ON OBSTRUCTION

of the

CENTRAL ARTERY OF THE RETINA

being a

THESIS

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by

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C O N T E N T S .

P R E F A C E.

It is the purport of this Thesis to study the Circulatory Disturbances due to Obstruction of the Central Artery of the Retina, with special reference to the Etiology of this affection.

After a short introduction, I shall deal, firstly, in detail, with the various theories as to its causation; secondly, give a concise report of cases of so-called Embolism of the Central Artery of the Retina, that came under my own observation; and lastly, enter into a critical consideration of the subject.

The four cases, dealt with in the second section, were patients of the Glasgow Eye Infirmary during my term, as House Surgeon, there; hence this gives some idea of the frequency of this affection, as during that time (two years) 27,675 new cases were dealt with.

I am indebted to the Surgeons of the Infirmary for permission to use these cases, and for facilities for prolonged examination to which their admission, as in-patients, contributed. I also consider myself fortunate in seeing these cases so

soon after the onset of the obstruction.

The other cases, used for illustration and comparison, were seen by me either in the Glasgow District Asylums of Woodilee or Gartloch, or in the Royal or Western Infirmaries of Glasgow.

J. Strathearn.

Woodilee Asylum,

Lenzie.

I N T R O D U C T I O N.

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Probably the first recorded case of obstruction of the Central Artery of the Retina was that of E. Jaeger who, in his work on Cataract, reports the case of a man, aged seventy-two, who became blind overnight. The principal interest, to Jaeger in this case, seems to have been the phenomenon of manifest circulation in the blood vessels, with interruptions in the blood stream; but the cause of this does not seem to have been understood by him.

Von Graefe, however, in 1859, published a case of sudden blindness and made; for the first time, the ophthalmoscopic diagnosis of "Embolism of the Central Artery of the Retina." The case is fairly typical although the ophthalmoscopic examination was not made until eleven days after the onset. He found the media clear, the veins small but fuller towards the periphery, the disc pale and the arteries much reduced. The man was also found to be suffering from aortic obstruction and endocarditis, so an exclusive diagnosis of em-

bolism was made. On subsequent examination he noted an irregularity in the filling of the veins, the breaking up of the blood coulmn into cylinders, the central region of the fundus cloudy and a cherry-red spot at the macula.

In the next few years we have records of similar cases by Schneller, Blessig, and Liebrich.

Soon, however, observers began to question the adequacy of the original explanation and theories other than embolism of the central artery were advanced.

Magnus brought forward the theory of Haemorrhage into the Optic Nerve Sheath; Steffan sought to show that the embolism was not of the Central Artery but of the Ophthalmic Artery. The theory of Primary Arterial Thrombosis found many supporters; and lastly, some have sought in arterial spasm, and others in arterial degeneration and disease, a cause for this obstruction.

S E C T I O N I.

EMBOLISM.

This is, at once, the oldest and most widely accepted theory and it must be admitted that in many cases it agrees with the clinical facts. Further, in the cases of Von Graefe, Schwigger, and Sichel, an embolus in the lamina cribrosa was demonstrated as the cause of obstruction. A similar cause was found in the more recent case of Priestley Smith, the examination being made four months after the occurrence. Gowers also reports a case, examined eight weeks after the onset, where a long granular embolus was found. Kern has collected from the literature 16 cases in which a pathological study has been made and in only three instances no embolus could be discovered. In these three cases the lumen of the vessel was quite free. The suddenness of the onset; the absence of previous warning, the ophthalmoscopic picture of arterial obstruction at once suggest embolism and the finding of a source, sufficient for the production of an embolus, seems to clinch the diagnosis.

On more critical examination of the facts, is embolus an efficient explanation? How does it

happen that soon after the total blockage of the artery we notice distinct evidence of circulation and, it may be, (even a moderate degree) fullness of the vessels. To account for this it is assumed (Schnabel and Sachs) that the embolus is not sufficient to entirely block the vessel and hence some blood may pass. Elschnig further elaborates this by supposing that at first the irritation of the partial embolus causes it to be tightly gripped by the vessel wall, and, as the spasm relaxes, a certain amount of blood begins to pass. Though plausible, this theory is in many ways untenable and as Haab states: "the idea of partial embolism is a very questionable condition and one that it is difficult to understand."

On the other hand, in many cases, an adequate cause for the origin of the embolus could not be found. In Kern's work on the subject, out of a total of 54,800 cases at the clinic at Zurich, there were twelve cases of obstruction of the central artery and in only two of these cases was an undoubted source for the embolus found: that is, in 83% of the cases no positive source of the embolus was detected. Further, out of 83 cases of obstruction of the artery, collected by him from the literature on the subject where a report

of the general condition was given, he found that in 66% of the cases no positive source for the embolus could be determined.

Again, is the existence or the finding of a heart murmur sufficient proof of an origin for an embolus? An objection, urged by Reimar to the theory of embolus, is the lack of ophthalmoscopic evidence of such an obstruction. This evidence, however, cannot, in the nature of things, be expected unless it is situated at the disc or is a branch embolism: with regard to this latter some authors have described a white, yellowish-white or gray-white plug in a branch artery, which was obstructed, and called it an embolus: but a study of degenerate vessels (retinal) in arterio-sclerosis, Bright's disease etc., where localised thickening of the intimal and other coats is common, raises a strong suspicion that their descriptions of appearances do not warrant the name 'embolus,' and, to say the least of it, their evidence is inconclusive. Of greater importance is the fact that several authors, as Reimar and Haab, maintain that in all the pathological studies of cases recorded, "in not a single instance, not even in which it was very likely to be an embolus, has the proof of embolic nature been

at all convincing." (Haab).

When we reflect, also, that the shortest interval between the period of obstruction and the time of enucleation was six weeks, and take into account the great changes that may take place within the vessels in this time, especially as enucleation was done, and can only be justifiably done, for some intercurrent affection, such as glaucoma, we must admit that it is no simple matter to state decisively the nature of the obstruction. Experimental work on this subject is of little value for, no one seeks to deny the possibility of embolism or that it could not give a very similar train of ophthalmoscopic appearances.

Hence Embolism of the Central Artery of the Retina is, in many cases, a probable diagnosis but, as a hypothesis, it is frequently so inadequate and un-needed that we are bound to consider the possibility of other causes for this obstruction.

EMBOLISM OF THE OPHTHALMIC ARTERY.

The theory that obstruction of the retinal arteries was dependent, at least, in some cases on embolism of the ophthalmic artery was brought forward by Steffan in 1866.

There has been no anatomical evidence even to suggest this and in not one of the sixteen cases, where, as previously mentioned, pathological examination was made, was this artery occluded. Indeed, when we consider the great anastomotic communications of the ophthalmic arteries with the branches of the external carotid, even temporary cessation of the retinal circulation, due to embolism of the ophthalmic artery, seems improbable. This hypothesis is then of merely historical interest.

HAEMORRHAGE INTO THE OPTIC NERVE SHEATH.

In 1878, Magnus published a case of sudden blindness with subsequent restoration of partial vision in the nasal half of the retina. To explain this case he brought forward the theory that haemorrhage had occurred into the optic

nerve sheath: the pressure of this abolished the power of nerve conduction and, to a certain extent, obliterated the artery: later, absorption took place and those parts of the nerve, least injured, regained their function to some extent. This theory, like Steffan's, has no anatomical evidence to support it.

To be sure, haemorrhage into the sheath has been noted to have occurred, e.g., in rupture of an aneurysm of the middle cerebral artery (MacKenzie), in rupture of an intra-cerebral extravasation into the meninges (Michel), and in Haemorrhagic Pachymeningitis (Manz). But in these cases I can find no record of an ophthalmoscopic examination having been made or history suggesting visual disturbance.

This lesion has been assumed to be present after injuries, such as blows on the head or falls. For instance, cases have been described where blindness came on a day or two after a fall and then gradual return of vision took place, this being accounted for by haemorrhage into the optic nerve sheath. Whereas, in those cases which lead to optic nerve atrophy, damage to the nerve by fracture of the orbital bones was more probable.

Further, some cases of sudden blindness, in girls, suffering from amenorrhoea, have been described in which this diagnosis seemed to be a very likely one. Knapp describes a case of blindness that came on during the progress of whooping cough, where he found the disc white, the retinal arteries, invisible in one eye, and mere streaks in the other. He suggests, as one explanation, haemorrhage into the optic nerve sheath. Landesberg describes a somewhat similar case of partial embolism probably due to this cause. From the foregoing it will be seen that this diagnosis can only apply to a limited number of cases, but it should be considered where we have a history of injury, of amenorrhoea, of vicarious menstruation, of epistaxis or other form of haemorrhage.

Clinically, in addition to the other, and ordinary, symptoms of obstruction, slight imperfection in the execution of lateral movements of the eyeball, with some degree of pain, would to my mind, be suggestive.

ARTERIAL DEGENERATION AND DISEASE AS A CAUSE OF OBSTRUCTION.

Great advances have been made in recent years with regard to our knowledge of arterial disease, especially in relation to the smaller cerebral arteries. Unfortunately, a similar exhaustive, and systematic, research (clinical and pathological) has yet to be carried out with regard to the ocular vessels. True, much good work has been done by Thoma, Raehlmann, Hirschberg, Reimar, and others but there exist many gaps in our knowledge and we have frequently to assume analogous conditions which may, or may not, be fully warranted. In Bright's disease, more especially in the chronic granular kidney, there is undoubted clinical and pathological evidence of arterial retinal change. Microscopically, Brailey and Edmunds have described this "as commonly a development of hyaline tissue, causing uniform thickening of the vessel wall - the change apparently occupying the middle coat; but sometimes said to start in the intima." This thickening necessarily diminishes the calibre of the vessel and, if great, may lead to its occlusion.

There is an example of this from a section by Buzzard in Gower's Medical Ophthalmoscopy. Reimar, also, gives histological demonstration of this from a case of sudden blindness where, in addition to a general narrowing of the calibre of the ocular arteries, through endarteritis, was seen at some places a complete obliteration of the lumen by a localised intimal swelling. The kidneys showed chronic interstitial nephritis.

In senile angio-sclerosis somewhat similar appearances, and results, have been noted: but instances of the purely senile type are becoming rarer as our knowledge increases concerning the albuminuric, and the syphilitic, varieties. Syphilis may produce changes that are very similar to those seen in senile and albuminuric sclerosis. The syphilitic variety, which manifests itself as an endarteritis, or even periarteritis, may arise in the early stage of the secondary period or at any subsequent time. The smaller arteries of the brain in marked syphilitic arteritis may have their walls greatly thickened leading even to occlusion, (Obliterative arteritis). The vessels are frequently the seat of thrombosis.

There is also a group of cases where,

for some unknown cause and apparently unassociated with any particular diathesis or disease, proliferation of the intima occurs. Physiologically, we see this process in the obliteration of the unbilical vessels and the ductus Botalli: and Thoma maintains that this process always occurs where there is a disproportion between the lumen of a vessel and the amount of blood passing through it.

The pathological variety has been described under various names (endarteritis obliterans, endarteritis proliferans etc.): it is said to occur in adults between the ages of 30 and 60 and to be more frequent in men than in women. Ofcourse, it may be, as Reimar suggests, merely one of the manifestations of a general arteriosclerosis having a selective action on the smaller arteries. The retinal arteries, from their position and the frequent movements to which they are subjected, are peculiarly liable to arterial disease.

We may take it for granted that a proliferating process may, and in certain conditions does, attack the retinal arteries: and we may surmise that this local intimal swelling may of

itself so reduce the calibre of a vessel that any diminution of blood pressure, however arising, may permit of the approximation of the vessel walls and a stoppage in the current: if of short duration, causing only a transient obscuration; but if more sustained, a permanent blindness: the functioning of the retinal cells, within certain limits, being more dependent on the continuity than on the quantity of the blood flow.

PRIMARY THROMBOSIS.

To explain the cases where we have premonitory symptoms many authorities have attributed the obstruction to Primary Arterial Thrombosis. Such cases are those, to quote Priestley Smith, where we have a history of previous attacks of transient blindness in the blind eye: a simultaneous attack of blindness in the fellow eye: previous, or subsequent, attacks of transient blindness in the fellow eye, especially if the conditions of onset were the same, in the permanent, as in the transient attack: and lastly, signs of disturbance of the cerebral circulation at the onset of blindness, e.g., giddiness, faintness,

headache. This is the teaching of most of our standard text-books on ophthalmology.

We must, then, discuss firstly the conditions in which arterial thrombi are formed. So far as our present knowledge goes, three classes of causes are assigned for thrombosis:-

(1) Alterations in the blood.

(2) Slowing and other irregularities of the current.

(3) Contact of the blood with abnormal surfaces.

Alteration of the blood is by itself a possible cause of thrombosis, admittedly rare. It is, in addition, only reasonable to expect that this thrombotic disposition would be general and hence take place primarily in the veins and, further, it would lead to such vascular disturbance that the question of its presence in the central artery of the retina be quite overshadowed by its malign effects elsewhere.

(2) Slowing or Irregularity in the Blood Current.- Mere slowing of the circulation does not suffice to form thrombi, for, as has been demonstrated, even a stationary column of blood in an artery or vein may remain fluid for weeks.

Similarly, the objection noted above also applies here, that a slowing of the current, dependent on a general cause, would more likely, in the first place, affect the blood in the veins; and if a local one then the central vein and not the central artery would be first affected; and as the actuality of a primary thrombosis of the central vein is very doubtful much more than is the occurrence of a primary thrombosis of the central artery.

(3) Contact of the Blood with Abnormal Surfaces.- The integrity of the endothelium is recognised as of the greatest importance in maintaining the fluid state of the blood. Probably the commonest factor in impairment of the arterial wall is the presence of atheroma, especially in its later stages and, unquestionably, is a frequent cause in inducing coagulation. On the other hand, arterio-sclerosis, when unaccompanied by atheromatous or calcareous changes, is a very uncommon cause of thrombosis; and when it does occur, is probably more directly the result of circumstances, such as excessive narrowing of the calibre of the vessel leading to diminution in the velocity of the blood stream etc. However, as yet, we have

no evidence of the existence of atheromatous changes in arteries of such small size as the central artery of the retina; and a narrowing, by arterio-sclerosis etc., of such marked degree as to diminish the blood velocity and cause thrombosis; might of itself induce stoppage of the current (vide section arterial changes). Hence the thrombosis would not be the primary factor but a secondary effect ensuing after the retinal obstruction had occurred. Therefore, we must conclude that Primary Thrombosis of the central artery of the retina is a possible, but not a very probable, cause; and further the symptoms, said to point to its existence, are more indicative of obstruction from disease of the arterial wall or from arterial spasm, as will be shown later; the thrombosis, if it occurs, being quite secondary and ensuing after the onset of the obstruction.

OBSTRUCTION BY SPASM OF THE RETINAL ARTERIES.

Since the publication, in 1862, of Raynaud's Thesis on local asphyxial and symmetrical gangrene of the extremities, the condition of spasmodic and recurrent contraction of the

arterioles has been recognised. Among his later contributions on this subject was the observation that "occasionally, temporary alterations in the fundus oculi alternate or coincide with manifestations of local asphyxia in the extremities."

His first case was that of a man, aged 59, who, after suffering from attacks of local asphyxia of the fingers of one hand, then of the other and lastly of the feet, presented symptoms of visual disturbance: the eyesight, previously good in both, became dimmed, more especially in the left. Ophthalmoscopic examination showed that "the central artery of the retina and its branches had very clear contours, and that they were definitely narrower round the papilla than at the periphery; here and there was a sort of partial constriction, the papilla was very clear; the veins were the seat of remarkable pulsations a little later than the radial pulse." ..... "The central vein dilated and elongated itself so notably in the region of the papilla as to simulate a small aneurysm, but the pulsation was also visible in the smaller veins." In the right the phenomena were similar but less marked. These examinations were verified by Galezowski.

His second case was that of a young man, aged 22, and suffering from diabetes insipidus. He had local asphyxia of the upper extremities, and some blueness of the face; during the attacks the radial pulse became very small, and at "the commencement of the cyanosis the patient complained of a notable obscuration of sight, which disappeared when the face and hands returned to their natural colour." Panas observed that at the beginning of the cyanotic attack the "arteries of the fundus oculi were definitely narrowed," and that "when reaction occurred they became widened." "The retinal veins were turgid, but presented no appreciable pulsation."

In these cases Raynaud assumed that this contraction of the vessels was a reflex act, being the result of a sensory excitation of the afferent nerves, an efferent impulse from the vaso-motor centre determining the contraction.

So far as I am aware the anatomical demonstration of vaso-motor nerves to the ocular vessels has not yet been made, and the investigation of the human eye affords no proof that they are directly under the control of specific vaso-motor nerves; but their existence is rendered

very probable by numerous observations, both ophthalmoscopic and experimental, upon men and animals.

In 1848, Reid isolated the sympathetic from the vagus fibres in the cat, and showed that only the sympathetic caused dilatation of the pupil on stimulating the peripheral end. Kuyper, in 1859, proved that in addition to dilatation of the pupil, contraction of the arteries followed upon stimulation of the cervical sympathetic. The later observations of Parsons' (1905) where on stimulation of the sympathetic in the dog, high up in the neck, or stimulation of the superior cervical ganglion itself, was followed, after a latent period, by a well-marked fall of intra-ocular tension, due to constriction of the intra-ocular arterioles: this latent interval which occurs between the dilatation of the iris and the fall of tension, strongly confirming the opinion that the fall is due to vaso-constriction; since, in the case of the iris it has been shown, by Langley and Anderson in their experiments, that the constriction of the arterioles follows the dilatation of the pupil after a distinct interval.

Leber, on rabbits, and Schaler, on cats,

have both observed the experimental contraction of the retinal vessels, following upon irritation of the cervical sympathetic.

When we come to seek for clinical proof of vessel-spasm, we are met with divergent opinions. Haab states that positive proof of vessel-spasm, in either normal or diseased retinal vessels, is largely wanting. Leber, and Wagenmann comment on the feasibility of the theory of vessel-cramp and the latter, recognising its importance, suggests further study of the subject. Hughlings Jackson, in his work on the Brain, found that sleep is accompanied by an anaemia of the brain, due to arterial constriction, and also that this constriction could be observed at these times in the retinal arteries. This was confirmed by Mosso, Durham and Kennedy, and Leonard Hill; the latter also maintaining that "we have every reason to suppose that the retinal circulation is an accurate index of the cerebral circulation and vice versa."

The correlation of arterial retinal constriction with the fall of blood pressure has been noted in anaesthesia and narcosis. I have seen this narrowing of the retinal arteries very

marked in a case of morphia poisoning; the fundus being extremely pale, the disc white, and the arteries mere streaks, whilst the veins were rather full.

A study of the visual disturbances sometimes noted after severe or repeated haemorrhage is also instructive, although many points in this dangerous, but fortunately rare, disease, are still unexplained. Why the loss of a similar, and not necessarily excessive, quantity of blood in one individual should produce grave visual disturbance and in another not, and why sometimes both eyes and in other cases only one eye should be affected is still unknown. Out of one hundred and sixty cases, collected by Fries from the literature of two-hundred and fifty years, "in eighty-nine and a half per cent. the visual disturbance was bilateral and in sixty-one per cent. both eyes became blind."

Many theories have been advanced to explain it. Von Graefe suggested retro-ocular haemorrhage as a cause: Samelsohn assumed the propulsion of arachnoidal fluid into the intra-vaginal space of the optic nerve by an existing cerebral oedema: others have attributed the

symptoms to inflammation of the optic nerves, to a disturbance of the circulation in the optic nerve and retina, and to rapid degeneration of the retinal vessels induced by the acute anaemia. Gowers states "that one effect of the loss of blood may be upon the retinal elements themselves." Haab is of the opinion that all these theories have been forced into the background by the anatomical researches of Ziegler. The latter resorts to the supposition of the local contraction of the blood vessels to explain the facts, maintaining that "a certain disposition, which has its origin in a sensitive vaso-motor system, is required for its production."

The following is a case of this form of blindness recorded by Lawford. A woman, aged forty-eight, had severe haematemesis, and became collapsed. There was no recurrence of the haemorrhage. The eyesight failed, and after partial recovery sank to complete blindness, which was permanent. Ophthalmoscopic examination revealed, in each eye, a very pale, hazy, slightly oedematous papilla, surrounded by cloudy retina by which the veins, somewhat enlarged and turgid, were partially obscured. The arteries were

narrowed; no haemorrhages were seen.

In the Western Infirmary, I observed a very similar case in a woman, aged forty-five, suffering from carcinoma of the stomach. After profuse haematemesis, she became very collapsed; several hours afterwards she was still but semi-conscious: the pupils were moderately dilated and did not react to light: Tension -1: she did not seem to have perception of light but this could not be accurately determined. Ophthalmoscopically the discs were pale, the retinae diffusely hazy, the arteries contracted, and the veins dilated and tortuous: slight pressure on the globe produced manifest arterial pulsation and the current could be readily stopped by increasing the pressure. The appearances, in fact, were similar to those we so frequently see in cases of so-called embolism.

An explanation of this is suggested to me by a recent contribution by Mr. Malcolm on "The Condition of the Blood Vessels during Shock." (Lancet Aug., 1905.) He traverses many of the usually accepted views on this subject and maintains that "a contraction of the arteries generally, and especially of the superficial arteries,

occurs during shock" and "there is no complication more likely to precipitate a condition of shock than the loss of a considerable quantity of blood." He elaborates this theory of arterial contraction with great minuteness and conclusiveness.

If this view be accepted many of the clinical facts of shock are easily understood and, further, in my opinion, these instances of blindness following haemorrhage resolve themselves (somewhat after the assumption of Ziegler) into cases of obstruction of the central artery of the retina from spasm of the vessel wall.

In the consideration of the Toxic Amblyopias we find that the belief in the existence of arterial retinal contraction is as widespread as it is authoritative.

In lead-poisoning the amaurosis has been ascribed to a spastic ischaemia of the retina (Elschnig). Tobacco Amblyopia, according to Parsons, is the result of two chief factors: (1) a toxic effect on the nerve cells: (2) a vascular effect, causing vaso-constriction of the retinal arterioles.

Again, in Quinine Amblyopia and those of the coal-tar derivitatives, there can be little

doubt that the vascular condition is the essential factor. In the severe Quinine cases we have blindness, complete or incomplete, usually developed with great suddenness; dilatation of the pupil, marked constriction in the size of the retinal arteries (with occasional obliteration of their peripheral portions). Cases have even been described (Gruening, Buller, Browne, etc.) with "the retinal haze and the cherry-red spot, closely simulating cases of embolism of the central artery of the retina."

The experimental researches of Barabaschew, Brunner, and de Schweinitz, show that "there is, primarily, an ischaemia of the retinal and optic nerve vessels caused by their intense contraction," the former believing that "this is brought about by a condition of irritability of the vaso-motor centres, caused by the Quinine."

De Bono and Ward Holden admit this vaso-constriction but place great stress on the early nerve changes of which this permits.

In the ophthalmoscopic examination of the fundus during the Algid period of Cholera, contraction of the retinal arteries has been observed frequently. Von Graefe has even seen

cases "exhibiting all the symptoms that may be observed in embolism of the central artery of the retina."

The occurrence of arterial retinal spasm in Migraine has been doubted by some, but many of the reported cases hardly admit of this doubt. In this connection Gowers states: "of great importance 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 the attacks, 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 are those of occlusion of the central artery." (vide also, Galezowski - Rec. d'Oph., Jan., 1882. Doyne - Trans. Ophth. Soc., Vol. IX, p. 148).

Clinically it has been noted in Migraine that the arteries on the affected side are often the seat of arterio-sclerosis; this has been confirmed anatomically by Thoma. Assuming then, that this affection is, in the first stage, a vaso-constriction due to a vaso-motor neurosis (Moilendorf, Latham, etc.) "the presence of arterio-sclerosis is a point of interest bearing

on this view." (Osler). Later on I shall have occasion to refer to this association.

Migraine has been considered by many to be the sensory equivalent of a true epileptic attack. Hence in epilepsy, the question of arterial spasm has been frequently discussed. There is much divergence of opinion on this matter. Gowers states that "no change can be seen in the retinal vessels, even during an attack, at least of a minor nature." Hughlings Jackson, on the other hand, describes a case where the retinal arteries suddenly disappeared whilst the fundus was being examined at the time of the seizure. Swanzy admits its occurrence, and so does Suckling. Doyen reports a case where, on trephining the skull for epilepsy, the patient had a fit during the operation, and it was observed that at its onset the brain became suddenly anaemic.

The difficulties in the way of getting clinical proof of arterial retinal spasm in epilepsy are, necessarily, very great. During the past eighteen months, in the Glasgow District Asylums, I have had many epileptics under my care. One epileptic, a lad of nineteen, usually

complained of loss of sight immediately before the onset of a seizure; during a series of fits I was able to make an ophthalmoscopic examination and I have no hesitation in saying that the fundus was pale, the arteries distinctly narrowed, and the veins full; thus accounting for the blindness, apart from the probability of a psychical factor. In other cases, again, although there was no apparent abnormality in the fundus or in the size of the vessels yet arterial pulsation, and in one case actual stoppage of the current, was obtained by comparatively slight pressure; and this fact, in the absence of any demonstrable increase in intra-ocular tension or general fall in blood pressure, points to some temporary interference with the blood current, most likely vaso-constriction.

In Chorea there is an opportunity for the investigation of arterial spasm. Swanzy has reported a case (Royal London Ophth. Hospital Reports, Vol. VIII, p. 181) where "embolism of the central retinal artery of the left eye, and chorea, chiefly of the left side of the body, came on simultaneously." It is interesting to note that no cause for the supposed embolus was

found, the heart being normal; no history of rheumatism, etc. Benson reports a case (The Ophth. Rev., 1886) where three attacks of chorea preceded "retinal embolism." The heart was normal and no source for the 'embolus' was found.

The interest of these cases lies in the fact that the embolic theory of chorea has been largely discarded and, as Gowers says, is now merely historical. Further it has been affirmed that, at least, some forms of chorea may be due to prolonged arterial spasm. If this be so, these cases of arterial retinal obstruction, occurring on the same side as the chorea, are instructive, more especially as no source for an embolus was detected to warrant the diagnosis.

There are several cases reported in which retinal ischaemia occurred as a more or less independent affection; the fundal arteries were found extremely thin, almost empty, and barely visible, the veins being broad and dark, and the papilla pallid. (Graefe, Rothmann, Knapp and others). In these cases there is no mention of any condition which might have given rise to reflex arterial spasm, but in the interesting case recorded by Priestley Smith, we have very direct

evidence of such a condition. Here in connection with the use of a vaginal syringe and the touching of a particular spot, sudden, but temporary, blindness repeatedly occurred, till, on one occasion the eye remained permanently blind. The appearance of the fundus was "indistinguishable from embolism." Wagenmann, also, reports a case where a patient suffered from repeated attacks of blindness in one eye. Ophthalmoscopically all the appearances of recent 'embolism' were present and yet after a lapse of ten minutes the circulation began to return, the vision, eventually, being quite restored. Some months later one such attack led to permanent blindness, with the ophthalmoscopic picture of 'embolism.' Benson reports a very similar case.

This retinal spasm may be either reflex or central. An illustration, probably of the latter class, came under my own observation. The patient was a man, aged twenty-five years, of neurotic temperament and a neuropathic ancestry. There was a clear history of acquired syphilis. His mental condition was that of mild, but progressive, dementia; probably

secondary to syphilitic arteritis; which was well marked. At infrequent intervals, he was subject to epileptiform convulsions, and at these periods he suffered from vaso-motor disturbances, such as marked pallor of the face, followed by flushing - usually limited to the head and neck - but sometimes more general. On several occasions he complained of loss of sight. At one examination I found both pupils moderately dilated, the left being larger than the right: both pupils were fixed: tension seemed normal: he had dim perception of form with the right eye and mere perception of light and shade with the left. On ophthalmoscopic examination both fundi were much paler than usual and slightly hazy: the arteries were considerably reduced and the blood current only detected as a slight rhythmical reddening on pressure on the eyeball: the veins were dark, fuller towards the periphery, and somewhat tortuous. This condition remained off and on for several hours and then passed away, the pupils becoming normal and the vision gradually returning. Apart from these manifestations I could never detect anything in the fundi that might be called abnormal. In a few days time his vision was V. A. R. = $\frac{20}{30}$.

V. A. L. = $\frac{20}{40}$, (with slight degree of uncorrected hypermetropic astigmatism). In the consideration of this case one must not lose sight of the fact that the man was suffering from a gross brain lesion: the vascular disturbances being due, probably, to some temporary interference with the vaso-motor centre: his blindness, in my opinion, was not central or psychical, but the result of the spastic interference with the circulation of the retina.

My reason for dealing at such length with the subject of arterial retinal spasm is due, in part, to my estimate of the importance of the subject and also to endeavour to place its actuality on a firmer basis: and when we consider the evidence here adduced, surely, we may claim, with due deference to Haab, that there is positive proof, experimental and clinical, of vessel spasm both in normal and diseased retinal vessels.

REFERENCES.

Anderson. Journal of Physiology, XIII., 1892.

Benson. The Ophthalmic Review, 1886.

Report of International Ophthal. Congress, Edinb., 1894.

Blessig. Archiv. fur Ophthalmologie, Bd. VIII, I, S. 216.

Brailey. Trans. Oph. Soc., Vol. I, p. 44.

Doyne. " " " Vol. IX, p. 148.

Edmunds. " " " Vol. I, p. 44.

Elschnig. Archiv. fur Ophthal., Vol. XXIV, 1892.

Galezowski. Rec. d'Oph. Jan., 1882.

Gowers. "Medical Ophthalmoscopy," 1904.

"Diseases of the Nervous System."

vom Graefe. Archiv. fur Ophthalmologie, Bd. V. S. 1859.

Haab. Norris & Oliver's "System of Diseases of the Eye."

"Atlas of Ophthalmoscopy."

Hill. "The Cerebral Circulation."

Jaeger. "Ueber Staar und Staar-Operation."

Kern. "Zur Embolie der Arterien Retinae."

Knapp. Archiv. fur Ophthal., Vol. IV, p. 448.

Kuyper. "Accommodation and Refraction of the Eye." (Donders).

REFERENCES Contd.

Langley. Journal of Physiology, XIII, 1892.

Lawford. Norris & Oliver's "System of Diseases of the Eye."

Landesberg. Med. Surg. Rep., Sept. 8, 1880.

Liebrich. Berliner medicinische Gesellschaft.

Magnus. Klinische Monatschrift fur Augenheilkunde, 1878.

Manz. Helmholtz's Festschrift, 1891.

Mosso. Centrabl. f. d. Med. Wissensch.

Osler. "Principles and Practice of Medicine."

Parsons. "The Neurology of Vision," 1904.
"The Ocular Circulation," 1905.

Reimar. Archiv. fur Augenheilkunde, Vol. XXXVIII, 1899.

Raehlmann. Fortschritte der Medicin, 1889.

Reid. Physiological, Anatomical and Pathological Researches, Edin., 1848.

Sachs. Archiv. fur Augenheilkunde, Bd. XV.

Schnabel. " " " " XV.

Schneller. Archiv. fur Ophthalmologie, Bd. VIII, I, S. 216.

Salkowski. Zeitschrift fur rat. Med. XXIX, 1867.

de Schweinitz. "The Toxic Amblyopias."

REFERENCES Contd.

Priestley Smith.	The Ophthalmic Review, 1884.
Suckling.	"On Epilepsy."
	"On Periodical Headache or Migraine."
Swanzy.	Royal London Oph. Hosp. Reports, Vol. VIII, p. 181.
Thoma.	"Pathological Anatomy."
Wagenmann.	Archiv. fur Ophthalmologie, 1892.
Ward Holden.	Trans. Amer. Ophth. Soc., 1898.

SECTION II.

CASE I.

C.G., aet. 66, housewife, widow.

Complained of loss of sight in the Left eye of half an hour's duration.

Patient was visiting the Glasgow Exhibition and on emerging from a comparatively dim interior, she felt dazzled with the glare of the sunlight on the white walls of the buildings. Recovering herself in a moment or two, she found that she had lost the sight of the left eye. She came to the Infirmary and was admitted.

On examination the pupils were unequal, the left being the larger: the left pupil, also, seemed fixed: tension in both normal, though somewhat soft. Visual acuity in left eye = No perception of light: in right eye = $\frac{20}{40}$.

Ophthalmoscopic examination of the left eye.-

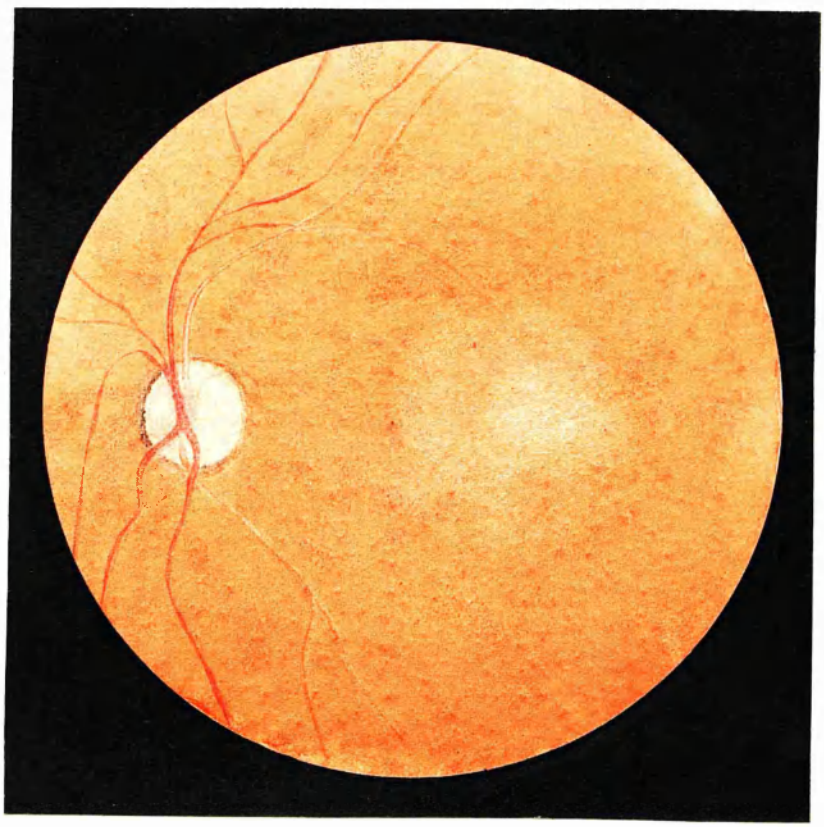
The fundus showed a high degree of retinal anaemia: the arteries were filiform and only the main branches could be traced: at places they showed a white border. The veins were narrowed but not to any great extent: this narrowing was

most marked upon the optic disc and they became relatively broader as they passed toward the ora serrata. The optic papilla was of a pale yellowish-white tint. The macular region was whitish and the whole fundus very clearly seen.

In a few minutes the inferior temporal artery began to fill with a fine, broken column of blood: these little cylinders oscillated and passed slowly onwards in a pulsatory fashion: it was also noted that the blood column in the corresponding vein had become broken and, with a to-and-fro movement, was slowly emptying - the vein becoming narrowed: slight pulsation seen. After an interval of three or four minutes a little column of blood was seen to be entering the superior artery, oscillating as it came, and at times almost disappearing in the reverse direction, it reached higher and higher up the vessel: the onward movement was distinctly pulsatory. The neighbouring vein behaved similarly to the inferior one. Very slight pressure on the eyeball emptied the arteries: the portions of the veins upon the disc could also be emptied in this way; whilst their more peripheral branches became dark and engorged. Immediately on releasing pressure the

Fig. I.

Case I. C.G.- The fundus of the left eye on admission: the arteries are narrowed and only the main branches visible: they appear empty and at places show a white border: the veins are also narrowed and uneven: whitish appearance in the macular region.



blood cylinders quickly ran into the arteries but in a moment they slowed and then behaved as previously mentioned. Prolonged massage had an equally transient result.

During these observations it was noted that there was a rhythmical alteration in the arterial retinal pulse: periodically the movements of the blood cylinders became quicker and the pulsation stronger, followed by a gradual slowing and weakening: this cycle was present in the veins in a lesser degree. (For further observation and commentary see Section III.)

About ten minutes after the case came under observation the fundus was seen to be getting hazy, and the outline of the disc blurred: by this time the circulation had improved considerably and the blood current - still broken into cylinders- was running much more rapidly and with distinct pulsation. As the oedema spread over the fundus the central area of the macular region, diffusely red and about the size of the disc, showed up by contrast - its margins fading imperceptibly into the surrounding haze. Arteries and veins became indistinguishable and, eventually, almost hidden. No trace of haemorrhage had been noted.

Arrangements being completed, paracentesis corneae was done: but no immediate improvement in vision resulted. Meantime further ophthalmoscopic examination was futile.

Ophthalmoscopic examination of the right eye.-

Inspection of the right eye showed a dark fundus and a reddish disc with blurred edges: the arteries were typically of the "silver wire appearance" and unduly tortuous: the veins were also tortuous and full, and at two places visibly obstructed by the overlying arteries. The pigmentary disposition at the macula suggested some disturbance there.

Family history:-

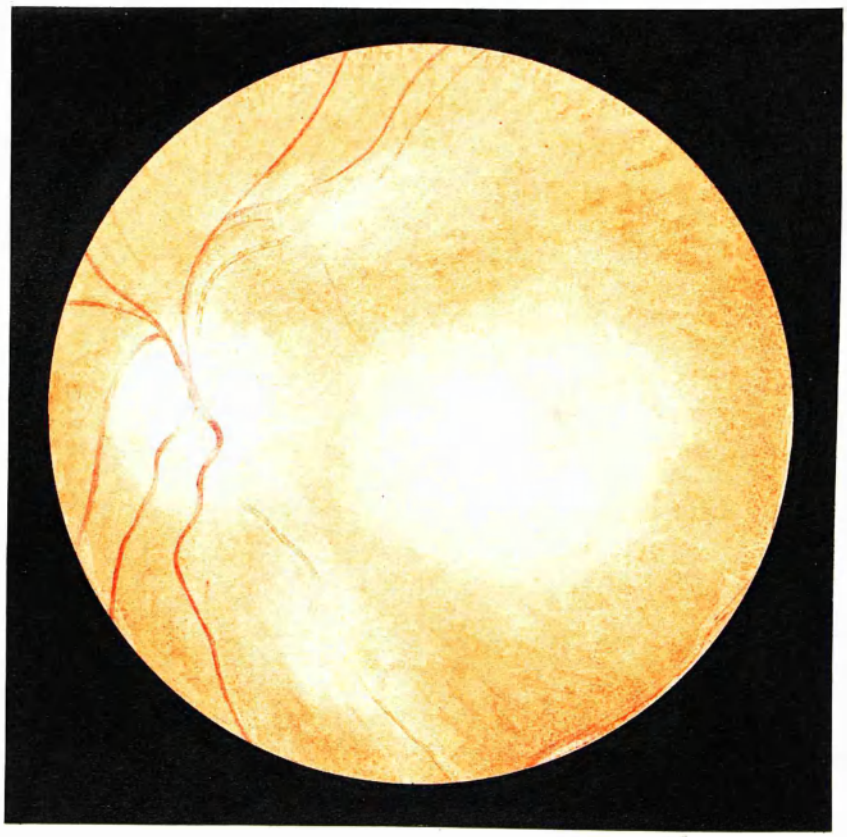
Father died suddenly at fifty-four (cause unknown).
Mother died at sixty from apoplexy.
Had only one brother, who died at forty-eight from apoplexy.

Personal history:-

Married and widowed: first three pregnancies ended in miscarriages: then two children were born but both died in infancy. Has suffered much from chronic bronchitis during the past ten years: two years ago she had a 'peculiar turn' which was

Fig. II.

Case I. G.G.- The fundus of the left eye twelve hours later: note the distribution of the oedema and the appearance of the cherry-red spot: the margin of the disc is very blurred: the arteries are of larger calibre and show blood cylinders: the veins are very prominent.



described then as 'a slight apoplectic stroke:' there was no affection of the limbs but she became giddy and confused and had difficulty in speaking: this passed away in a day or two and she has had no recurrence: has never had any transient attacks of blindness in either eye, etc.

General Condition:-

Tall, obese and complexion pasty: lips showed slight lividity: the arteries (radials and temporals) were rigid and tortuous and the tension rather high: chest emphysematous and signs of chronic bronchitis marked: no apparent enlargement of the heart and the sounds, though faint, seemed pure. Urine: sp. gr. 1024, acid: repeatedly showed a slight haze with the heat test: no tube casts found. No other abnormality.

On the following day ophthalmoscopic examination showed that the oedema was lessening: the disc was still very blurred and its margins swollen: the macular region was pallid and oedematous: here the diffuse redness, already noted, was better demarcated: on minute inspection a ring, a little smaller in circumference than the disc, and enclosing a slightly paler area, was seen and in the centre

of this was a small red spot. The arteries were barely discernable upon the disc and their course was, in places, quite obscured. They now seemed normal in size and the blood stream, though still broken into cylinders, was much more rapid. Pressure on the eyeball readily interfered with the current, but it now required considerable pressure to stop it entirely. The veins appeared unusually prominent but did not show much enlargement: the inferior branches were unduly tortuous and their peripheral portions dark and relatively full: pulsation was obvious and increase of intraocular pressure caused marked engorgement. There were no haemorrhages seen.

The last examination was made four days later, as patient was leaving hospital. The fundus was beginning to take on a dull opaque look: the posterior pole was greyish-white in tint and the red spot, delimited as before, was now dark red in colour. The arteries and veins showed little change though, perhaps, the current was less vigourous and more easily stopped. Perception of light was still quite lost.

As patient lived at a considerable distance I have had no opportunity of making a

further examination but I learned that the eye has remained quite blind.

CASE II.

J.C., aet. 33, railway-man, single.

Complained of loss of sight in the right eye of fifteen hours duration.

He stated that eight days ago, while at work on the top of a waggon, unloading coal, the sight of his right eye suddenly became dimmed. He had no other symptoms and felt in the best of health. The vision was reduced to dim perception of light but after the lapse of a few minutes it began to clear somewhat and three hours afterwards only central vision was affected; he described it as "a ball of mist in front of the sight" and when he looked at a face he saw the forehead and chin but not the central features. This gradually cleared, the misty ball getting smaller and smaller, and eight days after the onset he considered his vision normal. On the evening of that day his right eye

again suddenly went quite blind, and next morning he came to the Infirmary.

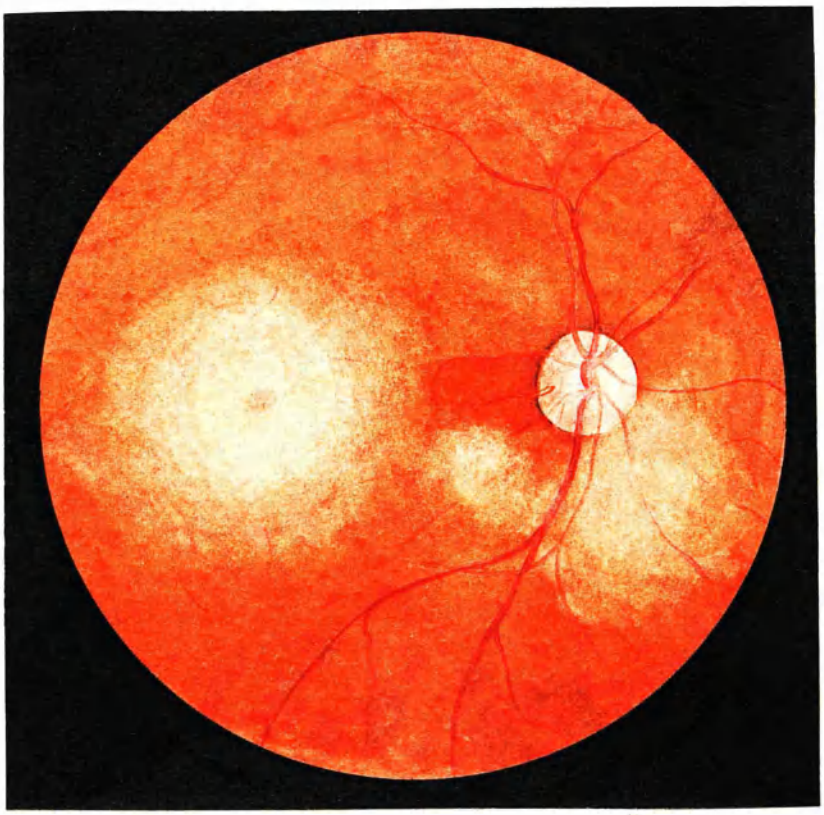
On examination the right pupil was slightly larger and reacted sluggishly to light: tension normal. Vision was reduced to faint perception of light with a small part of his field. In the left eye vision was normal.

Ophthalmoscopic examination of the right eye.-

The fundus, as a whole, was of good colour, the disc of an even pinkish-red, margins distinct except, for a small part at the inner and lower edge, where the outline was blurred and led into a hazy part of the fundus. At the outer side of the disc a quadrant-shaped patch of dark red fundus was seen, in size a little longer than the disc and three-fourths its breadth. The borders of this patch were fairly well defined, the superior macular artery being seen at its upper and the two branches of the inferior macular, crossing its lower edge. In the centre of this area, a cilio-retinal artery was seen: it emerged near the border of the disc and disappeared before reaching the outer limit of the patch. Two large areas of oedema were seen: one situated below the disc and partially obscuring the main trunks of the inferior

Fig. III.

Case II. J.G.- The fundus of the right eye on admission: on the whole it is of good colour: note the distribution of the oedema; and the area of the retina supplied by the cilio-retinal artery: the arteries are not markedly narrowed and contain blood: the veins are darker than normal and slightly irregular.



blood vessels, the other surrounding the macular region: the macula itself appeared as a very faint, diffusely red, spot, somewhat oval in shape: it had a 'stippled' appearance, the centre slightly darker and the edge fairly abrupt: amid this oedema, surrounding the macula, a circle of small parts of tortuous vessels was just discernable. No haemorrhages were noted.

Vessels.-

The ratio in size between the arteries and the veins is altered, but it is uncertain whether this is due to a slight constriction in the arteries or to a slight distension in the veins. Both the superior and the inferior arteries are imperfectly filled, and pulsation, especially over the disc is well marked. On careful examination the blood current is seen to be granular and runs along with a fluttering movement. On pressing the eyeball the arteries become thicker and redder and if the pressure is increased sufficiently, pulsation and circulation can be stopped. The cilio-retinal artery, above noted, seems well filled: no pulsation can be detected and no alteration is noted in it during the pressure experiments.

The veins are darker than normal and

slightly irregular in outline: just as they reach the disc's edge they become lighter in colour and at the centre of the disc appear comparatively empty: through this light section a tiny stream of blood runs in a wriggling fashion: very slight pressure on the eyeball stops this current and drives the blood from the sections of the veins upon the disc. The blood in the veins is decidely granular, and pulsation noted.

Ophthalmoscopic examination of the left eye.-

The fundus was of a dark-red colour and showed a dirty tesselation: the disc was 'fiery' and surrounded with a broad choroidal ring: both arteries and veins were skightly tortuous and very full in appearance: the periphery of the fundus was markedly pigmented, as was also the macular region. No abnormalities were noted.

Family history.-

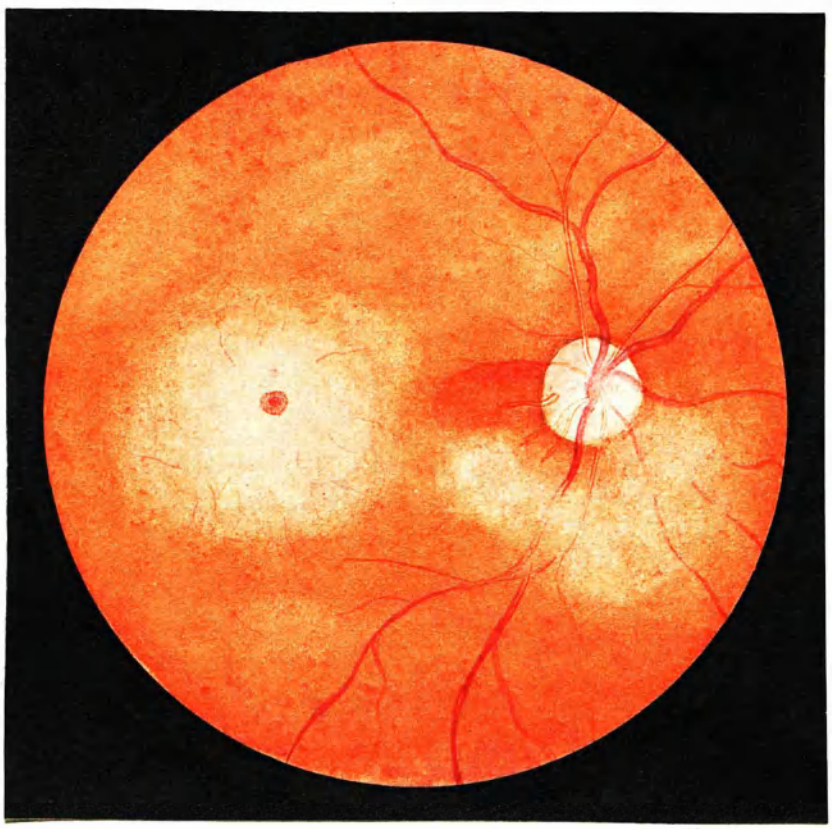
Father living and well, aged fifty-eight: mother died in childbed: five brothers and one sister alive and well: one died in infancy and three were still-born.

Personal History.-

Has always been very healthy: at twenty had a

Fig. IV.

Case II. J.C.- The fundus of the right eye ten days later: note the paller and the conspicuousness of the cherry-red spot; the arteries are paler and imperfectly filled; the veins dark and tortuous.



chancre, followed by the usual secondaries: drinks a large amount of alcohol.

General condition:-
Strong, well-built, muscular man: florid complexion: pulse forcible and moderately high-tensioned: heart normal: second sound accentuated. Urine normal.

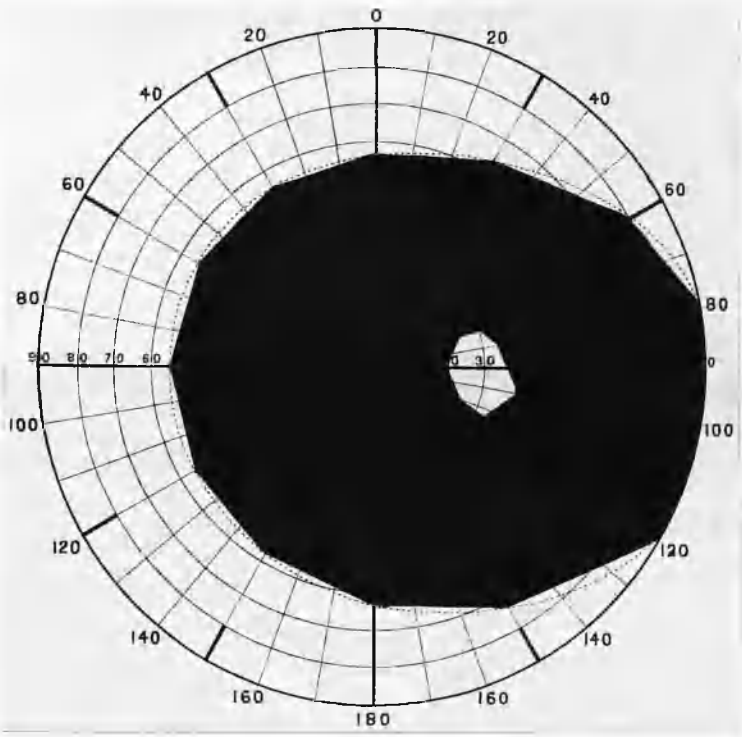
During the next ten days the fundal condition showed little change beyond clearing somewhat: his vision, however, had improved and he could now count fingers with a small part of his temporal field. The day afterwards the superior temporal retinal artery was dim, narrowed, and very imperfectly filled: from its origin and for the length of three discs-breadth upwards, it appeared quite empty: on watching, however, one could see, at infrequent intervals, a tiny and very granular, stream passing slowly up the vessel: on the other hand, the superior nasal retinal artery was well filled and but slightly granular: pulsation was detected on pressure. No noteworthy change in the inferior arteries, but all the veins were becoming more engorged and swollen and presented a livid, lumpy look. The macular region

Fig. V.

Case II. J.G.- Perimeter Chart of the right eye taken three weeks after the onset of the obstruction.

Fig. VI.

Case II. J.G.- Perimeter Chart of the right eye one year later; note the area of relative scotoma.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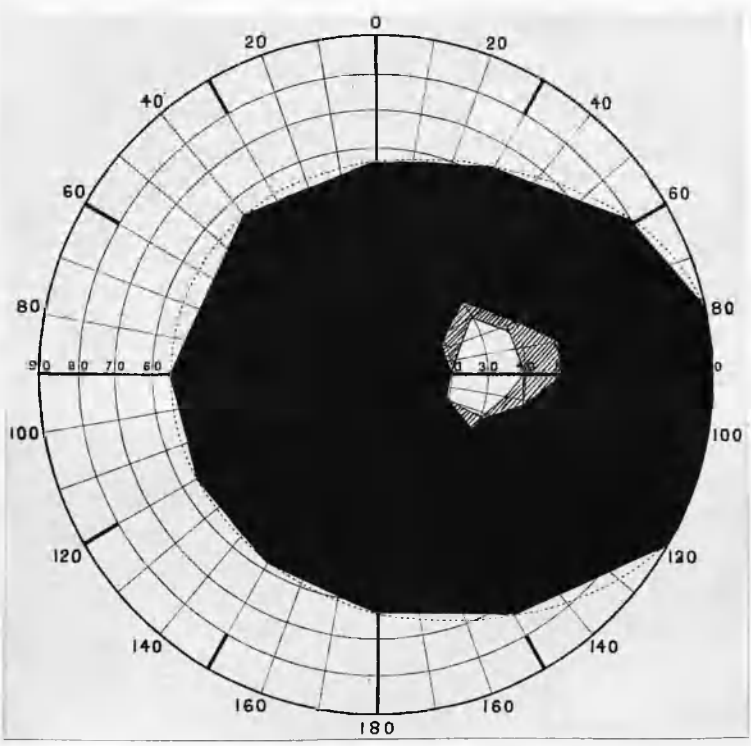
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in them could be stopped upon the disc. It could not be said that the cilio-retinal artery was visibly enlarged.

Ten days later the fundus was clear and of practically the same colour as its fellow: the retinal striae were still faintly seen: the disc was becoming paler: the whitish opacity in the macular region had disappeared and the macula itself was of a brownish, granular appearance, and inferiorly to it were numerous brilbiant, white pin-point spots. The veins were full but not at all tortuous: the arteries seemed fairly normal but it was noted that the arterial pulse could be more readily induced in this eye than in the left. On careful examination of the arterial walls I could now satisfy myself that at places they were covered with small areas of greyish-white opacity: similar appearances could be detected in the left eye on minute inspection. His visual field has improved considerably, more especially on the temporal side, and he was able to read $\frac{20}{200}$ on Snellan's Test Type.

When seen one year later the circulatory appearances were still very similar: the cilio-retinal artery had not increased in size: but two minute vessels, very tortuous, were now seen to

accompany it. I was unable to determine whether they were arterioles or venules. The disc was paler and its margins distinct; vision was certainly not improved but patient thought "the dimness was thinning."

CASE III.

J.K., aet. 54, engine-fitter, married.
Complained of blindness in the right eye of twenty minutes duration.
Patient had just ceased work at dinner-time and on coming out of the door into the sunshine he suddenly 'felt giddy and peculiar:' almost simultaneously the sight dimmed in both eyes: in a moment or two the vision quickly returned to the left eye and the giddiness passed away but the right eye remained quite dark. He came to the Infirmary and was admitted.

On examination the right pupil was semi-dilated and did not react to light: left pupil normal: tension in both normal. Visual acuity in right eye = Faint perception of light: in left eye = $\frac{20}{20}$.

Ophthalmoscopic examination of right eye.-

Fundus rather pale, especially towards the macular region: the disc was pallid with margins clearly defined and the arteries were thread-like and empty: the veins also looked empty and flat. Pressure on the eyeball produced no change. In three or four minutes oedema of the retina began to show, the edges of the disc getting velvety and soft: in the macular region the disposition of the oedema caused the central spot to appear as a diffuse reddish haze, in size, somewhat larger than the disc.

Thirty-five minutes after the onset the return of the circulation was noted in the superior temporal branch. Here, little cylinders of blood entered in a slow, jerky fashion, the current showing similar cyclic alterations to those noted in case I: at first the reverse waves caused the blood to entirely disappear at times, but it gradually mounted higher and higher until the whole visible length of the artery showed a procession of oscillating blood cylinders. Shortly thereafter, the inferior temporal artery began to fill in a similar fashion but here the current was much slower and at times quite stationary: whilst in

this latter condition a definite, and periodic to-and-fro, movement was manifested. The corresponding nasal branch showed similar appearances.

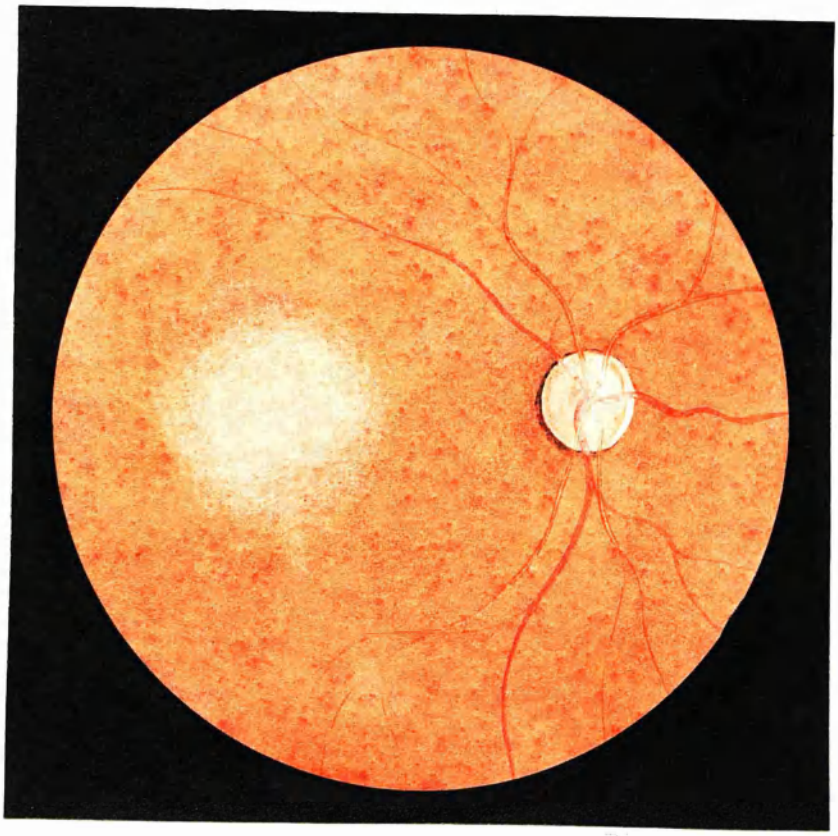
The veins were now filling up and becoming dark and uneven: the current was very slow and granular and only indefinitely pulsatory: pressure on the eyeball readily emptied their proximal portions and caused peripheral engorgement.

Two hours after the onset a small striate, flame-shaped haemorrhage appeared, just outside the disc and mid-way between the branches of the superior temporal and the superior nasal retinal arteries. The oedema had steadily increased and was especially marked around the disc, along the larger vessels, and in the macular region. The circulation was gradually improving and the current, more particularly in the superior temporal branch, was now very rapid: the little cylinders rushed along and were barely separately distinguishable. Increase of intra-ocular tension slowed the current and elicited distinct pulsation.

Soon the oedema became so marked that the vessels were in large part obscured and, especially around the disc, the differentiation of arteries and veins was impossible. At this time (six hours

Fig. VII.

Case III. J.K.— The fundus of the right eye on admission: rather pale, especially around the macular region: arteries are narrowed and colourless: veins flat and somewhat uneven: all the vessels upon the disc look empty.



after the onset) further ophthalmoscopic examination of the eye was postponed.

Ophthalmoscopic examination of the left eye.-

The fundus seemed healthy: no abnormalities noted in the vessels: disc normal.

Family history.-

Mother died at thirty-eight in child-bed.

Father died suddenly at seventy-eight - cause unknown.

Patient was one of a family of three - the other two are alive and well.

Personal history.-

Had typhoid fever in childhood: otherwise has always been exceptionally healthy: states that he has not been a day off his work in thirty years and was in the best of health on the day he went blind. No previous visual disturbance.

Had six of a family - two dead, one from pneumonia; the other, the result of an accident.

Is a total abstainer and no history of venereal disease.

General condition.-

Patient looked robust and healthy: no abnormality detected in the pulmonary or vascular

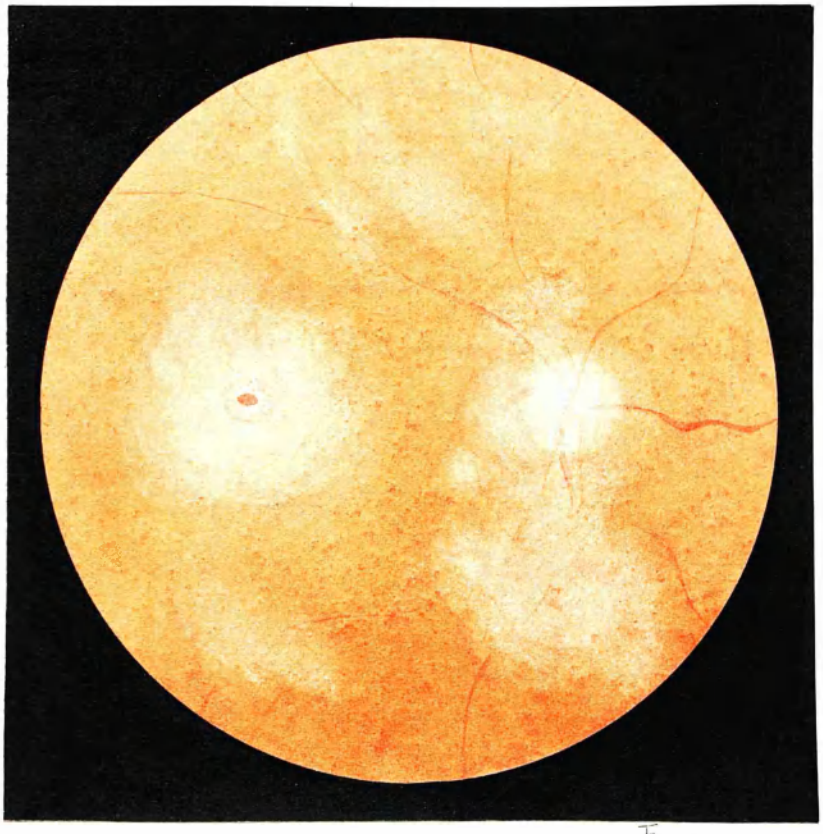
systems: urine normal.

Further ophthalmoscopic examinations.-

Next day the fundus was still remarkably oedematous but beyond this fact nothing further could be determined. On the following day it was beginning to clear towards the periphery and the outline of vessels could be dimly seen. Two days later the haze had cleared sufficiently to permit of further observation: what attracted most attention was a small, indefinitely flame-shaped, haemorrhage at the outer side of the disc. None of the vessels could be followed far and the arteries and veins were still largely indistinguishable. Six days after the onset a further small haemorrhage was detected, a little above, and to the inner side of, the macula: it was round and did not appear to be striate. The macular region was opaquely white: in its centre a thin reddish ring, slightly larger than the disc, was seen and in the middle of this a small dark red elliptical spot. The veins were dark, uneven and tortuous: the arteries still showed rapidly moving blood cylinders and pulsation was readily elicited on pressure. Faint perception of light was now

Fig. VIII.

Case III. J.K.- The fundus of the right eye twenty four hours later: very oedematous: vessels traced with difficulty: note the appearance of the macula, and the small haemorrhage at the outer side of the disc.



present. On this day patient left hospital.

Ophthalmoscopic examination one week later.-

The fundus was dull and opaque-looking and showed traces of oedema in its lower half: the disc was pale and yellowish and its nasal and inferior borders well defined: the outer side was still much swollen and obscured the vessels passing over it: faint traces of the two haemorrhages, noted here, could still be seen. The superior arteries were of good breadth and could be followed far up the fundus: they were very light in colour and empty-looking: these vessels probably contained some blood, as marked pressure on the eyeball whitened them considerably. The vein was large, dark and uneven, being especially swollen just before reaching the disc's border: upon the disc it was much narrower and paler. Pulsation in the vein was easily elicited on pressure and imparted an eel-like movement to the vessel. On account of the oedema, the lower vessels were more obscure; the arteries showed a faint circulation but no pulsation: the inferior veins exhibited appearances similar to the superior branch. At the posterior pole there was a markedly pale area about

twice the size of the disc: around this, and over its margin, tortuous vascular twigs were very evident. In the centre of this area was a small greyish-brown spot with soft edges: the haemorrhage in this region was now only visible as a faint granular, brownish pigmentation.
Perception of light was doubtful.

The last ophthalmoscopic examination I had the opportunity of making was about a year after this. I then found the typical picture of retinal atrophy. The disc was a dirty greyish-white with well defined margins: the arteries were mere streaks becoming invisible towards the periphery. The veins, which were much narrowed, pursued a straight course.
There was no perception of light.

Examination of the left eye revealed no change and vision was normal.

CASE IV.

W. A., aet. 69, watchman, single.

Complained of blindness in the left eye of four hours duration.

He stated that four days ago the sight of the left eye became slightly dimmed: it then cleared up considerably but on the morning of that day had gone quite blind.

On examination the pupils of both eyes were rather small and did not react to light: tension in both was normal. Visual acuity in the right eye was :- $\frac{20}{40}$: in the left eye there was no perception of light.

Ophthalmoscopic examination of left eye.-

The fundus was pale but not markedly so: the disc was of a dirty yellowish-red tint, its nasal border being well defined but the temporal one oedematous and swollen: the arteries were reduced in size, pale and empty-looking: upon the disc the arteries seemed quite bloodless: excepting the superior nasal branch, none of the arteries could be traced far, the superior temporal branch, a mere reddish streak, soon becoming invisible: the inferior branch was of larger calibre but about a disc

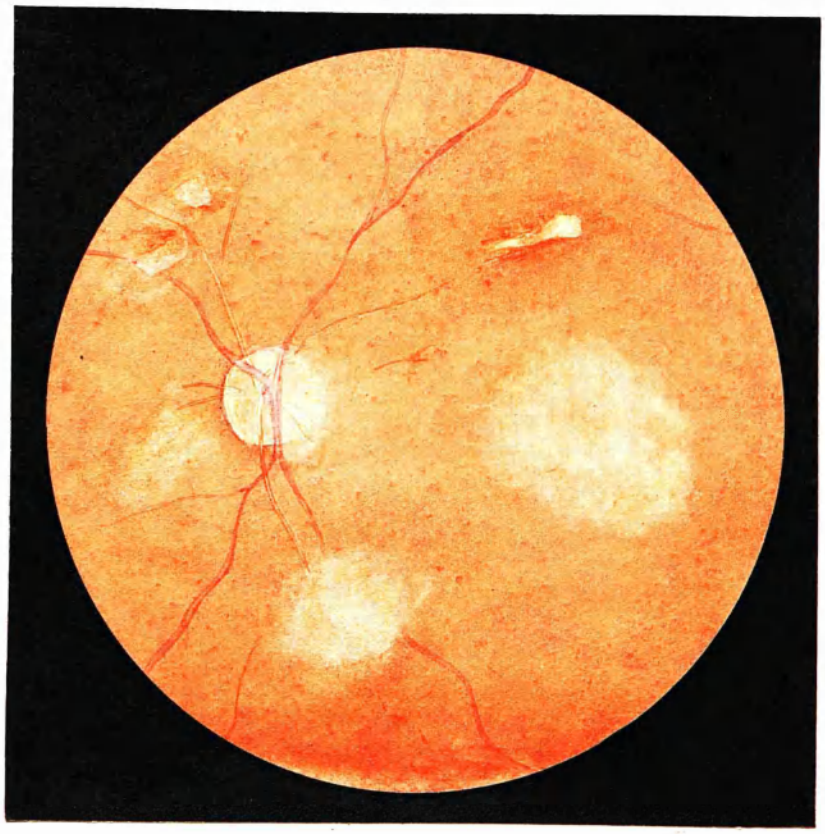
and a half breadth down it was quite obscured in a patch of oedema.

In marked contra-distinction to the arteries, the veins were very prominent and dark: their outline was uneven and irregular and in places much narrowed: upon the disc the veins were broad; empty, and of a bluish appearance: no pulsation noted. On pressure a faint pulsation was seen in the inferior artery, exhibiting itself as a slight rhythmical reddening of the vessel wall: at the same time the veins upon the disc became paler; whilst their more peripheral portions seemed fuller. One or two macular twigs were seen on the disc and on the nasal side two small arterioles were noted, evidently branches of the central artery of the retina, given off before its emergence on the papilla. There was an area of considerable oedema at the posterior pole of the eye and in the centre of this the red spot was faintly discernable.

In the upper half of the fundus there was evidence of gross retinal disturbance: there was a triangular atrophic area in the course of the temporal branch and above the macular region: its borders were well defined and showed recent striate haemorrhages. At a corresponding site in the nasal

Fig. IX.

Case IV. W.A.- The fundus of the left eye on admission: temporal border of disc oedematous: arteries are reduced, pale, and empty-looking: veins are prominent and dark, and on the disc, broad, opaque, and bluish: note the retinal disturbances in the superior half of the fundus.



half of the retina were two similar areas, one on either side of the superior nasal retinal artery: they were smaller and not so definite in outline: traces of recent haemorrhage were noted around these spots.

During the time the eye was under observation a small haemorrhage had appeared at the upper border of the disc: it was flame-shaped and striate and during the examination (about two hours) very slowly increased in size.

Ophthalmoscopic examination of the right eye.-

The fundus showed considerable abnormality: the disc was hazy and yellowish and its margins were blurred and swollen: the arteries were light-coloured, the central streak being well marked: they were unduly tortuous and irregularly narrowed at places. The veins were uneven and showed signs of the mechanical obstruction of the over-lying arteries. There were evidences of old extravasations and the macula showed a faint pigmentary mottling.

Family history.-

Father died at forty-five from a 'shock.'

Mother died at thirty-seven in 'child-bed.'

Has four sisters and three brothers: one sister died at fifty-six from apoplexy: no history of the others obtainable but he thought they were alive.

Personal history.-

Until within the past few years he had always been a very healthy man: was a heavy drinker and smoker and had led a very loose life: had gonorrhœa several times but never aware of a chancre.

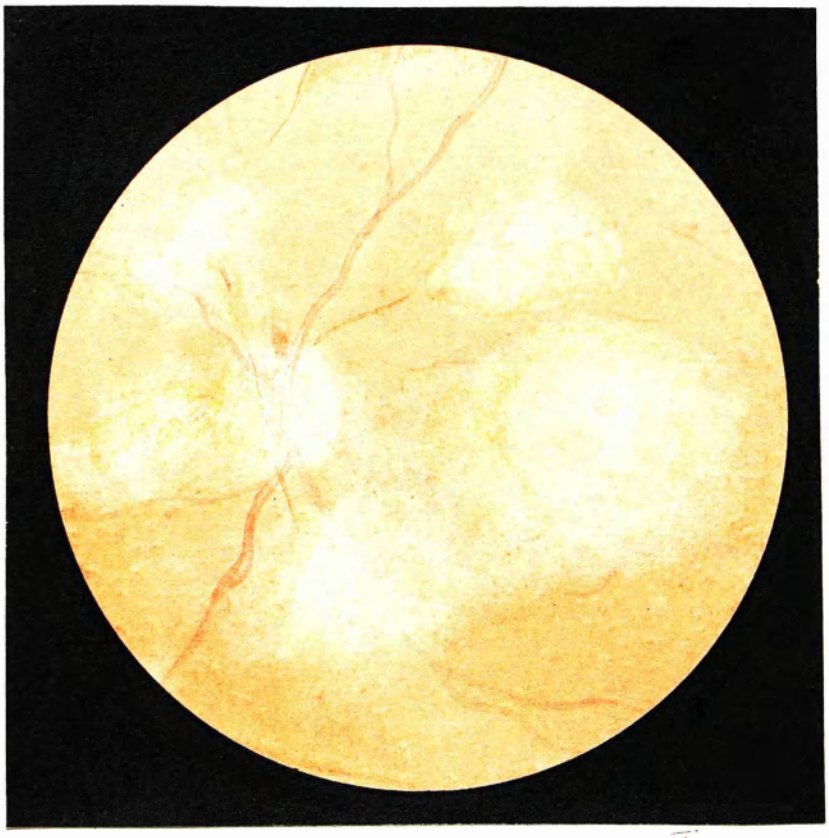
About a year before this he suddenly lost the sight of the right eye: the vision slowly returned during the next few weeks: three months later he had a recurrent attack of blindness in the same eye, and this time the vision did not return so completely. There had been no previous attacks of transient blindness in the left eye and before the onset of this affection it was his "good eye."

General condition.-

Tall and well-built: face florid and blotched: marked hardening and tortuosity of all the superficial arteries, the temporals and radials being specially affected. No abnormality of the heart detected. Urine - sp. gr. 1020. No albumin nor sugar.

Fig. X.

Case IV. W.A.— The fundus of the left eye twelve hours later: pallor and oedema now marked: veins prominent, engorged, and tortuous: note the obstruction in the inferior nasal venous branch due to an overlying artery: the arteries, where seen, are narrowed, but contain blood: observe, also, the small striate haemorrhage at the upper border of the disc, and the appearance of the macula.



Next day the retinal oedema had greatly increased, being especially marked in the macular region and at intervals around the disc. The small haemorrhage at the upper border of the disc was very evident but had not obviously increased in size. The veins were broad, turgid and tortuous and the inferior nasal venous branch was somewhat obstructed by the crossing of an artery. This vein was repeatedly seen to fill up and then discharge over its papillary portion in an irregularly pulsatory fashion: a similar, but less evident, phenomenon was noted in the other veins. Distinct circulation and pulsation was noted in those arteries which were unobscured: moderate pressure on the eyeball sufficed to stop the circulation entirely.

Faint perception of light with the upper half of the field was now present.

Patient left hospital on this day and the next examination (which proved to be the last) was made five days later. I then found that the oedema was clearing: the disc was very pale and the arteries mere threads: no evidence of circulation obtainable. The veins were dark, uneven and pulseless and marked pressure produced no change.

The haemorrhage at the superior border of the disc was slightly larger, but no fresh ones noted. The 'cherry-red spot' was now seen as a clearly defined elliptical reddish-grey spot with a faint reddish halo surrounding it: outside this the rest of the macular region was opaquely white and against this background minute retinal twigs were strikingly evident. The eye was now quite blind. Patient did not again return to hospital and I was unable to trace this case further.

SECTION III.

To formulate a diagnosis in these cases of Obstruction of the Central Artery of the Retina is a matter of some difficulty and complexity. So many conditions have to be investigated, so many factors weighed, that the temptation to rely upon the stereotyped diagnosis of 'embolism' is undoubtedly great. Yet, when we read many of the reported cases, we cannot but be struck with the discrepancy between the physical condition of the patient and the hypothesis of 'embolism.' The physical examination so often reveals nothing abnormal, the history of the patient before and after the occurrence so uneventful that the idea of the sudden appearance of an isolated embolus, and its impaction in the central artery of the retina, seems far-fetched and fanciful. Further, in those cases where we have a history of visual or cerebral disturbance, prior to the onset of the affection, any explanation based on the embolic theory seems improbable and inadequate.

In Section II, I have given reports of four cases of so-called embolism - a heart lesion

was not discovered in any. To what, then, must we look for a cause for the obstruction?

Let us consider first the mode of onset in these cases. In Cases I and III the blindness was sudden and permanent, no previous attacks of transient blindness in either eye: in both, the onset was accompanied by a simultaneous affection of the other eye and signs of cerebral disturbance. How could an embolus, minute enough to enter the central artery, cause all this disturbance and if there were several emboli why was the action of the others so transitory?

In Case II (J.C.) we have a history of an attack of blindness in the same eye eight days previously - the sight gradually returning until he thought his vision normal again, then a recurrent attack. Here, again, the heart was normal; the vessels, on the other hand, showed changes and the history of syphilis and alcoholism was definite. If this be accepted as a sufficient source for an embolus we are met with the difficulty of explaining the previous attack, with gradual but complete recovery, and the fact that when first seen there was a distinct pulsating current in the vessels.

In Case IV the heart showed no abnormal-

ity: the arteries, however, were markedly rigid and he was a heavy drinker. There was a history of two previous attacks of temporary blindness in the other eye (eventually, leaving the vision somewhat impaired) and an attack of blindness in the same eye four days before. Can the theory of embolism as the causative factor be accepted here? In the condition of the vessels we have a probable source for such a plug; but, in view of the history of attacks of prodromal periodic blinding, I consider this hypothesis largely untenable as well as improbable.

The theory of Primary Thrombosis I have previously considered and feel constrained to regard the idea of thrombosis, as the primary affection, quite inapplicable to the great majority of the cases: as a secondary process, however, it must be regarded as of the greatest importance. In arterial disease we have a factor common to three of our cases and in the other (Case III) we cannot negative its existence. As we saw, this may act primarily, and cause obstruction, either by obliteration of the lumen or by a narrowing, sufficient to permit of the incidental approximation of the vessel walls. That is, doubtless,

a probable explanation of our cases. But, perhaps, arterial disease acts more commonly through the occurrence of intimal changes, so that thrombosis, under certain circumstances, readily develops. Hence arterial disease, or thrombosis secondary to arterial disease, might explain the clinical facts of these cases; and Haab goes so far as stating that obliterative endarteritis, or thrombosis, is the usual cause (embolism, in his opinion, being very rare).

In Cases I and III the history of onset differs in some important particulars from the others. In both the blindness was sudden and complete, and accompanied by a very transient obscuration in the fellow eye, with slight cerebral disturbance. In Case I these symptoms were very slight and described by patient as "a dazzling:" in the other case (Case III) the simultaneous disturbance was more definite and lasted longer.

In view of the possibility of reflex retinal arterial spasm, we must bear in mind that both were affected by the sudden action of bright sunlight; the patients, themselves, attributed their blindness to this cause. This point is worth considering. Apart from the photo-chemical action

in the retinal elements, it may be that the sudden effect of bright light on the retina is to cause (in certain cases, at least,) some degree of reflex vaso-constriction. When a bright light is shone on the retina (hitherto in darkness) it has been noted that, in rare cases, the pupil suddenly contracts but almost immediately dilates to some extent: then either gradually and regularly, or with a hippus-like action, slowly contracts - the time occupied being very variable, for less than a minute to over an hour. I have on a few occasions observed this in the examination of the eyes in the insane; this reaction is supposed to be more common in this class and in those of a neurotic temperament (Juler, Oppenheim). As the primary contraction and the subsequent dilatation is often very sudden, it is sometimes overlooked and the pupils considered as not reacting to light or reacting very sluggishly.

This phenomenon is known as Paradoxical Pupillo-dilatation. Much experimental work has been done on this subject. Budge (1855) first called attention to its occurrence after section of the left sympathetic and of the branches above the right superior cervical ganglion in a rabbit.

Kowalewski (1886) observed it in a kitten after the administration of chloroform. Langendorff (1900) has noticed the phenomenon after anaesthetics and after death; the latter and Surminski explained the reaction as being due to the contraction of the vessels in the iris. Hence may we not conjecture, in those cases where it is demonstrable, that the light, primarily causes a reflex contraction of the pupil but almost simultaneously a vaso-motor constriction (through the action of the sympathetic): and that this action of the sympathetic counteracts the pupil-contraction, so leading to some degree of dilatation?

However this may be there is evidence enough to show that in those cases of spastic retinal obstruction, due to a general cause, the pupils are dilated (Sect. 1. - retinal spasm). Further, Cases I and II also showed this dilatation in the affected eye, gradually passing off in the course of a few hours.

In connection with the probable action of sunlight an interesting case is recorded by Dr. Rayner Batten (Ophth. Soc., Jan. 31, 1901). The patient was a woman, aet. 28, who watched the eclipse of the sun on May 28, 1900: immediately

thereafter, she noticed that the sight of the left eye was dark and the next morning found that she could only see "portions of things." First seen on June 6 and found to have lost the lower half of the field of vision of the left eye. Above the optic disc a white patch was seen, probably an absorbing haemorrhage, whilst the hazy and oedematous retina obscured the view of the disc and vessels. The oedema increased until June 20 and then rapidly cleared. One of the upper retinal arteries was occluded and the other reduced in size. Patient was in good health and no other cause for the affection could be found, except exposure to sunlight, which Dr. Batten thought had induced thrombosis of the retinal arteries. He offers no explanation for the occurrence of this arterial thrombosis nor does he state whether he thought it primary, or secondary to some other condition.

Before leaving this part of the subject, I should like to give an excerpt from a reported case of Embolism of the Central Artery of the Retina following Paraffin-injection for Deformity of the Nose (Drs. Hind and Holden.- Medical Record, July 11, 1903). The patient was an Italian, aet. 32, and the deformity was probably syphilitic.

At the third operation the needle, for injecting the paraffin, was first introduced at the tip of the nose and pushed upwards and then introduced at the root of the nose and pushed downwards to a spot just above the former injection. At this point the patient rubbed his right eye and stated that he could not see with it. Some ecchymosis appeared at the tip of the nose, indicating the puncture of a vein. The ophthalmoscopic examination was made twenty-five minutes thereafter. The pupil was then large and did not react to light: no perception of light present: media clear: retina clear and veins seemed normal: the inferior arteries were empty and collapsed, being recognisable only by the faint outline of their walls. The superior arterial branches contained blood cylinders and gentle pressure on the eye was sufficient to empty the vessels. Two hours later, the disc had become blurred and the retina hazy: the oedema increased and the red spot at the macula was clearly seen. The loss of vision persisted.

No further particulars are given. As was said, this was reported and has been since commented on, as a case of embolism of the central artery of

the retina, due to blockage by a small paraffin plug. To admit of this diagnosis, we must presuppose that the plug entered a small vein in the nose - reached the heart - found its way into the arterial system through some such abnormality as a perforate septum ÷ and then came back to lodge itself in the central artery of the retina ! Surely this explanation is far-fetched; to my mind this case is more explicable as one of reflex arterial retinal spasm, and the fact that the pupil was dilated and did not react to light favours this view. The patient was, probably, syphilitic and secondary thrombosis might have, eventually, occurred.

In the consideration of Cases I and III, we must bear in mind all these points; and after carefully considering the mode of onset, the early symptoms, and the general condition of the patients before and after the occurrence, I am of the opinion that these cases were probably, primarily, spastic in origin. Doubtless, the presence of arterial changes etc. had some part; not only in its determination (vide - spasm in Migraine and Raynaud's disease) but also in the

eventual, permanent blockage - whether thrombotic or proliferative.

The blindness in cases of obstruction of the central artery of the retina is, as a rule, complete and almost instantaneous; in two of our cases the blindness was complete but the other two had faint perception of light when first seen. The obscuration, however quick, is generally in a centripetal direction; occasionally, it takes place in a centrifugal manner, as in the previous attack of Case II.

In none of the cases was the intra-ocular tension appreciably diminished. This is the rule and is usually explained by the fact that the ciliary circulation is still intact; that is, doubtless, a sufficient explanation.

The appearance of the arteries is very variable and great differences are observed. If seen soon after the onset of the obstruction (as in Cases I and III), the larger arteries are markedly narrowed and the smaller ones invisible. Frequently, they appear to be quite empty and most authors describe them as such, but it is difficult to believe that any obstruction of an

artery could be so complete as that; under the blood pressure, either a tiny stream of blood or liquor sanguinis would surely be forced through.

The veins, on the other hand, do not show such marked changes. On the disc they are often narrowed and empty-looking - becoming either relatively, or actually, broader towards the periphery. They show an uneven calibre and sometimes present ampulliform swellings; they are darker in colour than normal.

It is somewhat difficult to understand why the intra-ocular tension does not force the blood out of the veins, after the arterial blockage. Parsons considers this to be the most potent factor: "the ocular venous tension is lowest at the disc, so that the veins are stopped here first by the extra vascular pressure, and the blood is dammed back."

The re-establishment of the circulation is of great interest. From the recorded cases it seems to take place at very varying periods - from less than an hour to several days. In Case I, the inferior temporal artery began to fill with a broken column of blood within forty minutes: in

Case III a similar return was noted in the superior temporal branch thirty-five minutes after the onset; (in Cases II and IV the circulatory return had taken place when they came under observation). Almost simultaneously a manifest circulation was noted in the veins - in Case I the blood column becoming broken into cylinders and in Case III, having merely a granular appearance. In no case did the stream move in a reverse direction to the normal blood current (ofcourse, excepting the reverse wave in the cyclical alternations - to be afterwards commented on). As many of the recorded cases, where this reverse current is noted, did not come under observation until some time after the onset of the obstruction, we must accept its reported occurrence with reserve. I have already stated, in the report of my cases, that, after the commencement of the retinal haze, the differentiation between arteries and veins, especially arround the papilla, became extremely difficult and occasionally quite impossible; hence, unless a case is under continued daily observation, errors are almost unavoidable. The phenomenon is undoubtedly much rarer than is supposed but has been described by Jaeger, von Graefe, Mayerhofer, von Hippel, Hirschberg, Perles and others.

The breaking of the blood column into cylinders, or becoming visible granular, is of interest and of somewhat obscure origin: some vessels show a tiny stream of red blood, others show blood cylinders of varying size and movement, while others again exhibit mere granularity. In our cases the cylinders first appeared at the initial portions of the vessels but cases have been described (Gowers, Fuchs) where pressure on the eyeball broke the continuous blood column into separate cylinders. Our knowledge of the physiology of the blood and blood vessels is not sufficiently exact to positively explain these and allied phenomena. As Welch points out, the absence of lateral pulsation may be a factor of great importance. The circulatory conditions are peculiar, and the physical properties of the blood, in relation to its viscosity and the presence of suspended particles, which readily stick together, have to be taken into account. In the experimental research of Welch and Mall, in the mesenteric circulation of the dog, they observed, in the veins and capillaries, "interrupted columns of compacted red corpuscles with intervening clear spaces which are sometimes clumps of white corpuscles, sometimes

of platelets, sometimes only clear plasma."
Although this experimental blockage was arterial, there is no mention of similar appearances in the arteries. In the retinal arteries, when the current is static or greatly reduced, the corpuscles evidently undergo some physical change and show a tendency to clump - and the absence of the normal pulse-waves prevents the breaking up of these masses of corpuscles. However, I have noted that in those arteries which show a continuous stream of cylinders in their initial portions their peripheral parts exhibit a compact blood column. So, perhaps, the question of capillary attraction and the ratio of the quantity of fluid to the calibre of the vessel ought to be taken into account.

A pulsatory advance of these cylinders in the arteries is not often noted: this was observed in Cases I and III; in addition, a rhythmical alternation of this pulsation was observed (vide Section II). This phenomenon is of interest and, so far as I can find, has not been previously commented on in cases of this kind. Physiologically, rhythmical alternation of the venous pulse upon the disc has been observed (Wadsworth and Putnam).

the cycle of stronger and weaker periods in the pulse, corresponding to about five respiratory movements. This periodic pulse must originate from causes independent of the eye and recalls those changes in arterial tension, noted by Traube, Hering, Meyer and others. It may result either from some rhythmic alternation in the blood pressure (Traube-Hering waves) or from rhythmical movements of the arteries, as observed, experimentally, in the ears of rabbits, by Schiff. In Cases I and III it was most evident in the arteries at the commencement of the circulatory return when the current was appearing as broken cylinders but not showing a continuous circulation. Under these circumstances, the opportunity for its accurate observance was great and, considering the almost complete suspension of the influence of the general blood pressure in these arteries, our observations seem to favour the theory that they may be, in part at least, dependent on some rhythmical movement of the arteries themselves. The subject is worthy of further study and these cases of obstruction appear to offer facilities for this.

The time of onset of the retinal oedema is very variable. Schnabel and Fischer have seen it two

hours, and Mittendorf three hours, after the obstruction. As usually stated, it may come on in a few hours but occasionally not for some days (Gowers). In the classical case of von Graefe the opacity did not come on until the eighth day. In Case I, it was noted forty minutes after the onset; in Case II it was present at the first examination - fifteen hours after the onset; in Case III it was noted twenty-five minutes after the onset; and in Case IV it was present at the first examination - made four hours after the onset. The distribution of the oedema is anatomical, being dependent on the thickness of the retina, and hence most marked around the papilla and at the posterior pole of the eye.

Haemorrhages sometimes appear co-incidently with the increase in circulation and enlargement of the calibre of the vessels. The haemorrhages occur in the vicinity of the disc and in the macular region, and, as a rule, appear from three to five days after the onset of the affection (Haab). They were seen in two of our cases (III and IV). In Case III, two hours after the onset, a small haemorrhage appeared just outside the superior border of

the disc; Two days later another small haemorrhage was seen at the outer side of the disc and six days after the onset; a third haemorrhage was noted in the macular region. In Case IV, a small haemorrhage appeared at the upper border of the disc, during examination, five hours after the onset; no others were observed. Haemorrhages are not very common and Fischer found them noted only forty-seven times in one hundred and fifty five reported cases. To explain their infrequency, the intra-ocular pressure, may be (among others) a contributing factor (Welch).

The red spot on the macula has been regarded by Fuchs, in some cases, as a haemorrhage and he states that he has repeatedly convinced himself of this. Graefe's explanation of the cherry-red spot was to ascribe it to the contrast between the white opacity of the retina surrounding the macula and the red chorioid shining through at this, the thinnest part of the retina. Blessig regarded it as a haemorrhage in the retina and Steffan as a haemorrhage situated, behind the macula, in the chorioid. Nettleship considered it as due to a circumscribed central chorio-retinitis.

Fischer explains it as being merely the pigment of the retina itself. Elschnig thinks that both the retina and the chorioid take part in its production. The consensus of opinion seems to be that the production of the cherry-red spot is essentially a contrast effect - the oedema of the inner layers of the retina stopping short of the fovea centralis (where these layers are absent) and leaving this part a bright red colour; this effect may be heightened by a hyperaemia of the chorioid due to an increased circulation in the ciliary arteries, consequent on the obstruction of the central artery. From observations on its development and appearance in our cases this view seems the most likely one - as its regular and ofttimes geometric disposition is certainly not consonant with the idea that it is either a haemorrhage or a chorio-retinitis.

One of our cases (Case II) was complicated by the presence of a cilio-retinal artery. The frequency of this in normal eyes, according to Elschnig, who examined one hundred and seventy persons is seven per cent. These arteries are derived from the scleral vessels (the circle of Zinn) and have no corresponding veins. Laqueur

collected sixteen cases of obstruction of the central artery of the retina, complicated by the presence of cilio-retinal arteries; the visual fields of these eyes were small, oval, triangular, or oblong, and extended from ten to twenty degrees in their horizontal diameters. In Case II, the perimeter chart, taken three weeks after the obstruction, shows a horizontal diameter of fully fifteen degrees: the chart taken one year later shows a slight increase to twenty degrees and a small area of relative scotoma surrounding it.

As to the large and debatable question of the establishment of anastomosis in these cases, clinical examination is of little assistance. Amid the great retinal changes that ensue after obstruction, decisive information on the enlargement of vessels or on the appearance of new ones, could hardly be expected: against the atrophic retina mere arterial twigs are seen with startling distinctness and on the pallid disc little vessels, erstwhile invisible, now make a notable appearance. However, there is no doubt that the terminal branches of arteries often appear almost normal whilst their initial stems are mere red threads upon the disc;

if this be due to the establishment of a collateral circulation we have, as yet, no anatomical knowledge of any such connection - because the chief anastomotic communications between the ciliary and the retinal vessels take place among the vessels of the optic disc: but the appearances noted, would require (if anastomotic) communications, not at the disc but towards the periphery.

CONCLUSION.

In this dissertation I have dealt with a subject of general medical, as well as special ophthalmological, interest. Its problems are the problems of Medicine and their solution, its solution. And, finally, I claim that sufficient evidence has been adduced on the etiology of this subject to warrant the discontinuance of the indiscriminate diagnosis of 'embolism' and, whilst awaiting further knowledge, to replace this term by the less definite but non-committal diagnosis of "Obstruction of the Central Artery of the Retina."

REFERENCES.

Juler. "Ophthalmic Science And Practice," 1904.

Oppenheim. "Diseases of the Nervous System,"1904.

Budge. "Ueber die Bewegung der Iris,"
Braunschweig, 1855.

Kowalewski. Arch. slav. de Biol., I, 1886.

Langendorff. Klin. Monatsbl. f. Augenheilkunde,
XXXVIII., 1900.

Surminski. Zeit. f. rat. Med., XXXVI., 1869.

Batten. Trans. Oph. Soc., 1901.

Hind. Medical Record, July 11, 1903.

Holden. Medical Record, July 11, 1903.

Mayerhofer. Inaugural Dissertation, 1873.

Hirschberg. Centralblatt fur praktisch
Augenheilkunde, 1884, S. 71.

Perles. Centr. f. prakt. Augen., 1891, S. 287.

Schnabel. Ueber die Embolie der Arteria
Centralis Retinae, Leipzig, 1891.

Fischer. Ueber Embolismus, 1891.

Mittendorf. Trans. Amer. Oph. Soc., 1892.

Gowers. "Medical Ophthalmoscopy," 1904.

Blessig. Archiv. fur Ophthal. Bd. VIII., S. 225.

Steffan. " " " Bd. XII., 1, S. 46.

Nettleship. Royal London Ophth. Hosp. Repts. vol VIII.

REFERENCES Contd.

Elschnig. Archiv fur Augenheilkunde Bd. XXIV., S. 140.

Laqueur. Archiv fur Augenheilkunde, 1895, Bd., XXX., S. 75.