine progressed to blindness.

The treatment is essentially that for the general state, but local applications, leeching and counter-irritation, have appeared useful in some cases.

Chronic Alcoholism.

Atrophy of the optic discs, sometimes of one only, sometimes of both, is occasionally met with in the subjects of chronic alcoholism, to which it has been thought to be due. It is said to occur especially in sedentary drinkers, to be more common on the Continent than in this country, and to result from spirit-drinking rather than from wine. Hence, according to Rominée,[2] it is much more common in the north of France, where much cheap brandy is consumed, than in the wine-producing districts of the south. Amblyopia may precede any ophthalmoscopic change, and is characterized by a central dimness or defect (Förster), very similar to that met with from tobacco,[3] but said to be more exactly central by Hirschberg. (The influence of tobacco has not always been excluded with sufficient care.) Before there is recognizable defect for white light, a defect for red and green may be discovered, extending from the fixing point to the blind spot and a little beyond each. To detect it in slight cases the ordinary colour tests do not suffice, since the coloured object should not be more than five millimetres in diameter. Extensive loss of vision for certain colours, as green and violet, has also been

[1] "Thèse de Paris," 1878.

[2] "Recueil d'Opht., 1881," Nos. 1, 2.

[3] It should be remarked that the symptoms here described (in accordance with the opinion of most authorities) as due to alcohol, are believed by some to be met with only in drinkers who are smokers, and to be really due to tobacco. *See* Nettleship, "St. Thomas's Hosp. Rep.," 1879. If it is true that smokers who drink suffer less from tobacco amblyopia than do abstainers from alcohol, additional doubt is thereby cast on the influence of the latter in causing the same symptoms (See p. 263.)

described by Galezowski as a symptom of retinal anæsthesia, in some cases of chronic alcoholism.[1]

A stage of congestion, before the onset of the atrophy, has been described by Allbutt and others. The appearances were generally those already described as "Simple Congestion" (p. 43), uniform redness of the disc, with softened edges. The disc gradually becomes paler, and ultimately passes into white or greyish atrophy, often with small vessels. Uhthoff[2] examined a thousand cases of severe alcoholism in inmates of asylums, and found that 13·9 per cent. of these suffered from pathological whiteness of the temporal half of each disc,[3] with a central scotoma in every case. He states that he found this in only one out of a hundred apparently healthy men, whom he selected for comparison. Moreover, Moeli[4] has stated that he has detected changes in the optic disc in 15 per cent. of the cases of delirium tremens examined by him. When the condition of the nerve has been ascertained by microscopical examination, granular degeneration of the nerve fibres has been found in some cases. Out of seven cases examined post mortem by Uhthoff, two showed distinct interstitial neuritis, with marked increase of the connective tissue. The changes were most distinct just behind the globe, and did not extend far back. Congestion, with slight œdema, is certainly sometimes to be seen in cases of chronic alcoholism, and is, no doubt, a condition analogous to the change in the meninges which leaves the thickening and opacity often to be found after death.

The progress of the atrophy is slow, and the prognosis better than in many other forms; considerable good being effected, especially in the pre-atrophic stage of amblyopia, by strychnia and tonics. Complete recovery of vision is

[1] *See* also Nuel: "Ann. d'Oculistique," Sept. 1878.

[2] "Ophth. Rev.," vii. p. 100.

[3] This condition alone is of doubtful significance; when the central cup is wide and shelving, there may be no redness on the temporal side in normal states.

[4] "Neurol. Centralbl.," 1884, p. 260.

said sometimes to take place, although the pallor of the disc continues.[1]

In a fatal case of alcoholism, Lawford found during life widespread cloudiness of the retina, with normal discs, and without any central colour scotoma. After death the retina of one eye was examined by Edmunds [2] and himself; there was œdema of the nerve-fibre and ganglion-cell layers, and in the outer nuclear layer there were spaces, filled with a clear effusion, between the Müllerian fibres. In a case of severe alcoholic paralysis related by Ord,[3] well-marked double retinitis was found, with white patches, of uncertain nature.

In acute alcoholism, ophthalmoscopic changes are uncommon. In one case Jäger found diffuse retinitis, with numerous hæmorrhages, in a patient suffering from delirium tremens. This was probably a mere complication, but we need more facts regarding slight changes.

Tobacco Poisoning.

The occurrence of defective sight from tobacco smoking was described in 1854 by Mackenzie, who was inclined to attribute most cases of amaurosis to this cause. The subject attracted little attention until Hutchinson,[4] in 1864, brought forward facts to show that amblyopia, accompanied by slight ophthalmoscopic changes, often results from this cause, while Förster[5] and Hirschberg[6] have demonstrated that the affection of sight presents special characteristics. The relation between these symptoms and tobacco smoking must be regarded as well established.[7]

[1] Berry: "Ophth. Rev.," iii. 1884, p. 101.

[2] "Trans. Ophth. Soc.," ix. 1889, p. 137.

[3] "Lancet," Feb. 11, 1888; *see* also Sharkey, "Trans. Path. Soc. of Lond.," 1889, p. 359.

[4] "Lond. Hosp. Rep.," 1864; *see* also "Med.-Chir. Trans.," 1867; "Ophth. Hosp. Rep.," 1871 and 1876.

[5] Graefe u. Saemisch's "Handbuch," vol. vii. p. 201.

[6] "Deutsche Zeitschrift f. Prakt. Med.," 1878.

[7] For an excellent *résumé* of what is known on the subject, *see* Nettleship's "Notes on the Diagnosis of Tobacco Amblyopia," "St. Thomas's Hosp. Rep.," 1879.

Förster has remarked that the sight suffers from tobacco, generally between thirty-five and sixty-five years of age. But cases are met with at any age above eighteen. Mental trouble, with its accompanying sleeplessness and nervous depression, seems often to be the determining cause of the effect. It has been thought that the disease occurs more frequently among abstainers from alcohol than among those who take alcohol, and some facts mentioned by Nelson [1] seem to show that in the latter the affection may come on more slowly. But a striking fact is the occasional influence of tobacco as an apparent excitant of the failure of sight in family atrophy (optic abiotrophy, *see* later), and also in other maladies which seem to involve a predisposition, as insular sclerosis.

In the ordinary form of tobacco-amblyopia, the failure of sight is gradual, and usually equal in both eyes, unaccompanied, as a rule, with headache or other cerebral symptoms. It is nearly always more marked in a bright than in a dull light. The characteristic of the failure is the presence of a defect in the centre of the field of vision, a "central scotoma," transversely oval or oblong, extending from the fixing point to the blind spot, and often embracing both. It is commonly a relative, not an absolute scotoma; there is dimness, not loss, of sight, and the failure is greater for certain colours (green and red) than for white. If the defect is slight, the coloured object must be of small size in order to detect it. The scotomata are symmetrical, or nearly so, in the two eyes (*see* Figs. 60, 61, p. 120), and seem to begin most commonly at or near the fixing point (Leber, Treitel, Nettleship). Nelson, however, has described a case in which the scotoma surrounded the blind spot, and the fixing point was free. The variations in the exact limits of colour fields in different individuals and with different degrees of illumination and different sizes of the object, render it uncertain whether there is a peripheral restriction of these fields; such restriction is certainly not always present, but probably may

[1] "Brit. Med. Journ.," 1880, ii. p. 774.

exist in severe cases (Treitel, *see* also Fig. 59, p. 120). Indeed, it is possible that, in rare cases, there may be only a peripheral restriction, without central loss, even for colour.[1] The symmetry of the scotomata is anatomical, not functional, and probably indicates a morbid process in the macular ganglion cells, with secondary affection of the axial fibres of the optic nerve (*see* p. 119, note). Sometimes a state of simple congestion may be the first stage—a "hazy disc," with slight uniform redness and soft edges, but without noticeable swelling. In exceptional instances, distinct papillitis, with a few small retinal hæmorrhages near the disc, has been found; in these cases the failure of sight has been unusually rapid, and there has been a history of recent great excess in tobacco.

The treatment consists essentially in the removal of the cause. Tonics and hypodermic injections of strychnine are also of use, especially in the pre-atrophic stage. Hutchinson believes the prognosis in most cases to be good, three-fourths of his cases having recovered, or presented great improvement in sight. Age does not render the prognosis markedly worse.[2]

Quinine.

Quinine in large doses may cause complete temporary amaurosis. Many well-marked cases have been recorded.[3] The amount of quinine which caused the symptoms varied from a minimum of 80 grains in thirty hours, to a maximum of 1,300 grains in three days. In most of these cases the quinine was given for malaria; that the affection of sight

[1] As in the case of a young man (seen with Mr. Higgens) in which the simple peripheral diminution of the fields, with some amblyopia, passed off after tobacco was discontinued.—W. R. G.

[2] For much valuable information on this subject, the reader is referred to the "Report on Toxic Amblyopia," "Trans. Ophth. Soc.," vol. vii. 1887, p. 85.

[3] Giacomini: "Ann. Univers. di Med.," 1841; Graefe: "Arch. f. Ophth.," iii. pt. 2, p. 396; and Knapp: ibid. x. pt. 2, p. 220. The last paper contains a very full discussion of the subject. *See* also papers by Browne, "Trans. Ophth. Soc.," vol. vii. p. 193 (with references to previously recorded cases), and Nettleship, in same volume, pp. 218, 219.

was due to the former, and not to the latter, is proved by the definite and peculiar character of the symptoms, and by the fact that, in some other cases, the patient was not suffering from any disease, and the quinine was taken by accident. In all the severe cases the loss of sight was at first complete, and was associated with loss of hearing. The deafness soon passed away, usually in twenty-four hours. The blindness continued for a longer time, which varied according to the dose. Central vision returned to the normal in a few days, weeks or months, but the peripheral vision continued absent for a very long time. This contraction of the visual field after the return of central vision seems to be invariable, and the restricted field is usually transversely oval. Colour-vision is also impaired. The pupils, in slight cases, have been said to be contracted (in association with transient increased sensitiveness of central vision),[1] but in severe cases they are dilated, and during total blindness are irresponsive to light, but act to accommodation (Grüning). The ophthalmoscope has shown pallor of the disc, and in all cases a remarkable diminution in size of the retinal vessels, which may be reduced to threads, and emptied by the slightest pressure on the eye. A cherry-red spot at the macula has been observed.[2] Vorhies found the choroidal vessels also empty. In the case of Giacomini, where three drachms were taken at a single dose, there was loss of consciousness at the onset. In nearly all cases a considerable degree of recovery has ultimately occurred. In the most severe case (Michel), in which seven drachms of quinine were taken, there was no improvement for several months, and it was thought that sight was permanently lost; nevertheless, fifteen months afterwards acuity of vision was nearly normal, although the fields were much restricted. The vessels had increased in size, but were still much below the normal. Recovery in six weeks has followed a dose of five drachms. Whilst the symptoms are passing off, relapses may be produced by insignificant doses of quinine.

[1] Barabeschew: "Wjestnik Oftalm.," 1901, No. 1.

[2] By Grüning, but not by Barabeschew.

Bisulphide of Carbon.

Bisulphide of carbon was the apparent cause of a "peri-neuritis," ending in partial atrophy, in a case recorded by Galezowski.[1] Atrophy of the optic nerves is also seen, not very rarely, among the workers in india-rubber factories, in which bisulphide of carbon is used. A special committee of the Ophthalmological Society (consisting of Messrs. Frost, Gunn and Nettleship) was appointed to investigate this form of toxic amblyopia, and in their valuable report[2] on the whole subject they tabulate twenty-four cases of amblyopia coming on after the development of other symptoms indicating great depression of the whole nervous system. In most cases examined there was a distinct central colour-scotoma, and the ophthalmoscope showed pallor and blurring of the edge of the optic disc, with loss of transparency of the retina for some distance from the disc.

Other Poisons.

Silver poisoning is said to be accompanied by amblyopia, in addition to the other symptoms of argyria. No ophthalmoscopic changes have, however, been recorded, but silver has been found in the eyeball (sclerotic sheath of the optic nerve, &c.) by Reimer, deposited in small round granules. The effect of silver is closely analogous to that of lead. It may, as I have seen, lead to wrist-drop, gout, and albuminuria, and it is therefore highly probable that the same ocular changes may, in some cases, result.

In *mercurial poisoning* amblyopia has been observed; in one case optic neuritis existed,[3] and in another optic nerve atrophy.[4] Of ocular changes in copper poisoning nothing is known. In phosphorus poisoning retinal hæmorrhages have been sometimes found.[5]

[1] Galezowski: "Des Amblyopies et Amauroses Toxiques," p. 141.
[2] "Trans. Ophth. Soc.," vol. v. 1885, p. 157.
[3] Square: "Ophth. Hosp. Rep.," vi. p. 54.
[4] Galezowski: "Des Amblyopies et Amauroses Toxiques," p. 141.
[5] Niederhauser: "Inaug. Diss. Zurich," 1875.

Salicylic acid may cause amblyopia, but without changes in the fundus oculi. The same effect has been observed from salicylate of soda.[1]

Amblyopia has also been described as occurring from poisoning by iodoform, opium, cannabis indica, tea, coffee, stramonium, osmic acid, and several coal-tar products.[2]

ACUTE GENERAL DISEASES.

Typhus Fever.

Loss of sight has been many times observed during convalescence from typhus fever,[3] and subsequently atrophy of one or both optic nerves has been found. In some of these cases there have also been cerebral symptoms, as in a case recorded by Benedikt, in which left hemiplegia was accompanied by atrophy of the right optic nerve. In such cases, probably, the atrophy was the result of a cerebral lesion. In other cases there were no symptoms except those in the eye, and a primary affection of the optic nerve appeared to have occurred. In some cases the ophthalmoscopic changes have been those of simple atrophy, but in others, where the affection of sight was first noticed during convalescence, optic neuritis has been found.[4] In a case at the Hospital for Sick Children, marked papillitis was found by Penrose and Marcus Gunn during the height of the fever. Of the origin of the neuritis nothing is known.

[1] Gatli: "Gaz. degl. Ospital," 1880, i. 4.

[2] For an exhaustive account of this subject, *see* De Schweinitz: "The Toxic Amblyopias," in Norris and Oliver's "System of Diseases of the Eye," vol. iv. p. 797, and the same author's essay on "The Toxic Amblyopias: their Classification, History, Symptoms, and Pathology."

[3] In a considerable number of the cases recorded abroad it is doubtful whether the disease was typhus or typhoid fever. The cases on which the statements in the text are founded appear to have been true typhus.

[4] Teale: "Med. Times and Gazette," May 11, 1867. Chisholm: "Ophth. Hosp. Rep.," vol. vi. p. 214.

Typhoid Fever.

The occurrence of amblyopia and amaurosis during convalescence from typhoid is well established,[1] although rare. It may or may not be attended with ophthalmoscopic changes. In the latter case the prognosis is favourable; the affection usually passes away in the course of two to eight weeks. The form of amblyopia varies; anæsthesia of the retina has been observed by Leber, and an annular defect in the field by Hersing. It seems allied to the loss of speech and mental weakness sometimes met with.

When ophthalmoscopic changes have been observed, there has been simple atrophy, single or double, without preceding inflammation; or double neuritis, ending in atrophy, partial or complete, or less commonly in recovery. Hutchinson has, for instance, recorded[2] the case of a boy whose sight failed at three years and a half, two to four weeks after a fever with diarrhœa and headache, a sister having suffered from similar symptoms at the same time. Symmetrical neuritis was found, and ten years later white atrophy with small vessels. The neuritis is so rare that Leber suggests, as Stellwag v. Carion had done long before,[3] that the cases in which it is found may really have been cases of meningitis which have been mistaken for typhoid fever—an error not very rare. It must be remembered, however, that neuritis does occasionally follow other acute specific diseases. It has been thought that the cases accompanied by hyperæmia of the discs are cases complicated by meningitis; but meningitis, except as secondary to suppuration in the ear, is exceedingly rare in typhoid fever.

Extreme narrowing of the retinal arteries, on both sides, with pallor of the discs and loss of sight, was found by Heddaeus[4] in a case of great emaciation after typhoid. On good food, the arteries regained their normal size, but the discs remained pale, and sight did not improve beyond $\frac{1}{20}$.

1 Nothnagel: "Deut. Arch. für Kl. Med.," 1872, ix. p. 480.
2 "Ophth. Hosp. Rep.," ix. p. 125.
3 "Ophthalmologie," Bd. ii. Abt. I. 1855, p. 662.
4 "Monatsbl. für Augenheilk.," Aug. 1865.

Embolism of the central artery of the retina has been observed during convalescence from typhoid.[1]

Relapsing Fever.

Extensive intra-ocular inflammation is apt to follow relapsing fever. Trompetter [2] found it in 21 out of 325 cases, or six per cent. There was inflammation of the choroid and ciliary body, with hypopyon, but without iritis. There were also opacities in the vitreous, amblyopia, and restriction of the field. Its origin is doubtful. Thrombosis in vessels, or embolism from the spleen, has been assumed as its cause.

Measles.

Amblyopia, without ophthalmoscopic changes and ultimately improving to the normal, has been seen, as a sequel to measles, by v. Graefe and Nagel; in some cases accompanied by cerebral symptoms, convulsions, and sopor. Nagel has also met with three cases of optic neuritis after measles, but in the epidemic in which they occurred there were many cases of meningitis. In three other cases recorded by Wadsworth [3] there were also symptoms of meningitis. Stephenson [4] has described optic neuritis after measles, without any symptoms indicative of meningitis. The observation is valuable, since the eyes were examined shortly before the attack, and found normal. The discs later became paler than before, but were not completely atrophied, and there was still good vision. As Förster remarks, the commonness of the disease, and the rarity of affections of sight in it, show that the connection between the two cannot be a very close one. It must be remembered, however, that measles may give rise also to myelitis.

[1] Galezowski: "Traité Icon."; also Snell, "Ophth. Rev.," i. p. 403.
[2] "Klin. Monatsbl.," April, 1880, p. 123.
[3] "Boston Med. and Surg. Journal," vol. ciii. p. 636.
[4] "Trans. Ophth. Soc.," vol. viii. 1888, p. 250.

Scarlet Fever.

The frequency with which renal disease accompanies and succeeds scarlet fever renders affections of sight, of this nature, not very rare. Occasionally, however, they arise independently of any renal complication.

(1.) Uræmic amaurosis is common in scarlatinal dropsy. It comes on suddenly, when the renal disease is at its height, is commonly complete, double, unattended by ophthalmoscopic changes, and passes away. Occasionally, cerebral symptoms accompany it—convulsions, and, in rare cases, hemiplegia, from a cerebral thrombosis or embolism, which persists after the cessation of the convulsions and the return of sight. Persistent Bright's disease may also affect the retina.

(2.) Optic neuritis has followed scarlet fever when there has been no renal disease or albumen in the urine. Betke[1] has recorded a case in which there was great dimness of sight seventeen days after desquamation. There was no albuminuria, but a marked neuro-retinitis was found on ophthalmoscopic examination, less developed in the right eye than in the left. There was no sign of meningitis, past or present. The neuritis disappeared, and sight was restored in eight weeks. A similar case has been recorded by Pfluger,[2] in a child ten years old. Three weeks after an attack of scarlet fever with much headache, loss of sight occurred, and was complete at the end of three or four days, when intense double papillitis was found, with some hæmorrhages. A month later, sight had much improved, but four months after it was not quite normal, and the neuritis had not entirely subsided. The urine throughout was free from albumen. In a case recorded by Hodges,[3] permanent loss of vision was due to embolism or thrombosis of one retinal artery.

[1] "Monatsbl. für Augenheilkunde," Bd. viii. 1869, p. 201.
[2] "Arch. f. Ophth.," xxiv., pt. 2, p. 180.
[3] "Ophth. Rev.," iv. p. 296.

(3.) Atrophy of the optic nerve has been met with after scarlet fever, having the aspect of a consecutive atrophy. It has been observed in association with the symptoms of a local cerebral lesion, hemiplegia, &c. (Loet), but in some cases has occurred alone. In two sisters,[1] sight gradually failed some months after scarlet fever, without albuminuria or dropsy. One became blind and idiotic, and the other epileptic. The optic discs were "pale," and the fundus in each case showed accumulation of pigment. The condition was, perhaps, that of retinitis pigmentosa.

It must be remembered that an intense albuminuric inflammation may leave some atrophy of the optic nerve.

Variola.

Leber has observed diffuse neuro-retinitis in variola, during the stage of drying of the eruption. In a case which came under my own observation, atrophy of one optic nerve appeared to have succeeded small-pox.[2]

Influenza.

Optic neuritis has been observed in a considerable number of cases of influenza during the last twelve years. Visual loss is the earliest prominent symptom; the amount of failure varies considerably in different cases. In some there is merely a central scotoma, while in other cases absolute blindness has been found. The loss of vision is usually only temporary, and the prognosis is generally favourable. Both eyes are usually affected, but sometimes the condition is unilateral.

The visual loss occurs from four to fifteen days after the beginning of the attack of influenza, and it is generally accompanied by orbital pain.

The condition is evidently an acute retro-ocular neuritis.

[1] Bayley, "Lancet," Sept. 15, 1877.

[2] *See* Case 60 in first and second editions.

In some cases there is gross papillitis, while in others only slight changes are found with the ophthalmoscope.

Acute Rheumatism.

Acute rheumatism is not usually associated with any changes in the fundus oculi. Embolism of the cerebral arteries sometimes, though rarely, occurs during the course of an attack, but embolism of the retinal arteries has only been observed as a late sequel of the resulting endocarditis. Schmidt once observed irido-choroiditis (such as is common in relapsing fever) after an attack of acute articular rheumatism without endocardial complication.[1] The relation of iritis to a chronic rheumatic diathesis has been already mentioned.

Malarial Fevers.

Changes in the fundus oculi sometimes occur in the severer forms of malarial fever, especially in tropical climates. Poncet,[2] for instance, found changes in ten per cent. of the cases of malarial cachexia in Algeria. The changes which have been observed consist of retinal hæmorrhages, neuro-retinitis, and atrophy of the optic nerve.

Hæmorrhages may occur without other change, sometimes in the posterior segment of the eyeball, sometimes chiefly in the ciliary region (Poncet). Three instances of retinal hæmorrhages in ague have been recorded by Sir Stephen Mackenzie.[3] One was a young man, aged twenty, who had one attack of ague on his way home from India, and a severe paroxysm immediately after his arrival. The attacks recurred daily for a fortnight, when he came under treatment, and numerous retinal hæmorrhages were found, most numerous near the disc, chiefly along the course of the larger vessels, especially arteries, which they in places obscured (Fig. 84).

[1] "Arch. f. Ophth.," Bd. xviii.

[2] "Ann. d'Oculistique," May, 1878.

[3] In a paper on "Retinal Hæmorrhages and Melanæmia as Symptoms of Ague," "Med. Times and Gaz.," 1877. I am indebted to Sir S. Mackenzie for the woodcuts from his paper.

Sprinkled about the fundus, and most numerous near the disc, were many small round bright spots, resembling pin-holes pricked in a piece of paper held up against the light. The retinal vessels were of normal size, and their sheaths did not appear thickened. These hæmorrhages were carefully observed day by day, and were seen to fade away gradually; and, as each died away, it left, to mark its former situation, one of the shiny white spots of which mention has been made above. There was no albuminuria or other symptom of Bright's disease. The spleen was large. The blood at first contained much pigment, but after the first few days, no more could be found.

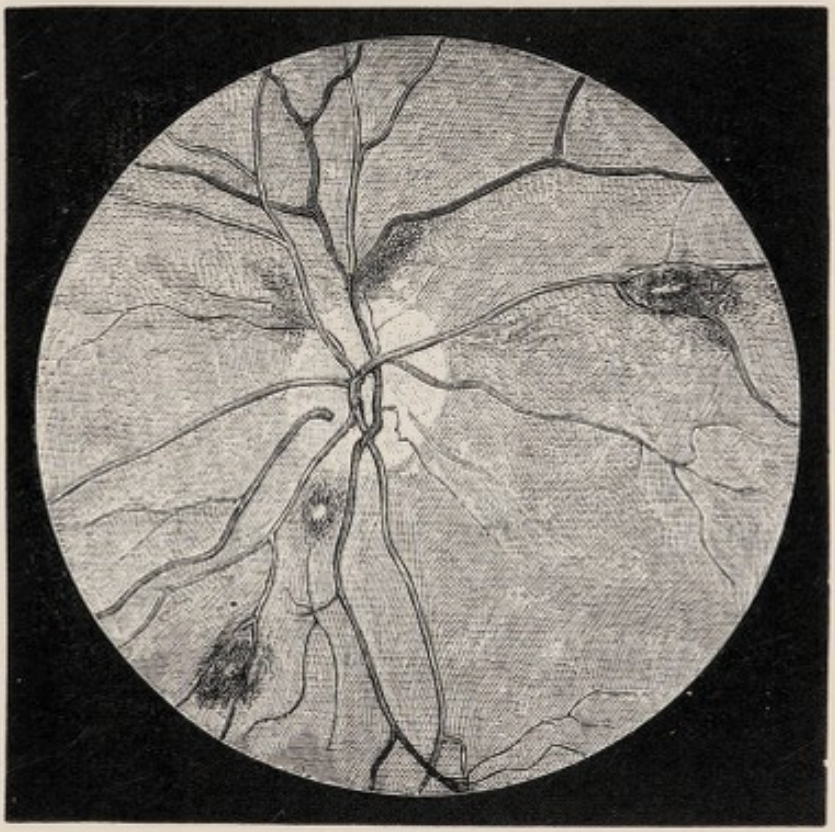


FIG. 84.—RETINAL HÆMORRHAGES IN AGUE (MACKENZIE).

In two cases at the Seamen's Hospital—a man, aged twenty-nine, with quotidian ague, and another, aged eighteen, with tertian ague—hæmorrhages were found; in the former case, numerous, large, and superficial, leaving white patches. One was paler in the centre than in the periphery (Fig. 85).

They quickly disappeared. Neither of these patients had melanæmia. In several cases subsequently examined, no hæmorrhages were found.

Hæmorrhagic retinitis has also been met with accompanied by an extensive hæmorrhage into the vitreous.[1]

Poncet observed, in Algeria, besides hæmorrhages, peripapillary œdema and even considerable neuro-retinitis. He also found, in the retinal and choroidal vessels, large cells containing leucocytes and pigment. Neuritis has also been occasionally found by others,[2] and has been associated with stellate deposits of pigment in the retina following the course of the vessels.

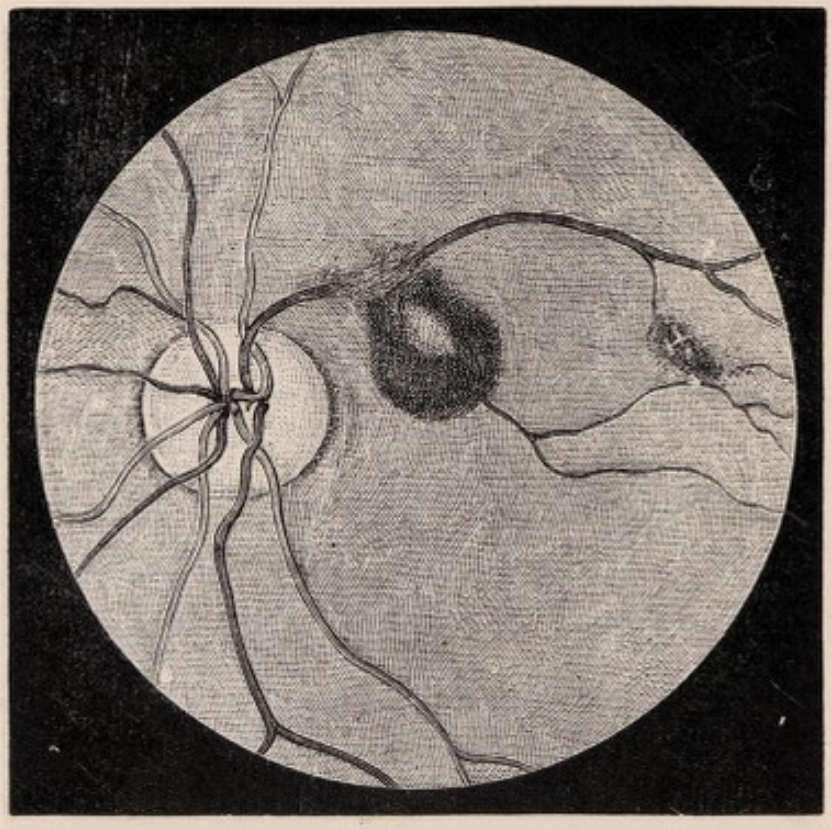


FIG. 85.—RETINAL HÆMORRHAGES IN AGUE (MACKENZIE).

Atrophy of the optic nerve has also been observed[3] after the severe malarial fever of hot climates. The disc is

[1] Van Kries: "Arch. f. Ophth.," vol. xxiv. pt. 1, p. 159.

[2] Galezowski: "Traité Icon.," p. 190; Hammond: "Trans. Amer. Neurol. Soc.," 1875.

[3] Bull: "American Journ. of Med. Science," 1877, p. 403.

white, the vessels small, and the field of vision is greatly restricted.

The pathology of the retinal changes is still obscure. The hæmorrhages have been ascribed to pigmentary embolism, but they are to be found when there is no melanæmia. Some effect exerted by the organisms, which have been found to be the essential agent, is doubtless the mechanism, but its precise nature is still unknown. It seems possible that the atrophy may be the result of such neuro-retinitis as is described above; it may also be a direct toxic atrophy.

Two remarkable cases which have been recorded by Ramorius,[1] suggest that spasm of the retinal vessels may be a consequence of malarial poisoning. The chief symptom was periodical amblyopia, and during one of the attacks, the optic discs were pale, the retinal arteries were filiform and almost bloodless, and the veins were scarcely perceptible. At the same time there was great congestion of the face and ears, and a sensation of heaviness in the head. Each attack was attended with a sensation of coloured circles moving from the periphery of the field towards the centre. In the intervals between the paroxysms the appearance of the fundus oculi was normal. Bromide of potassium had no effect, but quinine quickly cured each case. Yet it is strange how similar these symptoms are to those produced by fatigue.

Purulent affections of the eye (choroiditis,[2] iritis, &c.), such as are seen in septicæmia, have been described in intermittent fever, but are extremely rare, and some doubt may be felt regarding the diagnosis of the original disease when it is remembered how closely some cases of septicæmia simulate intermittent fever. Even the influence of quinine, on which diagnostic weight is often laid, is not entirely conclusive.[3]

[1] "Ann. di Ottalmologia," 1877, pt. 1, and "Ann. d'Oculist.," vol. lxxxii. p. 200.

[2] Peunoff: "Centralbl. für Augenk.," 1879, p. 120.

[3] For example, in a case of this kind described by Landesberg, in which, although quinine cut short the affection, abscesses formed during convalescence, in one toe and the forearm.

Erysipelas.

Erysipelas of the face is sometimes followed by loss of sight and by the signs of atrophy of the optic nerve (v. Graefe, H. Pagenstecher, Hutchinson, and others). It is probably always produced by the extension of the cellulitis into the orbit, and the resulting damage to the trunk of the optic nerve by invasion or pressure. V. Graefe has pointed out that there is commonly some exophthalmos, but this may be very slight, and may bear no proportion to the subsequent damage to sight. In most recorded cases any symptoms suggestive of orbital cellulitis have escaped notice, probably from the difficulty of the examination. In one, however (Story[1]), there was permanent limitation of the ocular movements. The loss of sight may come on rapidly. In one of Pagenstecher's cases it was complete at the end of fourteen days. Early observations of neuritis or neuro-retinitis have been recorded by Vossius and Lubinski,[2] and slight opacity of the retina has been seen by many observers. It rapidly passed into atrophy. Usually, when the examination could be made, there has been pallor of the disc and remarkable narrowing of the vessels, the arteries especially. Jäger has recorded, for instance, a case in which an adhesion of the eyelids required division with the knife five weeks after the erysipelas; the optic disc was grey and atrophied; one branch of the central artery and its corresponding vein were normal, the others reduced to lines with white borders. In Story's case some arteries were bloodless, and occluded veins were represented by dark radiating lines.

It is probable that thrombosis in the central artery is at least one mechanism by which the effect is produced. Thus, August[3] found the ophthalmoscopic appearances similar to

[1] "Brit. Med. Journal," March 16, 1878.

[2] Lubinski: "Klin. Monatsbl.," April, 1878, p. 168; Pflüger of Berne: "Augenklinik Bericht" for 1877; and Virchow's "Jahresbericht," 1878, vol. ii. p. 438.

[3] "Klin. Monatsbl.," 1884, 43.

those in embolism (arteries either invisible, or, in places, transformed into white lines), in a case in which the erysipelas caused orbital cellulitis, and in addition, visible clotting in supra-orbital and frontal vessels; he believes that the organisms penetrate the walls, and cause inflammation and clotting. Knapp,[1] however, thinks that the mechanism is compression of vessels in the orbit. In an early case he found the veins distended with stagnant blood. He quotes Panas, who found obliteration of the retinal artery. In a case observed by Nettleship, although the arteries were small, they pulsated on pressure. It seems probable that the mechanism is not always the same.

In one of Pagenstecher's cases there was a central scotoma and also peripheral restriction of the field. Necrosis of the nerve, less complete at the lamina cribrosa than farther back, was found by Nettleship.[2] Opacity of the vitreous and glaucoma have also been met with after erysipelas.[3]

Diphtheria.

The defect of sight which so often follows diphtheria, and is due to a paralysis of accommodation, is not attended by any ophthalmoscopic change. In rare cases vision is defective, apart from the paralysis of accommodation, and in such cases one or two observers (*e.g.* Bouchut) have found congestion of the disc, simple, or with œdema sufficient to veil the edges, and even in part, the vessels, and in very rare cases, an actual neuritis, which may go on to atrophy. The atrophy may be unilateral, as in one case figured by Bouchut. This case, however, was accompanied with partial right hemiplegia and defect of speech. The congestion and œdema are usually bilateral, but may be more intense on one side than on the other. I have also seen one case of primary atrophy after diphtheria. The patient was a woman, aged forty-one, with a family history of epilepsy. After the common paralysis,

[1] "Arch. f. Augenkr.," 1884, i. 83.

[2] "Trans. Path. Soc.," vol. xxxi. 1880, p. 254.
See also "Trans. Ophth. Soc.," vol. ii. p. 90; vol. xiii. pp. 82 and 87.

which passed away, except some diplopia, slight weakness of the right side developed and became permanent. With this there occurred progressive failure of sight, and two years later there was well-marked primary atrophy of the optic nerves, with considerable amblyopia. The pupils did not react to light in the least, and but slightly to accommodation. There was nystagmus on looking to the left, and the upward movement of the eyes was completely lost. There were no other signs of tabes, and the knee-jerks were perfect.

Parotitis.

Transient dimness of sight may succeed mumps, and a coincident congestion of the optic nerve has been described, but is of doubtful significance.

Tonsillitis.

In a case of tonsillitis v. Graefe once saw signs of diminished blood-supply to the retina accompanying sudden loss of sight. The known relation of tonsillitis to rheumatism suggests the probability of embolism in this singular case.

Whooping-Cough.

Blindness has been observed to come on during the progress of whooping-cough, and in one case Knapp[1] found the discs white, and the retinal arteries invisible in one eye and mere lines in the other. The patient was very weak, and Knapp suggests as explanations, anæmia from cardiac weakness, or hæmorrhage into the nerve-sheaths. Landesberg[2] also observed in one case symptoms of partial embolism, serous infiltration into the retina, slight swelling of the papilla, a red macula, thin arteries, engorged and tortuous veins.

[1] "Arch. of Ophthalm. and Otol.," vol. iv. Nos. 3 and 4, p. 448.
[2] "Med. and Surg. Reporter," Sept. 8, 1880.

Two upper arterial branches were found to be permanently obstructed. In another case he observed ecchymoses in the retina. Such affections are probably due to thrombosis, since they occur chiefly in children who are much prostrated.

Cholera.

In cholera v. Graefe found that, during the state of collapse and cyanosis, the circulation in the smaller, and even in the middle-sized, arteries may apparently cease. When the weakness of the heart was moderate, the artery pulsated on slight pressure with the finger on the eyeball; but when the heart was strong this could not be well produced. If the heart was so weak that the radial pulse could not be felt, and the second sound of the heart was inaudible, slight pressure on the eye caused emptying of the arteries without pulsation. The veins were large and dark, visible in the finest divisions. The papilla was of a pale lilac tint.

Pyæmia and Septicæmia.

The occurrence of a general inflammation of the eye in cases of septicæmia of various kinds, "metastatic panophthalmitis," has long been known. The ophthalmoscope has revealed the fact that slighter retinal changes are present in a large proportion of the severer forms of these affections, and constitute a symptom of considerable importance. The knowledge of their character is largely due to the labours of Heiberg,[1] Roth,[2] and especially of Litten.[3] All forms are most common in the septicæmia of puerperal women, but are also met with in other cases.

Panophthalmitis.—The general inflammation of the eye, "pyæmic or metastatic ophthalmia," is usually attended with suppuration in the various structures—iris, choroid, retina,

[1] "Med. Centralblatt," 1874, No. 36.

[2] "Deut. Zeitschrift für Chirurgie," 1872, p. 471; Nagel's "Jahresbericht," 1872, p. 349.

[3] "Charité Annalen" for 1876, p. 160.

vitreous—with rapid destruction of the eyeball. It was shown by Virchow (1856) to depend upon septic embolism, and later researches have fully confirmed the fact. Plugs in the vessels, containing micrococci, have been frequently found. It is often associated with endocarditis, but may occur independently of this.[1] Even in such cases, however, the presence of infarcts in other organs, and of suppurating thrombi in the source of the septicæmia, demonstrate the probability of embolism, although not directly from the heart. It is well known that the small pyæmic emboli may pass through the lungs to the general system. The septic inflammation, once excited, has strong tendency to spread; it may start from the choroid or the retina, and probably from other structures. Litten, when the process commenced a short time before death, found plugging in one case of choroidal, and in another of retinal vessels. When the retinal vessels are plugged, hæmorrhages are invariable, as Virchow demonstrated, and the process, with opacity of the retina and vitreous, may be watched with the ophthalmoscope. The opacity of the retina depends apparently in most cases on acute degeneration. It was found by Roth to contain granule cells, although the other structures of the eye were infiltrated with pus. A layer of pus has been found on the surface of the retina, and even in the nerve-fibre layer. Rarely, the changes have been limited to a small area of the retina and adjacent choroid.

It is probable that this severe ocular inflammation is always produced by the agency of septic organisms circulating in the blood, such as have been found in the vessels of the eye in many cases.

The affection is met with in one or both eyes, it may be unequally. It occurs only in intense forms of septicæmia, commonly not long before death. In rare cases it may occur when the general symptoms of the disease are not advanced, but is generally of fatal augury.

Retinitis Septica.—Roth has described a peculiar form

[1] Litten: *loc. cit.* Case 8; Meckel: "Charité Annalen," Bd. v.; Virchow: "Ges. Abhand.," p. 539; Schmidt: "Arch. f. Ophth.," xviii. p. 1.

of retinitis in cases of pyæmia, characterized by the appearance of small white flecks in the neighbourhood of the papilla and macula lutea, varying in number, and occurring in most cases in both eyes. Sometimes small hæmorrhages were present. The white spots were found to consist of groups of swollen nerve-fibres, among which were granule cells, fattily-degenerated capillaries, and pigment granules. The affected spots were of small size, and showed little tendency to extension, or to the involvement of the vitreous or choroid. The affection was met with especially in cases in which decomposition was occurring in inflamed parts, such as extensive sloughing with secondary suppuration, and especially in pronounced septicæmia. It was found also in one case of putrid bronchitis.

Retinal Hæmorrhages constitute, however, by far the most common and most important change in the fundus in cases of

FIG. 86.—RETINAL HÆMORRHAGES IN A CASE OF ACUTE ULCERATIVE ENDOCARDITIS AND CHOREA.

The rounded hæmorrhage at the lower part of the figure has the characteristic white centre.

septicæmia. They usually accompany the suppurative panophthalmitis, especially when the process commences in the retina.

They may also occur in the form described by Roth. But they may exist alone, without any sign of retinal inflammation, and as such constitute the most common ophthalmoscopic change in these cases. They have been very carefully studied by Litten, in cases of puerperal septicæmia, in which they almost invariably occur during the last two or three days of life. They are always bilateral, round, or irregular in form, and of variable size, sometimes very large. They are commonly adjacent to vessels, especially veins, but occasionally are situated apart from visible vessels. Most of the round extravasations present pale or white centres, which are often distinct as soon as the hæmorrhage appears.

In some of the cases in which these hæmorrhages were seen, there was endocarditis, but not in all. In no case could Litten find any plugging of the retinal vessels, and from these facts he concludes, with Roth, that embolism is not the cause of these extravasations. But micrococcal obstruction gives rise to similar appearances, and this condition is difficult to exclude, and may be independent of endocarditis. When this exists, it probably varies in its degree of malignancy in different cases. In some cases of pyæmia, similar hæmorrhages have been noted on the mucous membrane of the conjunctiva or mouth (Litten, Leube).

From the fact that the retinal hæmorrhages often precede death by a few days only, they afford important and very grave prognostic information. Now and then they are useful also in diagnosis, since they are apparently not found in acute specific diseases, even in those severe cases in which cutaneous hæmorrhages are present. Litten mentions two cases of women admitted with high fever, cutaneous extravasations, and cardiac murmurs. One had been recently confined. They had the aspect of cases of septicæmia rather than of typhoid, but the absence of retinal extravasations led to a diagnosis of typhoid fever, which, in each case, was confirmed by a post-mortem examination. I have seen one case in which the presence of retinal hæmorrhages was of considerable assistance in establishing the fact that

a post-puerperal illness, supposed to be typhoid, was really septicæmia.

The effect of the retinal hæmorrhages on vision can rarely be ascertained with exactness, on account of the general state of the patients, but they seem to cause little impairment.

Purulent meningitis sometimes occurs in cases of septicæmia. In one such case, recorded by Leube,[1] there were retinal extravasations, but after death intense inflammation of the optic nerves adjacent to the inflamed membranes was found.

OPTIC ABIOTROPHY.

(Family Optic Atrophy.)

Since some cases were discerned and described by Leber, much attention has been given to the form of optic atrophy which occurs in families, sometimes through more than one generation, soon after adult life is attained.[2] Similar cases occur in sporadic form, isolated, as do other family maladies. The facts suggest that the inherent vital energy of these structures is inadequate to maintain their nutrition much beyond full development, so that they gradually fail and degenerate. They fail from imperfect life, from abiosis, in what may be designated abiotic atrophy, or abiotrophy.[3] The same atrophic failure is met with in other parts of the nervous system, as in Friedreich's disease, and conspicuously in the muscles in the varieties of muscular dystrophy.

This optic abiotrophy has been already described (p. 105). It is here referred to chiefly to denote its place among the many maladies by which the optic nerve suffers. Yet it is noteworthy that even vital failure is often associated with extraneous influences. Even in the cases in which the

[1] "Deut. Arch. für Klin. Med.," Bd. xxii. 1878, p. 263.

[2] They have been described, among others, by Habershon ("Trans. Ophth. Soc.," 1888, p. 190) and (Snell, *ib.* vol. xvii.).

[3] *See* Gowers, Lect. on "Abiotrophy," "Lancet," April 12, 1902; and "Lectures," 2nd Series, Churchill, 1904, p. 108.

family disposition is most marked, the onset is often the immediate sequel to some adventitious cause. It follows some acute specific disease, or, in males, excessive smoking, and this in cases in which the family tendency is so marked as to compel us to regard the excitant as merely such, an opinion which is confirmed by the slow progress of the atrophy after the cessation of the immediate influence. In such cases the interstitial tissue undergoes overgrowth, in consequence of the solidarity of the vital tendency of the two structures, both of which arise from the same embryonal elements. When the vitality of the higher, neural, elements fails, that of the residual, neuroglial, elements becomes exuberant, and may even display an energy which carries it beyond the strict limits of the neural failure. Thus we are able to understand some features of this form which at first seem mysterious. It must be remembered, also, that the defect in vital endurance varies in degree and in extent, even within the range of the tissue that presents it. How grave it may be we cannot know until it is revealed by time, but the influence of a powerful excitant may at least afford ground for hope, and indicate room for treatment. It should be remembered also that, in such maladies, therapeutic measures, when most effective, may only prevent further failure.

THE OPHTHALMOSCOPIC SIGNS OF DEATH.

The stoppage of the heart's action, and the consequent arrest of the circulation of the blood, which constitute the chief events in the cessation of systemic life, lead to striking changes in the fundus oculi, changes which are among the most unequivocal signs of death. Attention was first called to them by Bouchut in 1863,[1] and they have since been studied by many observers, especially by Poncet,[2] Arlidge,[3] and Gayet.[4]

[1] "Traité des Signes de la Mort," 1863.
[2] "Arch. Gén. de Méd.," 1870, p. 408.
[3] "West Riding Asylum Reports," i. 1871, p. 73.
[4] "Ann. d'Oculistique," t. lxxiii. 1875, p. 5.

As the heart's action is failing, the arteries may be observed to diminish in size (Arlidge). On the cessation of its contractions, the diminution in their size becomes more marked. A few minutes after death the capillary redness of the disc disappears, and its surface becomes very pale; in it, however, the central cup, if present, may still be conspicuous. The arteries quickly cease to be recognizable upon the disc, appearing to commence at its edge, beyond which they are at first distinct, narrow but otherwise of normal appearance. The veins may present normal characters at first, or have the same aspect as the arteries. The columns of blood within them soon become interrupted and broken into segments, which give the vessels a beaded appearance. The indistinctness of the arteries, due to their contraction emptying them of blood, quickly extends towards the periphery, and in the course of half an hour, sometimes in ten minutes, they are unrecognizable. The veins remain distinct, but in most cases the beaded appearance increases. The choroid, during the first few minutes, presents nearly its normal tint, but this quickly lessens in intensity, and its colour depends on the amount of pigmentation. In dark eyes it acquires a yellow-brown colour, in lightly pigmented eyes it gradually assumes a pale, reddish-yellowish, sometimes a greyish, tint. Commencing opacity of the retina may sometimes be observed, and may be accompanied by a red spot at the macula lutea (Gayet), due to its freedom from opacity, and similar to that seen in embolism of the central artery.

These appearances persist until, generally after five or six hours, the progressive opacity of the media prevents further observation.

APPENDIX.

HOW TO SKETCH THE FUNDUS OCULI.

NOTHING gives dexterity in the use of the ophthalmoscope so quickly and so effectively as an attempt to draw what is seen, and nothing gives ability to recognize details with accuracy and perceive every feature presented, as a habit of drawing does. Yet ophthalmoscopic drawing is hardly ever practised. It is supposed to be difficult, but it is neither difficult nor does it need any ability or facility for ordinary drawing. The process is within the reach of every student, and it may be well therefore to describe the method which is most useful. It is indeed so simple as scarcely to need even descriptive instruction, but it may be well not to assume that it can be discovered by each student for himself.

A pencil drawing should be first made, and from that either a more perfect pencil drawing on any paper that has a grain, on lined paper; or, what is better, a coloured drawing, which requires, however, a skill a pencil drawing does not need.

The disc should be drawn from $\frac{3}{4}$ inch to 1 inch in vertical diameter. The fundus should be observed first by the indirect method, and the outline of the disc made much larger than it appears, by a faint pencil line, and other simple pencil lines should indicate the position of the chief vessels that can be seen by this method, the veins being made darker than the arteries. This being done, the observer should continue and complete the drawing by the direct method. If he is drawing the patient's left eye, his pencil and materials should be on a small table to his right; if he is drawing the patient's right eye, this may be on his left or immediately in front of him, he sitting to the side of the patient. To continue the drawing, he must turn his paper upside down. This brings the disc, as drawn from the indirect image, into the position in which it appears in the direct image. Both arteries and veins must now be represented, as they are seen, with a double contour. After indicating more precisely the position and outline of the physiological cup, some one large vein should be selected, and its position at the edge of the cup and edge of the disc noted, and, if necessary, corrected.

Then its double contour should be marked by a broad pencil line on each side, with a very slight pencil tint between them, where the reflection is seen. This must be darkened wherever the reflection is lost in consequence of the vein being in some other plane than that at right angles to the line of vision, *e.g.*, when passing over some prominence, or receding into the physiological cup.

The branches of this vein and the artery accompanying it should then be drawn in like manner, the artery being represented by paler pencil lines and its central reflection being left white. Great care must be taken to depict accurately the relative width of each vessel, both on the surface of the disc and beyond its edge. The width of the vein first drawn must also be compared with the size of the cup and the disc generally, and this vein taken as a standard with which to compare the others; in this manner the precise representation of the size of the various vessels—a very important point—is much facilitated. Each of the other vessels should then be drawn in the same way; as a rule, each different vein first and then its artery.

It is necessary to indicate many features by some arbitrary signs, or by reference-indications to words written on the margin. Among the points to which attention should be given, is the presence of white lines along the vessels, due to the tissue of the wall. It will be remembered that when we speak of the vessels we are drawing we mean only the columns of blood within them; we cannot see the vessels themselves except in the appearance now referred to. These white lines may be indicated conveniently by dotted lines outside the darker ones. Where the vessel becomes indistinct, there should be, of course, a corresponding indistinctness of the lines representing it; but often this is not enough to indicate the degree of concealment, and then lines may be drawn across the vessel.

The shape of the central cup should be carefully attended to, as well as its depth, and the course of the vessels down its side. The latter, together with the change in their aspect, represents the steepness of the side, and shows it at once to one who is used to ophthalmoscopic examination. The manner in which the vessels disappear at the bottom of the cup varies, and must be carefully indicated in the sketch. Often they gradually pass from view as they penetrate the tissue, which at length conceals them; then they not only become fainter, but also narrower, because at the edge of the column the depth of blood is less and its tint is less deep; hence the margin becomes first concealed, and the vessel seems to lose in width as it loses colour.

In normal discs it is common to have a little softening of the edge where the chief vessels cross above and below, striated in character, and due to the large number of nerve-fibres which cross the edge there. This may be indicated by faint lines across the edge.

In general it is convenient to indicate all features that are white by dotted lines. Thus the outline of a white spot or patch should be made with dots instead of by a continuous line. If there is a difference in the

intensity of the whiteness in different parts of the area, it must be drawn black with the pencil, the intensity of the white tint being inversely indicated by the intensity of the pencil shading, and the fact that it is white indicated by a dotted line around it (not then indicating the position of the edge, which should be the edge of the shading), or else by a written indication adjacent to the spot or in the margin.

So, too, the sclerotic ring should be shown by dotted lines, and also the outline of a posterior staphyloma. White spots of albuminuric retinitis may be made dark, without the risk of error, if—as should always be the case—the more finished drawing is made without any delay, to prevent the features of the case being forgotten.

The finished drawing should be carefully made from the completed sketch, and should be, if possible, repeatedly compared with the actual object. It may be, and should be if possible, in colour. If in wash monotone, sepia is the most convenient, and was employed for all the drawings reproduced by the autotype (gelatine) process in the following plates. The defect of all monotone is the absence of distinction between the grey and the red. For phototype drawings to print with text, sepia answers well, but has to be reproduced by the expensive "tone" process, in which a tint is broken up into dots by a screen in the process of photography. The cheaper "line" blocks are available if the drawing is made on paper with raised tinted lines, which yield, on erasure, first a lighter tint consisting of dots, and ultimately white. That is the method I have used for the figures in the text. To get an adequate tone-effect, the drawing must be made larger than the block desired, according to the coarseness of the lines, and reduced in the photographic reproduction.

Some remarks on the production of coloured drawings may be acceptable to those who have no knowledge of water-colour work. The following suggestions embody the lessons obtained by attempts to make such drawings without any knowledge of the art.

Colour is necessary for a perfect representation of the aspect of the fundus, but care must be taken not to lose sight of form, even in its minutiæ. The knowledge of pigments that is requisite is best gained by experiment, and it is wise to obtain pigments of the proper tints, if possible, and to mix as little as possible. The "wash" is the chief thing that is needed, and this is easily acquired with a little practice. Details only need patient care. Fortunately, in the features that can only be represented in colour, the exact shade of colour is comparatively unimportant. The precise tint of the choroid varies so much, that correspondence with nature, so far as relates to the individual eye, is not appreciated, indeed is not observed by most persons, and hence a slight divergence from nature is equally unobserved. Of course the limits of the variations that are met with in the tint of the choroid must not be exceeded, and in the cases in which there is a choroidal change to be depicted the amount of pigment in the choroid must be carefully observed and represented.

The chief difficulties with the colour are—to obtain a natural appearance as regards texture, and to obtain evenness of the tint. The beginner may take comfort in the fact that any defects can be concealed, to a large extent, by details afterwards added. The conspicuous forms of the vessels prevent even a considerable unevenness from being noticed. Professional artists use Bristol board for the coloured drawings, and the surface of this, after having been well washed, is not so bad as might be imagined. A thin hot-pressed paper, damped and stretched, answers well, as the grain thus obtained is finer than that of a paper which has not been hot-pressed, and yet it takes the colour well. All the sepia drawings in this work were done upon such paper.

The difficulties with the colour arise from two causes—(1) getting the proper tint of red; (2) putting the colour evenly on the paper. They may thus be overcome: (1) Do not use vermilion. Any colourman will give you a choice of reds sufficiently large to represent every possible tint the human blood can assume. A little light red, added to carmine, answers very well; but if the tint of any drawing which seems near nature, is taken and compared with sample tints, no difficulty will be experienced in selecting one that corresponds sufficiently closely. (2) Evenness of surface is best obtained by washing the coat of colour with water, after it is quite dry, and then placing blotting paper on it. This, of course, takes off a good deal of colour; but by laying on another coat (before the surface is quite dry), and repeating the process three or four times, a very even tint is obtained without difficulty. If any inequalities are seen they may be removed by "stippling" with the point of a brush. General stippling may be necessary when there are peculiarities of the choroid, for the distribution of its pigment can only thus be represented. The method of successive coats has an advantage in the softness of the edge of an inflamed disc that can be obtained. It is better to take out, from each wash, the area of the disc, by the end of a small roll of blotting paper, than to leave it, on account of the hardness of the edge which the latter method involves (except in the hands of a skilful artist, for whom these directions are not intended). By softening the edge of the colour with a wet brush, then pressing blotting paper firmly on it, and repeating the process several times, any part may be made as soft in its gradation as can be desired. Sharp-edged, perfectly white spots may be produced with a penknife.

These directions will seem absurd to the skilled artist, but there may be some who strive, without skill, to whom they will be useful.

DESCRIPTION OF PLATES.

The ophthalmoscopic illustrations contained in the following plates are autotype facsimiles of sepia drawings by the author, reduced in size in the process. All, with the exception of Pl. IV. Fig. 1, and Pl. X. Fig. 2, are made from the direct image, although in some instances they have been much reduced.

The disadvantage of absence of colour has some compensation in the greater degree in which attention is directed to changes in form, the importance of which it is difficult to over-estimate. But it is essential that they should be studied with the aid of the following descriptions.

PLATE I.

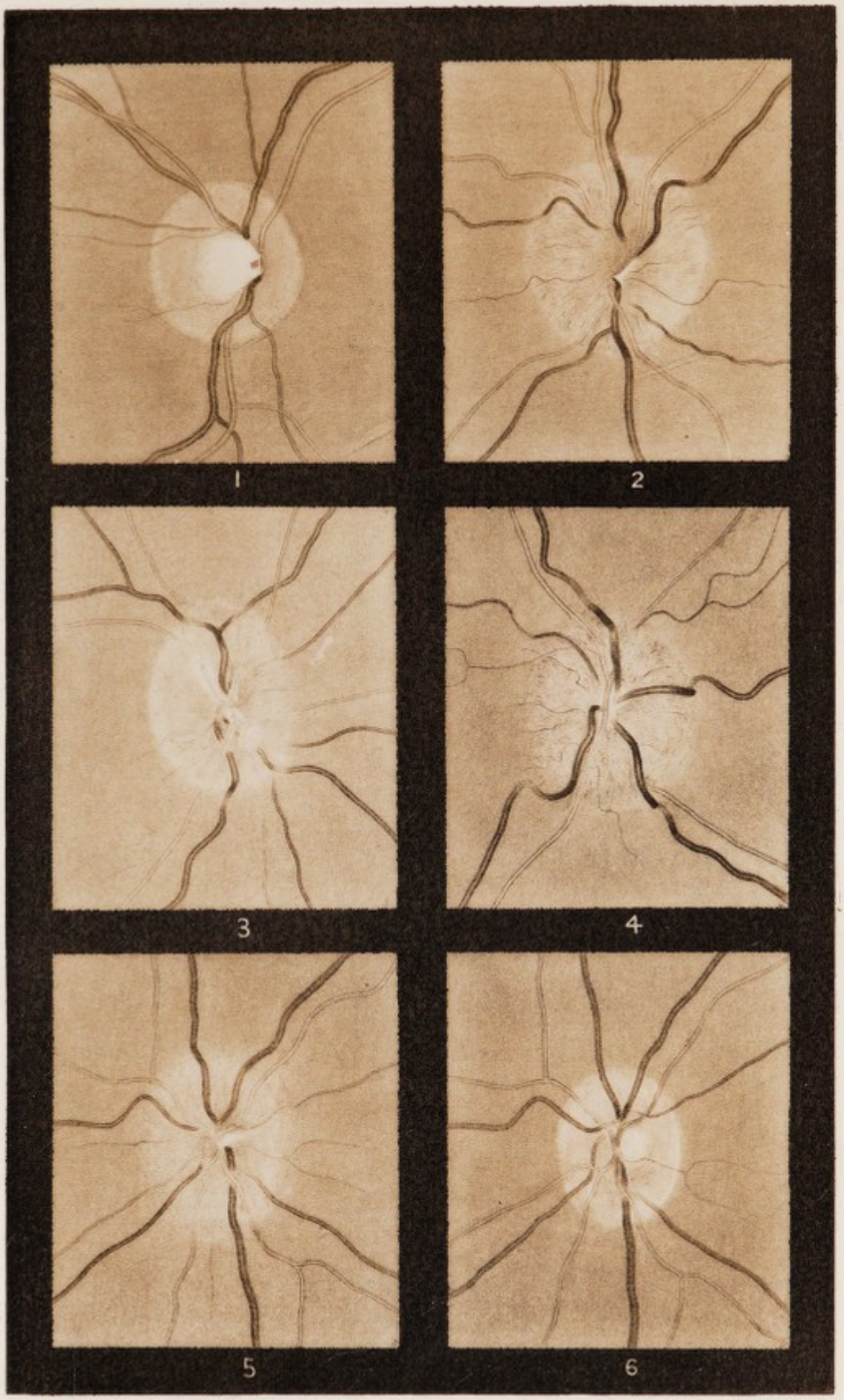
Figs. 1 & 2.—*Right and left optic discs; caries of sphenoid bone, with secondary meningitis.* (*See* p. 170.)

Fig. 1.—*Right optic disc.* Characters normal. Outline clear; central cup deep; vessels lost to view as they pass down its sides. The termination of the vein can be dimly seen in the middle, beneath the nasal edge of the hollow. (Vision normal.)

Fig. 2.—*Left optic disc.* Well-marked neuritis. Edge of disc invisible; concealed by a reddish-grey swelling, which extends beyond the normal limits of the disc. The central cup is encroached upon but not quite obliterated, a small area of white reflection from it being still visible. Vessels of normal size. The veins emerge from the central depression, one, which passes directly upwards, being partly concealed at its emergence; they present conspicuous curves, and lose their central reflection as they course down the sides of the swelling. The arteries present a straighter course, but cannot be easily distinguished upon the papilla. The degree of swelling is moderate; it presents fine striation, partly due to minute radiating vessels and partly to the nerve fibres. (Vision quantitative only.)

Fig. 3.—*Neuritis in cerebral tumour. Man aged twenty-four.*

PLATE I.



W.R.C. del.

AUTOTYPE.

Right optic disc. Outline recognizable on the temporal side, although not sharp; concealed on the nasal side. Tint, greyish-red, finely striated. Swelling distinct but slight. The veins, of normal size, lose their central reflection on the sides of the swelling and are concealed just beyond its edge; one, which passes downwards and to the right, is concealed near the middle of the papilla by a white opaque spot. The arteries are narrow, and near the middle of the papilla are bordered by white lines. (Vision could not be ascertained.) For the microscopical appearances at a later stage, when the inflammation was greater, *see* Figs. 11 (p. 56), 12 (p. 57), 20 and 21 (p. 60).

FIG. 4.—*Optic neuritis in cerebral tumour: tubercular masses in cerebral hemispheres, cerebellum, and the other eye. Boy aged eight.*

Left optic papilla. Disc concealed by very prominent swelling with a marked central depression. Veins large; they form conspicuous curves as they course down the steep sides of the swelling, some being even lost to view. Beyond the edge they are obscured for a short distance. Arteries partly concealed. Minute red stippling in swelling, but no hæmorrhages. Much white tissue about the vessels in the central depression. Vision: slight failure only.

FIGS. 5 & 6.—*Optic neuritis in traumatic meningitis and after recovery. Right eye.* (Case mentioned on p. 176.)

FIG. 5.—*Appearance ten days after the injury.* A pale red, striated opacity conceals the whole disc, the edge being nowhere visible; prominence slight but distinct. The central cup is not quite obliterated; its white reflection is visible at the bottom of the central depression. Veins of normal size; the upper branches, where the swelling is greatest, lose their reflection at the side. Arteries of normal course. Vision: no evidence of impairment.

FIG. 6.—*The same disc a month later*, presenting normal characters. Edge clear and fairly sharp; sclerotic ring visible on nasal side (to the right). Central pit clear and apparently normal; steep on the temporal (left), sloping on the nasal side. The edge of the disc is seen, from its relation to the vessels, to be considerably within the limits of the swelling shown in the preceding figure. The vessels have a normal course. Vision normal.

PLATE II.

FIGS. 1 & 2.—*Optic neuritis in a case of probable syphiloma of brain, and disc after recovery. Right eye. Man aged thirty-three.*

FIG. 1.—*Inflamed papilla.* Disc concealed by a prominent, red, striated swelling about twice the normal diameter of the disc. A slight central depression can be seen. The veins, not larger than normal, appear dark as they pass down the sides of the swelling. A white patch lies across and conceals one which passes downwards. On the lower part of the swelling is a small hæmorrhage. Vision: No. 1 Jäger at six inches with a little difficulty.

FIG. 2.—*The same disc three months later*, presenting very little trace of the preceding inflammation. Outline quite clear and sharp; sclerotic ring distinct; no disturbance of adjacent choroid. Central cup small but not apparently "filled-in," as the veins can be traced down its sides to their junction at the bottom. Some of the arteries on the disc are accompanied by white lines, especially one which curves downwards. A comparison of the vessels with those in the last figure will show how much they were altered in their course by the swelling. [A vein which passes upwards and to the left has by an error been drawn as an artery]. Vision normal.

FIGS. 3 & 4.—*Optic neuritis from cerebral syphiloma, and same disc after the subsidence of the neuritis. Woman aged thirty-seven.*

FIG. 3.—*Inflamed papilla.* Disc concealed under a swelling of moderate prominence, about twice the diameter of the normal disc, concealing the veins and arteries. Colour red, and finely punctate in the centre; greyish-red and striated on the peripheral portions of the swelling. The central reflection of the veins is lost as they slope down the sides of the swelling. Veins a little larger than normal; arteries nearly of normal size. One vein, which courses from below, passes over the disc more superficially than the others and presents a double curve. There is a small hæmorrhage in the centre of the disc, and a faint white spot to the right of the centre.

Vision, $\frac{1}{13}$ and $\frac{8}{10}$. Field normal. Blind spot double normal size. (Fig. 40, p. 68).

FIG. 4.—*The same disc two months later.* Neuritis gone; outline of disc clear in whole circumference. There is a fringe of pallor beyond the nasal edge, to the left (atrophy of choroidal pigment). Tint of

PLATE II.

W. R. G. del.

AUTOTYPE

disc normal, but too uniform, and the disc has a "filled-in" aspect, the two lower veins being narrowed and partly concealed by new tissue at the centre left by the inflammation. The normal central "cup" is being re-established, as shown by the curve at the central end of the lower vein; the bright reflection is lost as the vein curves down the edge of the cup; at the centre it is still almost concealed. Arteries normal.

Vision the same.

FIGS. 5 & 6.—*Subsiding neuritis and subsequent atrophy (cerebral syphiloma); process of obliteration of vessels. Left eye. Man aged thirty-four.*

FIG. 5.—*Neuritis subsiding.* A month previously intense inflammation with hæmorrhages. Now a pale reddish-white prominence remains, with soft edges, paler in the centre than at the margin. The veins, large and dark, curve over the side of the swelling, and are obscured just beyond the edge. The arteries are small and partially concealed by the new tissue. On the surface several vessels are seen in process of obliteration. One, apparently an artery, ends suddenly at a small extravasation, and the terminal portion of the vessel is very dark, as if plugged. From the central portion of the vessel two small branches proceed. Sight quite lost. Voltaism causes no stimulation.

FIG. 6.—*The same disc six weeks later.* The swelling has subsided almost to the level of the retina; the surface of the disc is very pale, the centre being a little whiter than the rest. Veins and arteries are somewhat smaller than normal, the latter especially. The veins have now a straight course, and the arteries can be traced, although narrowed and obscured, to their emergence near the centre of the disc. The small vein in the other figure, which had a peculiar serpentine course, has disappeared. The artery, which appeared to be in part plugged, presents a very different appearance; the distal part has disappeared, and the proximal portion has dwindled in size to that of the branch, which appears to be carrying on the blood from it. Its origin from a larger trunk is now clear.

Vision: very slight perception of light; retina again sensitive to electrical stimulation.

PLATE III.

FIGS. 1 & 2.—*Optic neuritis (right and left eyes) in cerebral tumour.*

The patient was a man aged thirty-five, suffering from left-sided convulsions, beginning with a visual and auditory aura (the latter referred to the left ear), and from left hemianopia of both eyes. Subsequently coarse tremor came on in the left arm, with weakness, which gradually increased to complete left hemiplegia. The symptoms were found to be due to a tumour of the right hemisphere, in the parietal and temporo-sphenoidal lobes, extending inwards.

FIG. 1.—*Left disc.* Inner half veiled beneath a reddish striated swelling, sufficient to alter a little the course of the veins and partially conceal the arteries. The outer half is paler, and the outline can be seen, but is softened. A flame-shaped hæmorrhage lies across the edge. There is another small extravasation, near an artery on the lower margin. Vision normal, except for the hemianopia.

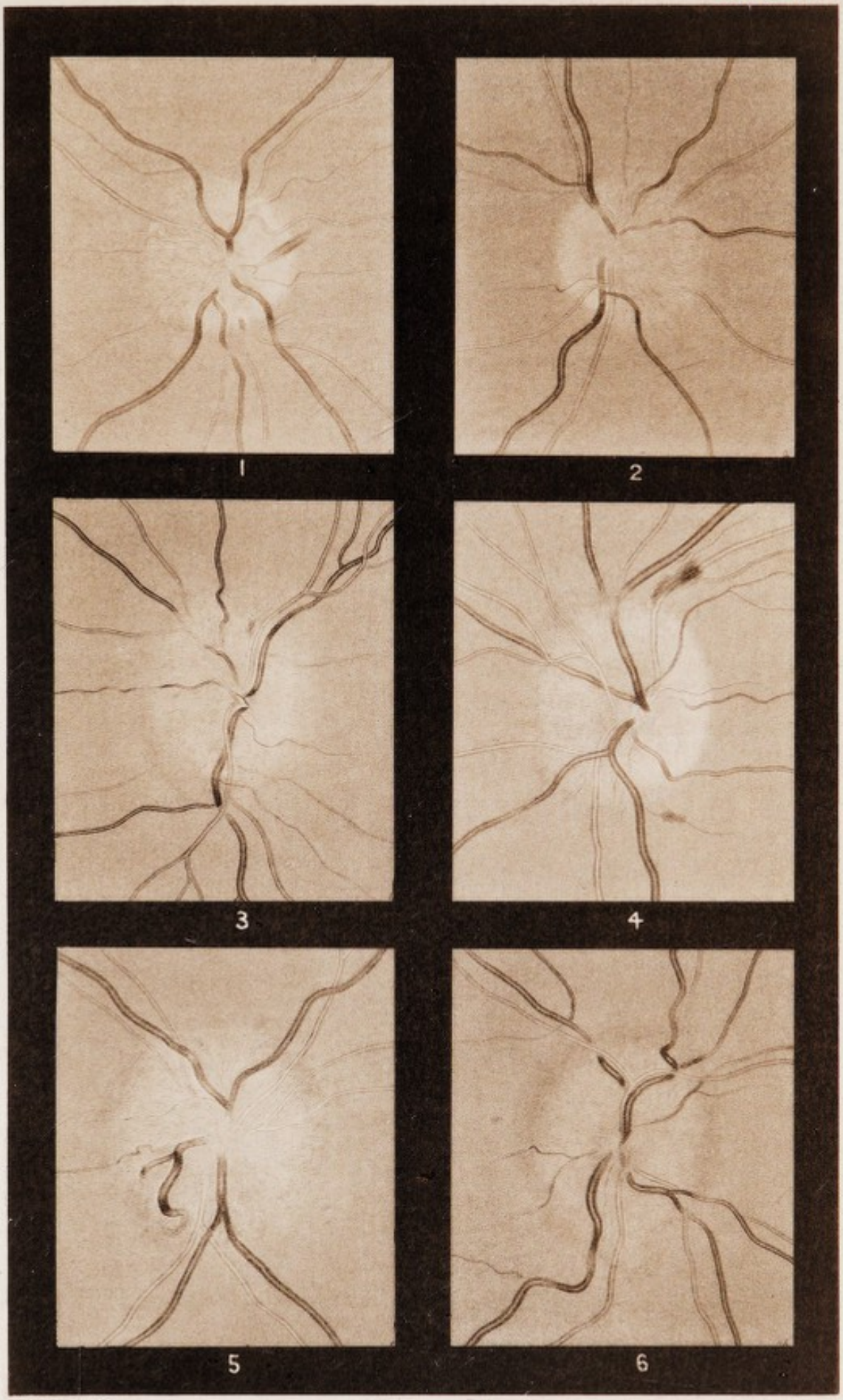
FIG. 2.—*Right disc* presenting a similar appearance; the inner half concealed, the outer visible, but not clear. No extravasation.

During about six months that the patient remained under observation not the slightest change could be seen in the discs except the disappearance of the hæmorrhages. A year and a half later (two years after the drawings were made), vision had entirely failed. The inner halves of the discs were still concealed under a reddish striation, but the outer halves had become grey, and their edges sharper.

FIG. 3.—*Optic neuritis in a case of cerebral syphilitic disease, causing left-sided weakness and convulsions beginning in the hand. Left eye.*

Disc completely concealed on nasal side (to the left), while on temporal side (to the right) the position of the edge can just be detected. Swelling moderate, altering the course of the veins, which can, however, be traced up to their emergence in the centre. The curve they present at the edge of the swelling is gentle, but sufficient to abolish the central reflection. The more abrupt backward curve presented, just beyond the edge of the disc, by a vein which passes directly downwards, is apparently determined by the position of an artery which crosses it, and which, a little above this point, in crossing it again, has again depressed it. A small vein which passes upwards and to the left (in the figure) is concealed for some distance

PLATE III.



W. R. G. del.

AUTOTYPE.

by the striated opacity. On the upper edge of the swelling is a small hæmorrhage. Neuritis bilateral. Vision $\frac{1}{5}$. Colour-vision normal.

FIG. 4.—*Optic neuritis in cerebral tumour, probably tubercular, causing left hemiplegia and hemianopia. Left eye. Girl aged fifteen.*

The temporal part of the disc (to the right in the figure) is clear Elsewhere the margin of the disc is concealed by a (reddish) striated opacity, of slight prominence. The veins are large, and those which pass downwards curve a little over the edge of the swelling, while one, which passes upwards, is concealed at the edge. A small striated hæmorrhage lies over an artery above, the striation being in the direction of the nerve fibres. The course of the artery is not changed. Below is a still smaller extravasation upon a minute branch of a vein. Vision: No. 10 Jäger at one foot; hemianopia; all colour-vision lost. Both eyes similar. The patient improved under treatment, the disappearance of the neuritis being the first sign of the improvement which was maintained for at least five years.

FIG. 5.—*Optic neuritis in cerebral tumour.* A glio-sarcoma, springing from the membranes, and compressing, without invading, the right side of the pons and right hemisphere of the cerebellum, causing right-sided convulsions beginning in the hand, and afterwards left-sided attacks beginning in the face; weakness and coarse tremor in the right limbs, deafness in the right ear.

The drawing was made as the neuritis was beginning to subside. Disc concealed beneath a considerable swelling, red and striated. Veins large (beginning to lessen in size), curve over the edge of the swelling. One which passes down cannot be traced beyond the edge, where it apparently disappears. (Even when the neuritis had subsided still more, its further course could not be detected.) Arteries small, not more than one-half the size of the veins. Vision 0.

FIG. 6.—*Optic neuritis in cerebral tumour. Right eye. Woman aged thirty-three.*

The neuritis was in course of subsidence. Swelling considerable, completely concealing the disc, pale, but still reddish, darker around the margin. The veins form conspicuous curves at the edge of the swelling, one above forming a double curve in consequence of passing beneath an artery just within the edge of the swelling. All the veins are concealed for a short distance beyond the edge, and then resume a normal course upon the retina. Vision: No. 12 Jäger at a foot. The neuritis subsided into consecutive atrophy, sight failing until vision was completely and permanently lost.

The exact nature of the intracranial condition remained obscure; there was a family history both of tubercle and cancer. Mental power failed greatly. The condition was similar in the two eyes.

PLATE IV.

FIG. 1.—*Intense optic neuritis, with retinal hæmorrhages, in a case of cerebral tumour. Right eye. Man aged thirty-six.*

The region of the optic disc is occupied by a large swelling, in width about four times the diameter of the disc. It is irregular in outline, with very steep sides, and is bounded in every direction by extravasations. Some of these are more or less striated, others have a sharp convex edge, due to their position in the overhanging edge of the swelling. The surface of the prominence is of about the same tint as the fundus. The vessels are concealed in the substance of the swelling, except one or two, the position of which is dimly seen. Most of them appear first beyond its edge, and are then of about normal size, but they form conspicuous curves, the deeper portions of which are concealed. They then assume a nearly normal course. The arteries are narrow, some being scarcely visible. Numerous hæmorrhages, small and striated, are scattered over the retina in the posterior half of the eyeball, except on the temporal side (to the left). In this direction the swelling reaches almost to the position of the macula lutea, in the neighbourhood of which are many minute white dots adjacent to the edge of the swelling. Vision 0.

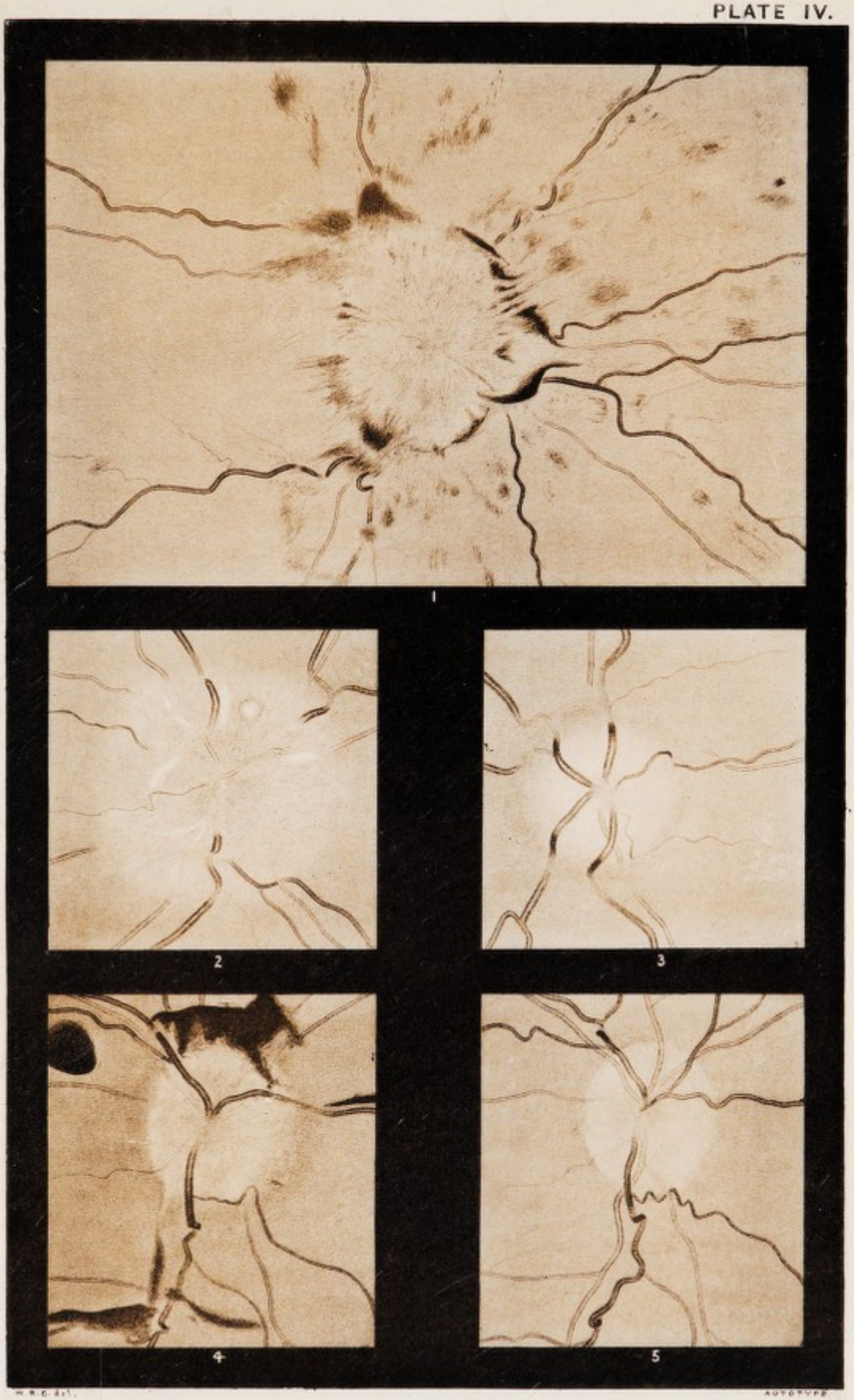
The patient died, but no post-mortem examination was permitted.

FIG. 2.—*Optic neuritis in a case of old fractures of the skull: inflammatory growths beneath them; at the base the results of previous meningitis. Man aged forty-nine.*

The position of the disc could be recognized by the indirect method of examination, but the edge was softened. The area of the disc was bright red, and beyond the edge was a pale halo. In the upright image the edge is completely concealed under a greyish-red swelling, of nearly three times the diameter of the disc, striated. Upon it are many white spots and lines (due to granule corpuscles, &c.), some of which correspond to the course of the arteries. One, above, is surrounded by a narrow zone of hæmorrhage. The vessels are concealed in the middle of the swelling; the veins more completely than some of the arteries. The course of the veins is very tortuous. Vision $\frac{1}{12}$. (The microscopical appearances are shown in Figs. 13, 23, 24, 33.)

FIG. 3.—*Neuritis subsiding into atrophy; slight retinal changes; tubercle of cerebellum. Left eye. Boy aged eleven.*

PLATE IV.



The disc is invisible beneath a pale, almost white swelling, depressed in the centre. Over this the veins curve. After sloping down its sides, they are concealed by the adjacent opacity of the retina for a short distance. One artery, which passes downwards, is visible on the surface of the swelling, but is also concealed beyond its edge. The other arteries appear only some distance from the edge. Midway between the retina and the macula lutea is a group of small white granular-looking spots, apparently just behind the level of a retinal vessel which passes among them. (They slowly lessened under observation. The swelling gradually subsided, the edges of the disc reappearing and its aspect becoming that of "consecutive atrophy." Its appearance is shown in section in Figs. 49, 50.) Vision 0.

He died from meningitis, probably tubercular. At the necropsy tubercles were found in the cerebellum and medulla oblongata.

FIGS. 4 & 5.—*Subsiding neuritis, recent hæmorrhages, and the same disc after recovery.*

The patient had been in the London Hospital, under the care of Dr. Hughlings-Jackson, suffering from the symptoms of cerebral tumour, and presenting intense optic neuritis. Under treatment the symptoms subsided and the neuritis gradually lessened, but during subsidence several fresh hæmorrhages appeared. He died some years later, and the brain presented softening of one anterior lobe, with the remains of an absorbed syphilitic gumma. Cicatrices were also found in the liver.

FIG. 4.—*Subsiding neuritis.* The outline of the disc can be seen, but is not clear; its surface is reddish in tint, and the swelling of the papilla is still considerable, as evidenced by the curves formed by the veins in passing over its edge. Several large extravasations are seen. One of these, below, follows the course of an artery. Another, above and to the left, is round, not striated, and therefore probably situated in the deeper layers and not in the nerve-fibre layer. Vision $\frac{1}{1}$: fields normal.

FIG. 5.—*The same, two months later.* The hæmorrhages have entirely disappeared. The disc is clear, and its swelling has almost subsided. But the tortuosity of the vessels has increased, probably on account of their permanent extension by the long-continued swelling.

PLATE V.

FIGS. 1 & 2.—*Unilateral optic neuritis; probably cerebral syphiloma. Man aged forty-four.*

FIG. 1.—*Left optic disc* concealed by a swelling—reddish, striated, depressed in the centre. The veins, a little larger than normal, curve over it, and some are concealed beyond the edge. In the central depression the veins pass behind the arteries and are unduly concealed by the swollen tissue. The artery which passes upwards is visible throughout; those which pass downwards are distinct at their emergence in the depressed centre, but are concealed by the swelling, to reappear at its edge. No hæmorrhages. Just beyond the edge of the papilla is a series of pale concentric lines parallel to the edge, due to the folds in the displaced retina; they are limited above and below by a small vein. Vision: counts fingers only.

FIG. 2.—*Right optic disc* presenting normal characters. A small deposit of pigment lies across a vein.

The right disc never became inflamed, but both discs eventually became atrophied, doubtless from an intracranial cause. It is possible that both nerves were damaged in front of the commissure, and that in one only did the inflammation descend to the eye.

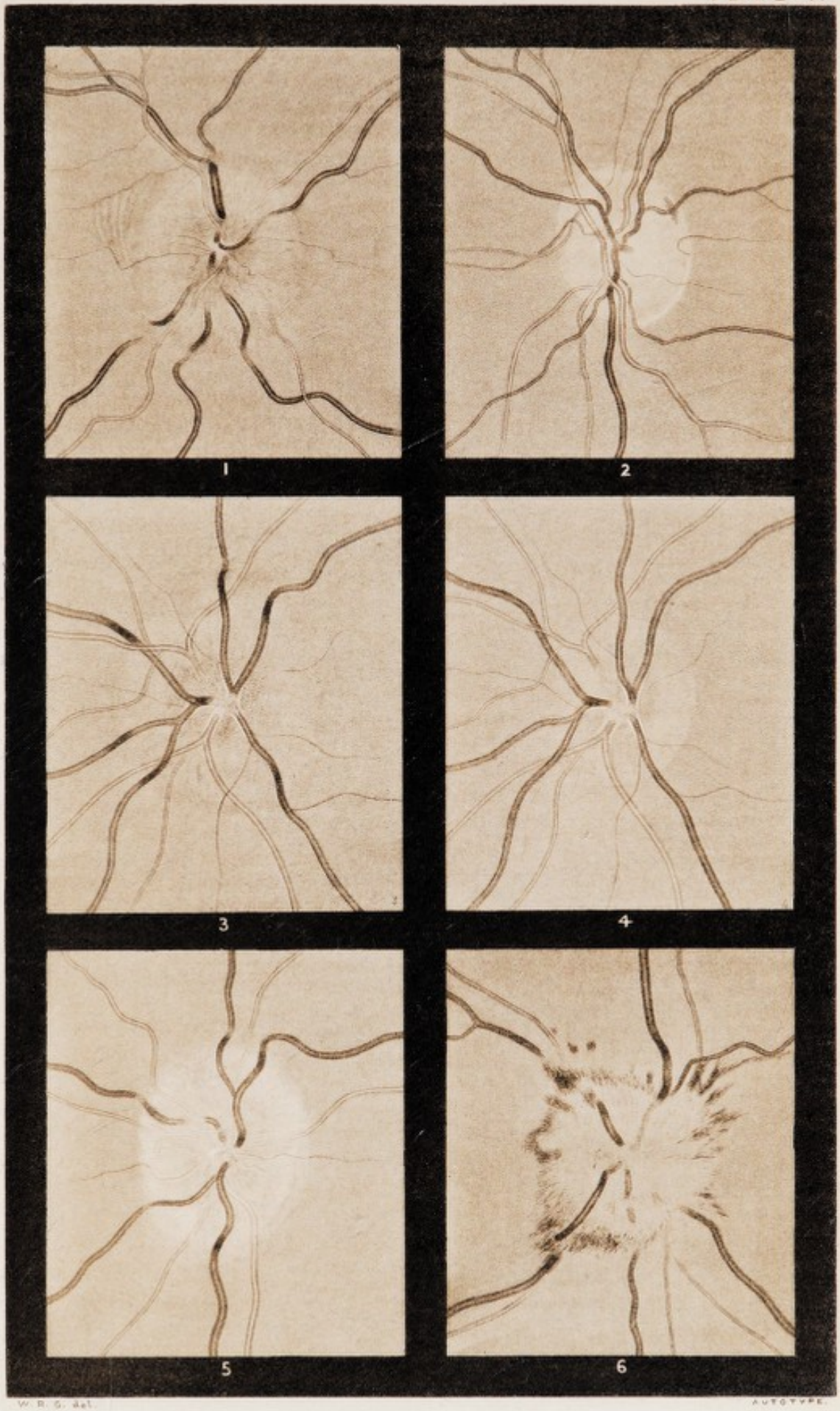
FIGS. 3 & 4.—*Very chronic optic neuritis, in a case of epileptoid convulsions. Girl aged fifteen.*

FIG. 3.—*Left disc.* Outline obscured by neuritic swelling of slight prominence: the centre stippled red, the periphery only slightly lighter in tint than the fundus. Veins, of nearly normal size, concealed in centre by whitish tissue which accompanies the larger trunks of both arteries and veins for a short distance. The double contour of the veins is lost on the sides of the swelling. Vision: No. 2 Jäger, spells No. 1. Appearances unchanged during four months' observation.

FIG. 4. *The same disc two years later.* All swelling is now gone. The outline is clear on the outer (temporal), indistinct on the inner (nasal) side. Veins large; at their junction in the disc they are even more concealed than before, and the white tissue about them is still very conspicuous. Vision, same.

FIG. 5.—*Optic neuritis in anæmia. Girl aged seventeen.*

The outline of the disc is lost under a pale, reddish-grey swelling, of slight prominence, a little larger than the disc. The veins, of normal



W. R. S. del.

AUTOTYPE

size, lose their reflection as they curve down the sides of the swelling, and some are obscured beyond its edge as they dip into the substance of the retina. Some of the arteries are concealed; others distinguishable with difficulty. There is a small white spot near the centre of the swelling. Vision (uncorrected) $\frac{1}{3}$.

Eyes hypermetropic. Both discs cleared and vision became normal. A few months later there was a temporary return of the anæmia and of the papillitis, but vision remained normal. (*See* p. 231.)

Fig. 6.—*Optic neuritis in a case of lead poisoning, with cerebral symptoms. Man aged forty-five.*

The disc is concealed by a swelling of moderate prominence, bordered by a fringe of striated hæmorrhage, and of a colour nearly that of the fundus. Veins, a little larger than normal. Arteries concealed by the swelling, and most of them very narrow on the retina.

His vision was considerably impaired, but could not be accurately tested, owing to his mental state. (*See* p. 258.)

PLATE VI.

FIGS. 1 & 2.—*Intense neuro-retinitis, probably idiopathic, in a chlorotic girl, leaving changes simulating albuminuric retinitis.*

FIG. 1.—*Right fundus oculi during the height of the neuritis.* The papilla presents a very large pale red swelling, five times the transverse and six times the vertical diameter of the disc. The peripheral portions are paler than the central. Its sides are steep, and marked by scattered striated hæmorrhages. Even the tortuous veins are almost completely concealed by the swelling, the highest parts of their curves alone being seen. At the edge all reappear, are greatly distended, and form conspicuous curves, most of them being again lost for a short space in the retina. The arteries are all concealed. Many extravasations fringe the swelling. The largest lies over a vein which passes downwards: it is striated, and has a paler centre. The pale edge of the swelling is irregular, presenting several projections, and beyond it are many pale spots in the retina. The swelling on the temporal (left) side reaches as far as the macula, and just beyond it is a group of white, rod-shaped spots, arranged in a fan-like manner, and evidently situated on the temporal side of the macula. There are a few small hæmorrhages here and there in the fundus beyond the limits of the swelling. Vision: No. 19 Jäger; considerable limitation of field, especially upwards and inwards. Loss of colour-vision except for red.

FIG. 2.—*The same fundus three months afterwards.* All the swelling has disappeared. The disc is clear, but has a "filled-in" look, the vessels being partly concealed at their emergence. Both arteries and veins are very narrow. The extravasations have disappeared; the white spots in the retina persist, but have a more granular aspect. Some extend along the vessels, and one or two have an irregular linear course as if corresponding to the position of choroidal vessels. Many white areas lie in the part of the retina around the disc which was formerly occupied by the swelling. The fan-like group of spots, adjacent to the macula, has become still more conspicuous, and others appear adjacent to them, and of similar arrangement; so that the aspect of albuminuric change is very closely simulated. Vision: quantitative perception of light only. (*See* p. 231.)

PLATE VI.

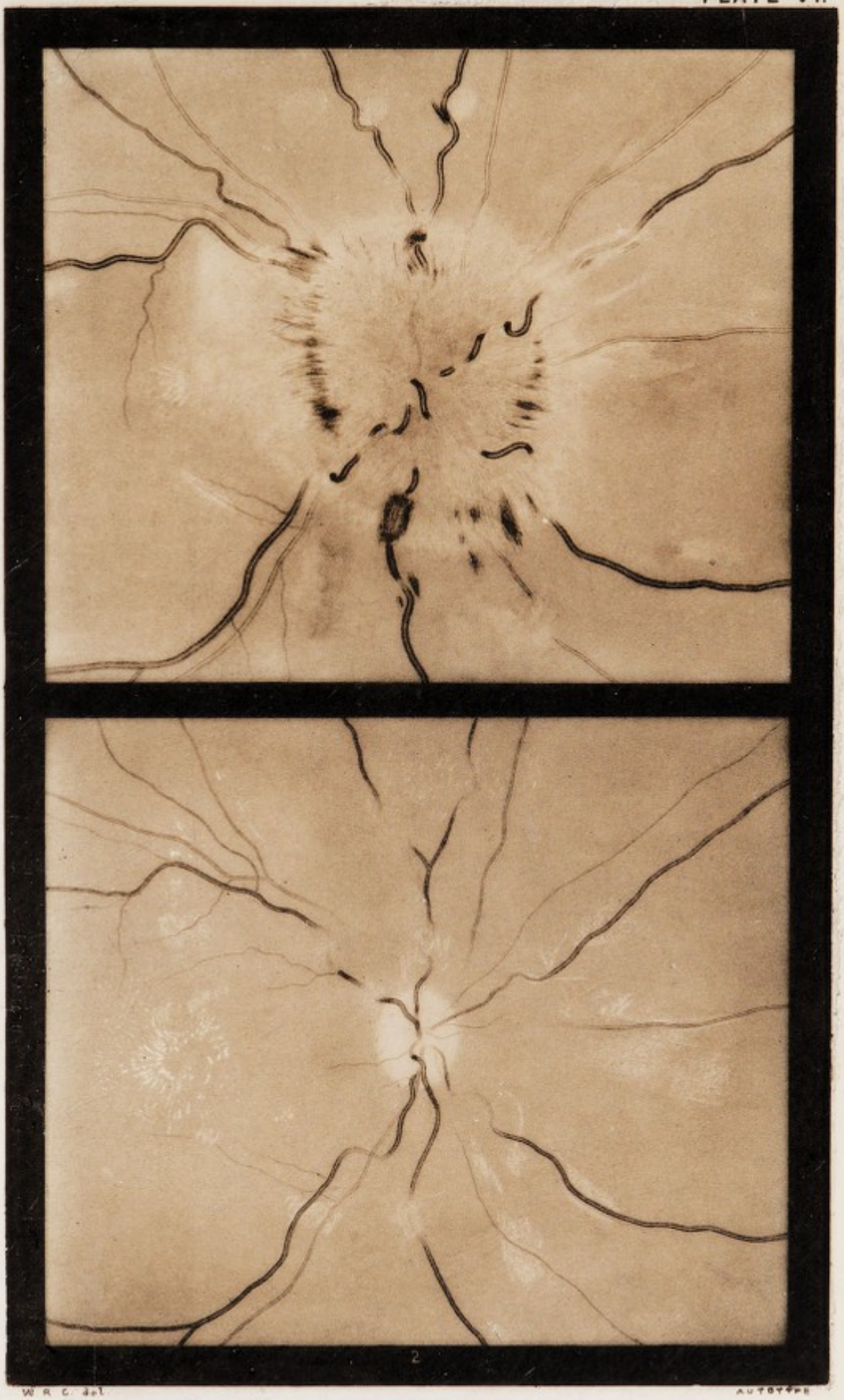


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PLATE VII.

FIG. 1.—*Hæmorrhage on optic disc in a case of renal disease, arterial disease, and acute cerebral lesion. Right eye.*

The optic disc is otherwise normal; the central cup distinct, narrow but deep; the arteries and veins of normal size. On the temporal side of the disc is a small extravasation, striated, extending on the retina about a disc's breadth. It has apparently arisen from the rupture of a small vessel, which can be traced to, but not beyond, the hæmorrhage. It had given rise to no symptoms.

FIG. 2.—*Neuritis albuminurica. Right optic disc of a man suffering from chronic renal disease, convulsions, and mental derangement.*

The disc presents the signs of slight but distinct neuritis. Its outline can be nowhere seen; there is slight swelling; the tint of the papilla is red, and the redness is striated. Many small vessels radiate from it on to the retina—more than is common in neuritis. The veins are rather large. The arteries are very narrow—not more than one-half the diameter of the veins. One small hæmorrhage exists on the temporal (left) edge of the disc. On the surface of the papilla are several white spots, irregular in shape. One is situated over an artery, another near the middle of the disc, and one near the lower edge. One small soft whitish spot can be seen on the retina near a vessel above the disc, but this is the only trace of retinal affection. (There were no spots near the macula lutea.) Vision: No. 12 Jäger.

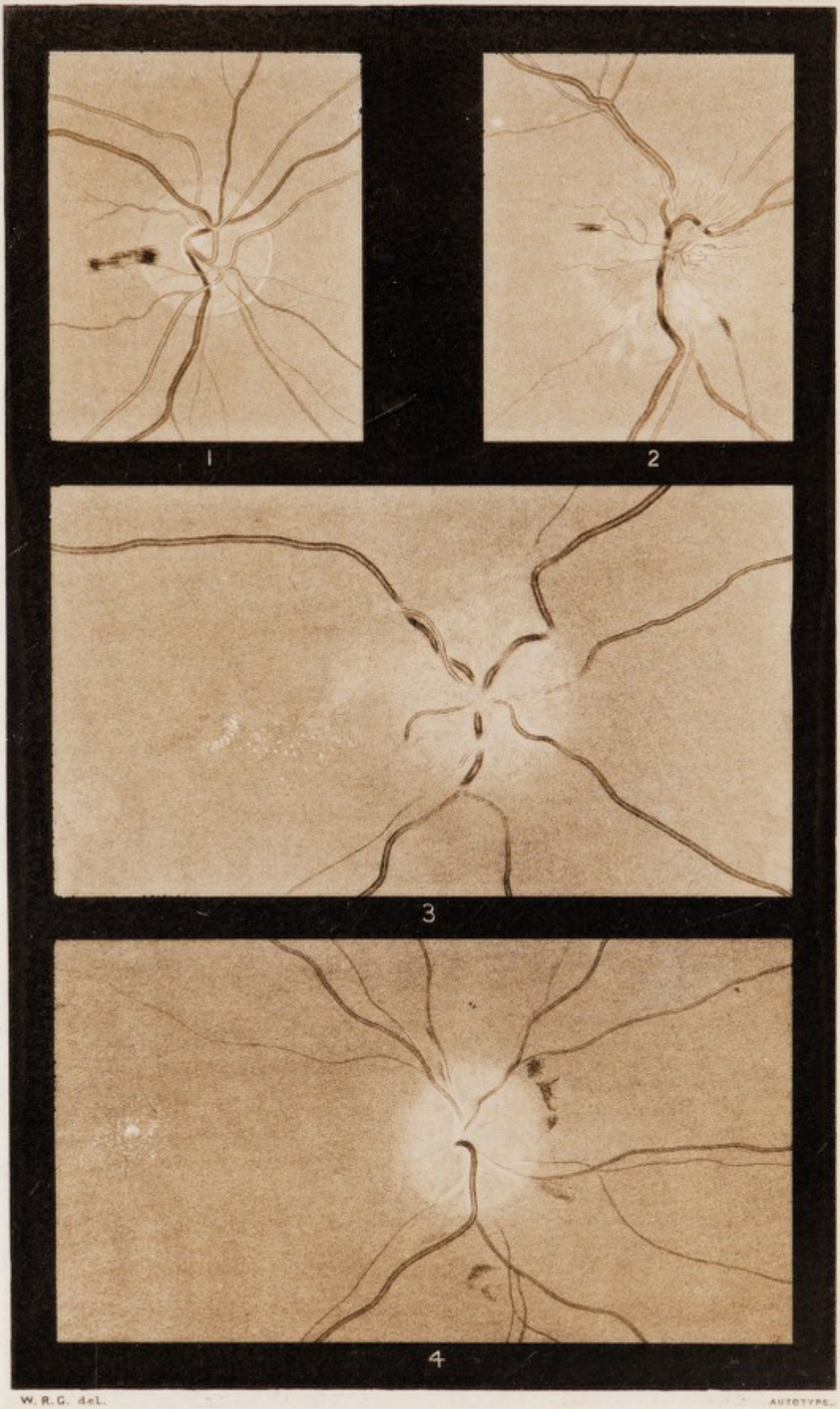
FIG. 3.—*Albuminuric neuritis in a man suffering from chronic renal disease (granular kidney) intense headache, and who died shortly afterwards of uræmia. Right eye.*

The disc is concealed by a considerable greyish-red swelling, stippled and striated. The veins are concealed at their point of emergence, curve over the prominence, and are again concealed at its edge. Beyond, they have a normal course and size upon the retina. The arteries, where visible upon the papilla, are a little below the normal size; but beyond, upon the retina, they are much smaller than normal, some being scarcely visible as mere lines, and two cannot be detected beyond the edge of the papilla. There are a few very minute shining white spots upon the centre of the swelling; between it and the macula are several white flecks, and close to the macula a few radiating dots and lines are arranged in a fan-like form. Vision: reads No. 6 Jäger. (*See* p. 93.)

FIG. 4.—*Subsiding albuminuric neuritis. The fundus of a patient suffering from chronic Bright's disease (probably granular kidney), with a pulse of very high tension.*

The papilla is slightly prominent, greyish-white, the edges of the disc being concealed by it. The veins are narrow and the arteries extremely small, recognizable only in narrow lines. One or two small extravasations are seen near the disc, and farther off are several small collections of pigment, probably the remains of former extravasations.

PLATE VII.



W. R. G. del.

AUTOTYPE.

PLATE VIII.

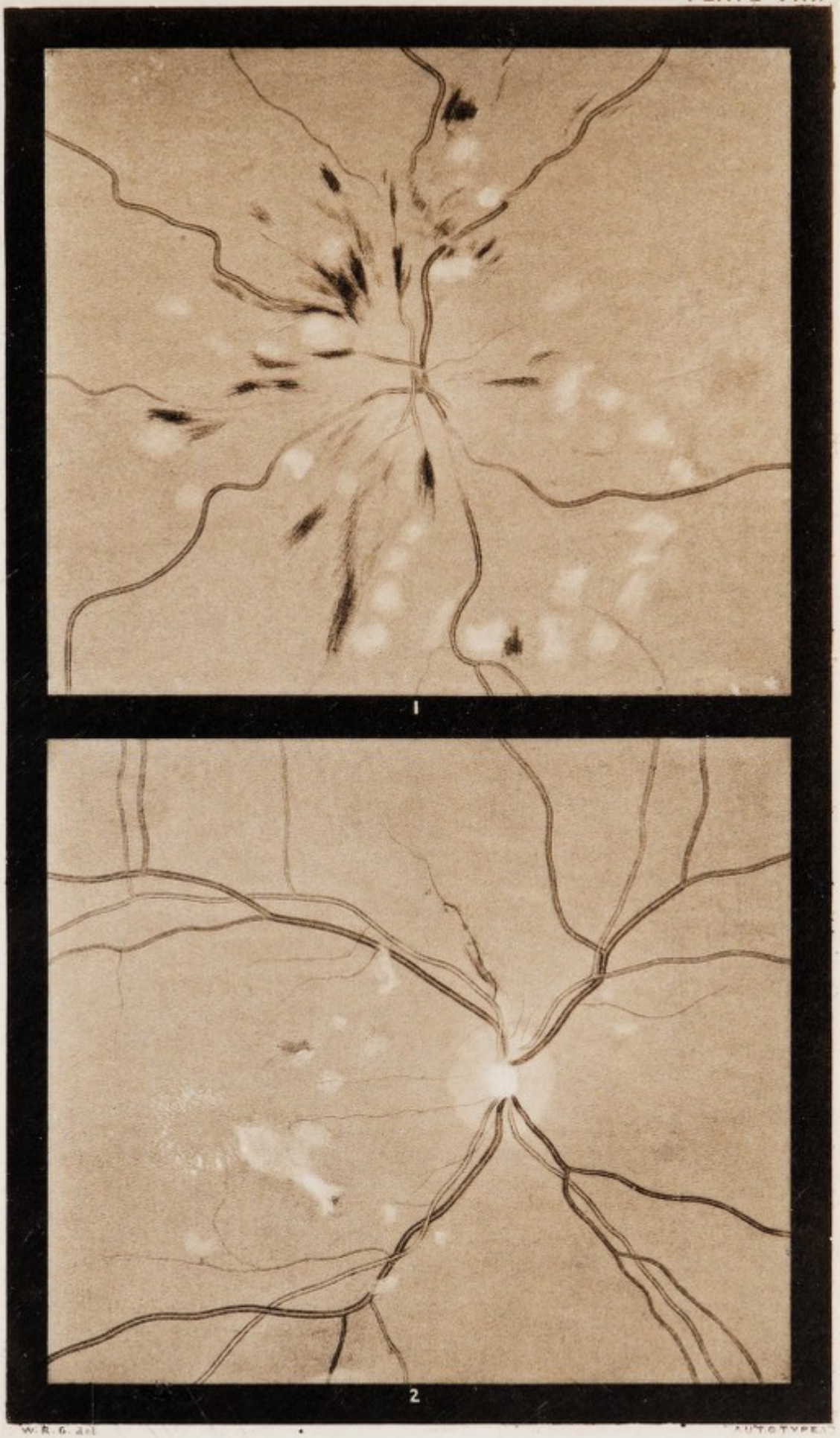
FIG. 1.—*Acute nephritic retinitis, in a patient suffering from chronic renal disease, consecutive to an acute attack twelve years previously. Man aged twenty-one.*

The disc is veiled by a pale opacity, not prominent, which extends on to the adjacent retina. Many soft white areas and striated hæmorrhages are scattered over the posterior segment of the retina. The veins are a little larger than normal. Many of them are much concealed at the edge of the papilla. The arteries are large and conspicuous over the disc, but cannot be traced beyond (probably because they become contracted in size, and are concealed by the retinal opacity). Vision $\frac{1}{12}$. For the microscopical appearances *see* Figs. 71 (p. 207), and 75 (p. 209).

FIG. 2.—*Chronic retinal changes in albuminuria, from a case of acute renal disease passing into the chronic form. Right eye. Woman aged twenty-four.*

The disc and its central cup are normal. The vessels have a normal course. Many irregular white spots lie around the disc, especially between it and the macula, around which is a halo of small spots, for the most part very minute; one or two larger and very white. The other spots are soft-edged; some of them are superficial to the veins. There are a few small hæmorrhages, most of them adjacent to white spots. A small vessel which passes upwards is accompanied by extravasation, as if into its perivascular sheath. Vision: reads No. 12 Jäger.

PLATE VIII.



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AUTOTYPE

PLATE IX.

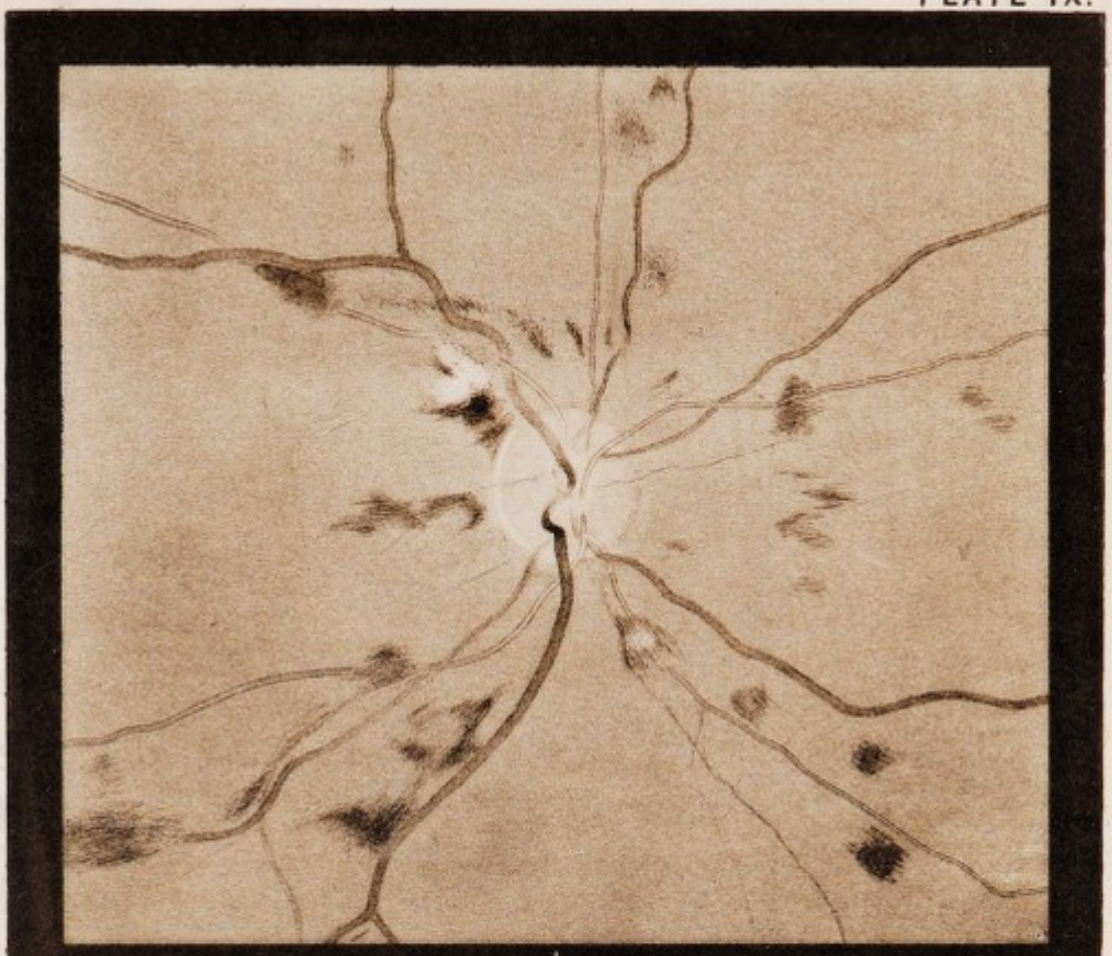
FIG. 1.—*Retinal changes in a case of progressive pernicious anæmia. Right eye. Man aged forty-seven.*

The general tint of the fundus is paler than normal. The disc is clear and the vessels distinct almost to their origin in the centre. The veins are very broad and pale, scarcely darker than the arteries. Their central reflection is broad and indistinct. The arteries are rather narrower than normal, and very narrow in proportion to the veins. A large number of striated hæmorrhages lie around the papilla. Many of these are adjacent to vessels, in front of or beside them, but the course of the vessels is not disturbed. Some white spots are seen, most of which are adjacent to extravasations, one or two being surrounded by a halo of hæmorrhage. One large white spot above the disc has an irregular extravasation below it, but only a few small spots of blood above it. (*See* p. 233)

FIG. 2.—*Retinal changes in leucocythæmia. Right eye. Man aged twenty-seven.*

The optic disc is clear. The course of the vessels is normal. The retinal veins are very broad—at least twice their normal width. Their central reflection is in some veins narrow and indistinct, in others it is broad. The disproportion in size between the arteries and veins is thus very great. The veins are exceedingly pale, scarcely darker than the arteries. An annular zone of hæmorrhage surrounds the macula lutea, broader on the temporal than on the nasal side. On the latter, adjacent to it, the retina presents a grey reflection. Between this and the disc is a striated hæmorrhage, in which are one or two white spots. On the outer side of the annular extravasation is a small, soft, white spot surrounded by a halo of hæmorrhage. The extravasation had caused a corresponding central defect in the field of vision. (Subsequently the veins became still larger and more tortuous.)

PLATE IX.



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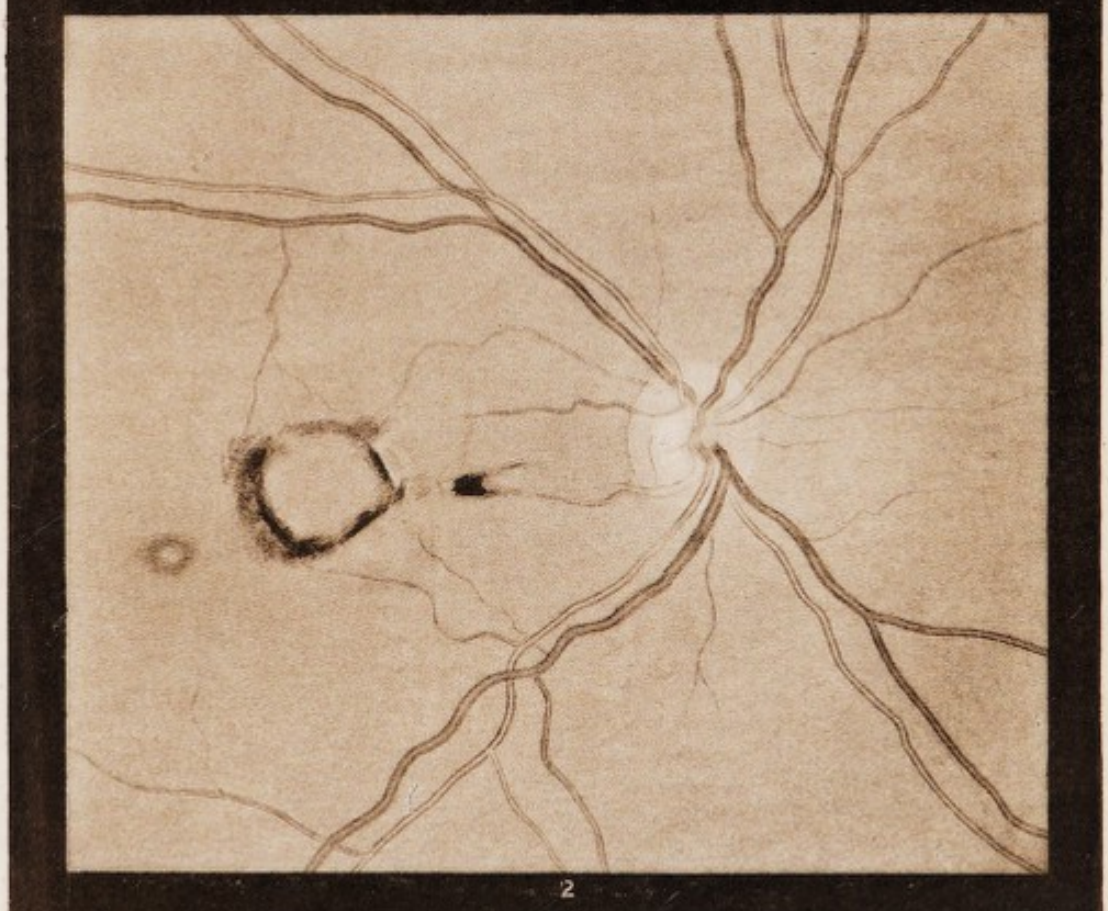

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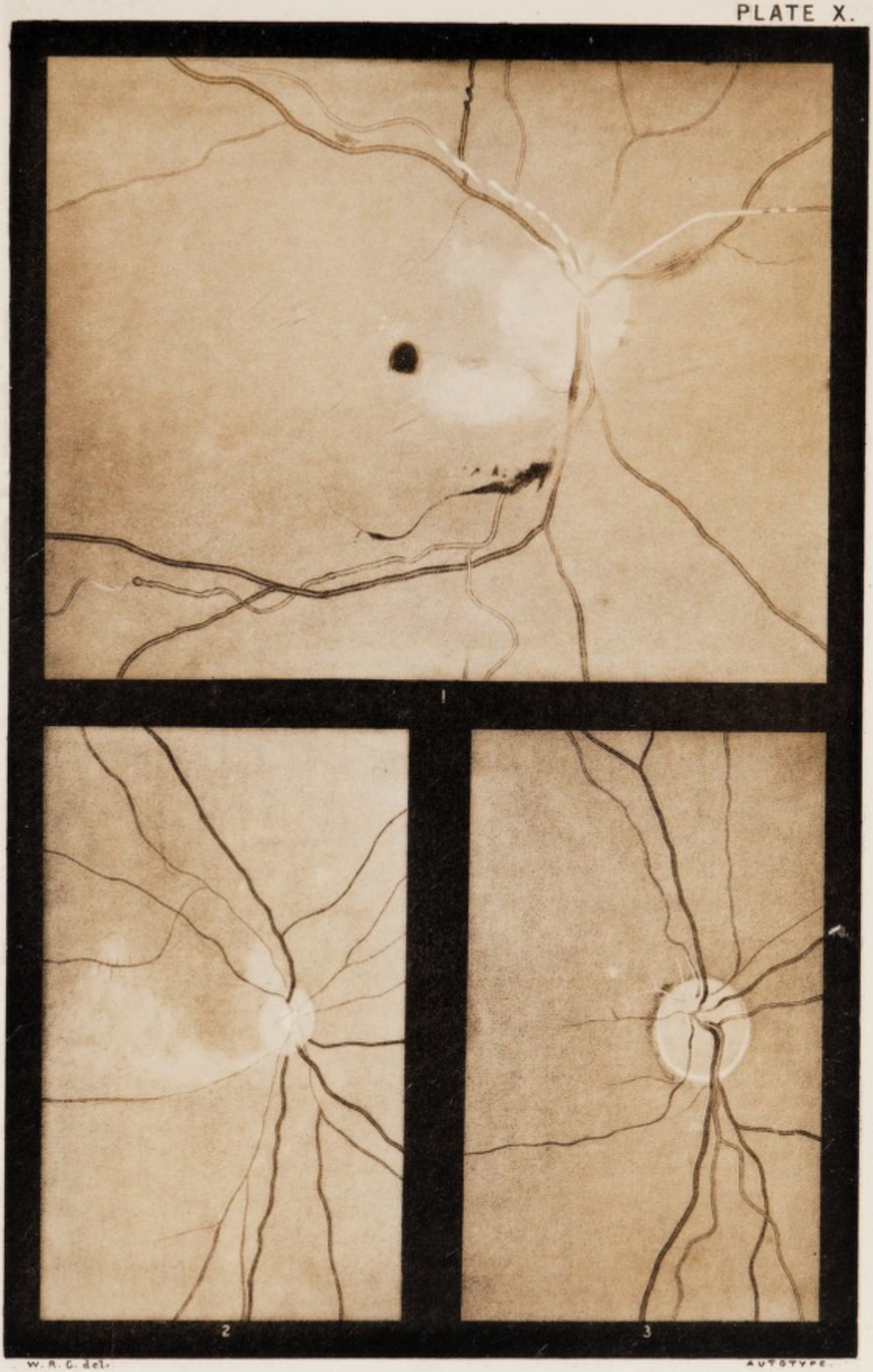
AUTOTYPE.

PLATE X.

FIG. 1.—*Retinal changes (perivascular disease, aneurisms, &c.) in a case of chronic renal disease. Right eye. Woman aged thirty-six.*

The outline of the optic disc can be seen on the nasal (right) side, but is not very distinct. Its temporal portion is concealed by a pale opacity, which extends on the adjacent retina towards the macula lutea. Near the latter are a few minute white spots. Several small extravasations are seen: one, rounded in form, near the macula, and another below, which extends for a long distance along the course of a small vessel, wider at parts than at others, and in one place interrupted. Another extends, as a linear extravasation, along the course of a vein which passes directly downwards. Three arteries which pass upwards present a peculiar appearance, being concealed more or less completely by white bands, corresponding in width to the vessels. One, which passes upwards and to the right (in the drawing), is masked for a considerable distance by such a band, which ceases suddenly, and, before its termination, presents two interruptions. The blood-column beyond this sheath, and in the interruptions, is seen to present perfectly normal characters. Another artery, which passes upwards and to the left, is free at its origin, but just beyond the edge of the disc is concealed by a similar band. It pursues a somewhat wavy course, the lower parts of the curves being indistinct. Like the other, the band ends abruptly, and beyond appears normal. Another artery, which arises in the disc from that last described, has a similar white sheath from its commencement to its disappearance behind a vein. It emerges some distance beyond, free. A vein passing upwards presents peculiar corkscrew-like curves. The vein which passes downwards is invisible for a short distance beyond the extravasation just described, together with its accompanying artery. The arteries are, for the most part, otherwise normal, but one, which passes directly downwards, presents, some distance from the disc, several—at least four—distinct dilatations, evidently minute aneurisms. The central reflection from the vessels broadens out in these dilatations. The last one is globular, and appears at first sight to terminate the vessel, but closer inspection reveals a narrow white band passing from it, which farther on broadens, and gives origin to a branch of an artery of normal appearance. Here and there in the retina are small collections of pigment. Vision: counts fingers only.

PLATE X.



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AUTOTYPE.

FIG. 2.—*Embolism of the central artery of the left retina, occurring simultaneously with an embolism of the middle cerebral artery. Indirect image. Man aged thirty.*

The drawing was made about a fortnight after the occurrence of the embolism. The disc (previously veiled by opacity) is clear and pale (not quite pale enough in the figure), the peripheral part almost, but not quite so clear as the central cup. Its edges are sharp. The veins have a normal size and course. Several of them, however, disappear at the edge of the disc. The arteries are filiform on the disc and for some distance beyond. Some remain, as far as they can be seen, narrow (even to the periphery of the retina); others become wider at a distance from the disc which varies in the case of different branches. From the upper part of the disc a white opacity extends a short distance on to the retina. A similar, but narrower white area extends from the lower part of the disc, being evidently situated behind the level of an artery; it gradually widens and becomes less intense, and is continuous with a mottled opacity which occupies the region of the macula, and is the remnant of a large white area which at first occupied this region. A branch of an artery which courses across the upper part of this area is evidently dilated, and the minute branches which come from it are abnormally distinct. Vision 0.

For the microscopical appearance of the embolus in the central artery, *see* Fig. 4, p. 35.

FIG. 3.—*Partial embolism of the central artery of the retina. Right eye, direct image, much reduced. Woman aged twenty.*

The disc is clear; the central cup and sclerotic ring distinct. The veins are of normal course and character. One division of the central artery, comprising the branches which course downwards and to the right (in the figure), is perfectly normal. The branches of the other division emerge from the upper part of the disc. Of these, two which pass upwards and outwards (to the left) are completely obliterated, visible only for a short distance as white threads. Two others which pass upwards are very narrow, but the central reflection can just be distinguished. One of them is accompanied for a short distance by fine white lines along its sides. Both vessels, some distance from the disc, become wider and resume their normal appearance, as if by anastomoses; a branch of one, which passes to the right, remains filiform throughout. No changes visible in the neighbourhood of the macula. Vision: the field presented a defect corresponding to the area supplied by the obstructed vessels. (*See* Fig. 5, p. 38.)

INDEX.

The Index does not contain references to the Description of the Plates. The subjects of these are enumerated in the Table of Contents.