GLAUCOMA

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ABSTRACTS

I.—GLAUCOMA


(1) Derby, Waite, and Kirk in this paper describe their methods and apparatus for accurate and delicate testing of the light minimum and difference sense. For details of these the original, which is adequately illustrated, must be consulted. The authors begin by plotting out graphs under the standard conditions for 100 normal eyes, discussing the nature of the curves obtained, and comparing them with those obtained from glaucoma patients and from glaucoma suspects. Their conclusions are as follows:

1. Opposed to the beliefs expressed in the standard texts, we believe with Hecht that, properly measured in the normal eye, dark adaptation proceeds at a precipitous rate for several minutes after the onset of darkness, and that it reaches almost its maximum in ten minutes.

2. For uniformity of results we believe that pre-exposure of the tested eye for five to ten minutes to a known intensity of light is a necessary procedure.

3. We believe that standardization of the pupil is a sine qua non for an accurate study of the light-sense, and we offer pilocarpine fixation as the method of choice over all other methods.

4. With the low intensities at which we worked we have been unable to find any significant alterations in the light difference (L.D.) in glaucoma, either in early or established glaucoma.

5. In established glaucoma, light minimum (L.M.) is regularly altered in two ways: first the rate of dark adaptation is retarded, and second the ultimate L.M. is raised.

6. In glaucoma suspects before there is any clinical evidence of the disease, our results show an abnormal alteration in the light minimum, manifested as a retarded adaptation rate, and as an increased ultimate threshold. In brief our results suggest that the light sense in early glaucoma is affected before there is any sustained
increase in intraocular tension, any cupping of the disc, or any visual field changes characteristic of glaucoma. The nature of this disturbance in the light-sense appears to be twofold; first a tardiness in dark adaptation, and second, in the dark-adapted eye, a dulling of sensitivity to dim light.

7. Finally we wish to stress the fact that the results which we present must only be regarded as suggestive until further research on a much larger series of cases has confirmed them."

'The nature of this disturbance in the light-sense appears to be twofold; first a tardiness in dark adaptation, and second, in the dark-adapted eye, a dulling of sensitivity to dim light.'

A useful bibliography accompanies the paper.

E.E.H.


This paper is a continuation of the work published in the Trans. Ophthal. Soc. U.K., 1925 (abstracted above) and contains a number of additional graphs confirming the previous results. Derby, Waite, and Kirk consider that they are justified in concluding that examination of the light minimum sense is of real value in making the diagnosis of early glaucoma. They further suggest that some simple apparatus, such as the glasses described by Tscherning may be found to give a perfectly satisfactory result, and will make the light minimum sense examination possible in the ordinary consulting room.

E.E.H.


In this paper Knapp records two cases of Morgagnian cataract accompanied by raised intraocular pressure. In the first case the pressure was raised before operation (T. 55 mm.), but improved under treatment with pilocarpine. During the operation, a combined extraction, the capsule gave way when grasped with the forceps. The nucleus was expressed and there did not seem to be much fluid cortex, the pupil remaining black. During convalescence the anterior chamber remained shallow and the tension low; a choroidal detachment was present. Later the tension rose to 55mm. with a deep anterior chamber and steamy cornea. After a Lagrange operation the tension remained normal but there was considerable iridocyclitis with corneal opacity. The pupil was adherent to thickened capsule attached to the wound. In the second case the tension was also raised and reduced by treatment on two occasions before the performance of an iridectomy. Later the cataract was successfully extracted with blunt capsule forceps in the capsule. Recovery was uneventful and normal vision was regained.
GLAUCOMA

In commenting on these two cases, Knapp considers that they are suggestive of a low-grade uveitis in which the increased tension is the most striking feature. He does not agree with Gifford in thinking that the liquid cortex is the toxic source of the disturbance but inclines to the opinion that the displaced nucleus must be the cause of some mechanical irritation of the ciliary processes and thus produce the glaucoma. Extraction in the capsule is undoubtedly the operation of choice in these cases, but is technically by no means easy of performance owing to the condition of the capsule.

In the ensuing discussion in which opinions on the toxicity or otherwise of the Morgagnian fluid were illustrated by a number of cases, all were agreed as to the importance of not allowing a cataract to advance to the Morgagnian condition, and as to the advisability of intracapsular extraction in such cases.

E.E.H.


(4) Hamburger points out that mydriasis and miosis in the treatment of glaucoma are held to have actions diametrically opposed to each other, and yet adrenaline by subconjunctival injection, the most powerful mydriatic known, can effect the following:

(1) Reduce the tension of a normal eye to a "soup-like" softness for hours or even for days; (2) favourably influence glaucomatous eyes, an influence which can be prolonged by repeated injections at regular intervals; (3) reduce the tension in the fellow uninjected eye, thus settling the question whether an irritation can be transferred from one eye to another; (4) that its hypotonic power can be considerably improved by a combination with eserine. This is especially useful in cases where eserine alone has proved impotent. That is to say that the most powerful mydriatic has its hypotonic power enhanced by combination with a miotic. The injections are made as follow:

A few drops of a 2 per cent. solution of holocaine are instilled with sufficient adrenaline to contract the vessels. A fold of conjunctiva is raised on the temporal side and about 0.3 c.cm. of 1:1,000 adrenaline injected. After about five minutes the pupil begins to dilate, always eccentrically, and after about fifteen minutes is dilated ad maximum, sometimes the iris almost disappears. In iritis synechiae are torn away, often with considerable pain, even when they have failed to yield to atropine and scopolamine. The mydriasis is so powerful that it persists even when the anterior chamber is opened and the aqueous escapes. In most cases of glaucoma the tension falls, sometimes after a slight preliminary
rise. In a few cases an acute glaucoma has supervened. Seven such have been described but all were cured by the use of eserine without operation. This complication can generally be avoided by instilling eserine energetically about an hour after the injection, just as one would proceed after a diagnostic instillation of homatropine, but more frequently. The softening caused by adrenaline is easily explained. It constricts the afferent small vessels, because adrenaline is the strongest stimulant for the sympathetic vaso-constrictors. The cavernous sinuses of the choroid are compressed like the squeezing of a sponge, since the massive uveal water cushion that encircles the whole eye is relatively poor in capillaries.

It is more difficult to account for the hypotonic action of eserine, since the pupil contracts, the iris vessels dilate and become engorged, and even the ciliary body is hyperaemic. This can be seen with the naked eye in an albino rabbit. The secretory action of the ciliary body is stimulated and the albumen content of the aqueous rises. We have increased supply of blood, increased metabolism, and yet the tension falls, a physiological paradox! The explanation lies in the fact that the circulation of the iris is quite distinct from that of the rest of the uvea, the former is supplied by the long, the latter by the short ciliary arteries. The iris in consequence does not share in the affections of the choroid and ciliary body, and the converse is equally true. The two circulations are antagonistic to each other, one compensates the other. Eserine causes hyperaemia of the anterior part of the eye, and as a compensation the posterior part is ischaemic. The combined effect of adrenaline and eserine is due to the fact that adrenaline constricts the capillaries of the eye and so squeezes out the choroidal water cushion, whereas eserine draws the blood that still remains in the eye forward, and smooths out the iris folds, so increasing the absorbing surface at a time when the adrenaline mydriasis is passing off.

Glaucoma is due to a variety of conditions and naturally adrenaline treatment like all others occasionally fails. Fisher calls glaucoma "ocular dropsy" and draws attention to the capability of the vitreous to become swollen, and it is possible that the acute forms of glaucoma are due to swelling of the vitreous. This may explain the fact that adrenaline treatment is ineffective in acute glaucoma. Finally, the indications for treatment in glaucoma are: first miotics, then a combination of eserine drops with injections of adrenaline, and finally, only when this fails, operation. It is necessary in chronic glaucoma to repeat the injections at about three-weekly intervals.

T. Harrison Butler.

Agnantis describes in detail, with a number of charts of fields of vision, a series of eighteen cases of secondary glaucoma. He refers to the book by F. Lagrange “Glaucome et Hypotonie” in which glaucoma is divided into “glaucome vrai”—referring to primary glaucoma—and “glaucome faux”—secondary glaucoma, and in which Lagrange denies the existence of typical field contraction in the second group (secondary glaucoma).

As a result of the cases described, he claims that in every case in which for an unknown reason (i.e., primary glaucoma) or for a known reason (i.e., secondary glaucoma) the ocular tension is raised for a sufficient length of time, contraction of the visual field results. This is due mainly to ischaemia of the peripheral retina from pressure. Failure of this general statement occurs occasionally in both primary and secondary glaucoma when the rise of tension is only temporary and occasional. The nasal field suffers particularly owing to (1) the greater length of the vessels to the part of the retina corresponding with this field; (2) the early affection of the temporal half of the disc by cupping. Agnantis concludes by pointing out that nine of the eighteen cases show conclusively that nasal loss of field is just as typical in chronic secondary glaucoma as in primary glaucoma. Is it not possible, he says, that primary glaucoma owes its development to some lesion as yet not recognized, and that it is secondary to such a lesion, so far unknown, but perhaps to be discovered in the future?

Humphrey Neame.

Meesmann, A. (Berlin).—Contributions to the physical chemistry of the modification of the intraocular fluid under normal and pathological conditions, particularly in glaucoma. (Beiträge zur physikalischen Chemie der intraokularen Flüssigkeitswechsels unter normalen und pathologischen Verhältnissen insbesondere beim Glaukom.) Arch. f. Augenheilk., Bd. XCVII, December, 1925.

As the details of this research are too long and complicated to be abstracted, the interested reader is referred to the original for them. The following is Meesmann’s summary:

“(1) Glaucoma is not to be explained by differences in the osmotic pressure of blood. The membranes separating blood and aqueous are wholly permeable to crystalloids. Hence differences in the crystalloid osmotic concentration of blood cannot lead to prolonged change of tension inside the eye. But in regard to colloid osmotic pressure there is such a possibility. Nevertheless, according to
Serr's researches, such deviations from the normal of colloid pressure do not occur in the blood of the glaucoma patient. Neither does the view that osmotic pressure accounts for variations of intraocular tension derive any support from Donnan's formula for osmotic equilibrium.

(2) The significance of the actual reaction of the aqueous to the intraocular tension is again stressed and confirmed by new experiments. If the alkalinity of the aqueous rises above the normal there is corresponding increase of tension, and fall of alkalinity below normal regularly leads to fall of tension. The physico-chemical processes underlying these relations are the swelling and detumescence of the vitreous and the lens.

(3) As a rule a corresponding change in the blood could be shown to be the cause of the actual change of reaction of the aqueous, e.g., an acidosis of the blood in diabetic coma and in the later months of pregnancy, an alkalosis in primary glaucoma. The alkalosis is a permanent condition in chronic glaucoma whereas in acute glaucoma it appears to be transient. On the contrary secondary glaucoma is wholly dependent on local changes.

(4) The essence of primary glaucoma is an increased swelling of the contents of the eye, caused by alkalosis of the blood and consequently of the aqueous. Hence in grave diabetes and the later months of gestation primary glaucoma is improbable, a surmise which is well supported by clinical facts. Similarly the relative infrequency of primary glaucoma in high myopes is intelligible on account of the degeneration of the vitreous and its consequent diminished capacity to swell.

(5) The individual symptoms of primary glaucoma and the effectiveness and failure of different therapeutic measures are easily explained by the alkalosis. The mydriasis—hitherto not satisfactorily explained by any theory—is, according to the researches of Wieland and Schoen, also to be regarded as a consequence of alkalosis of the blood, i.e., a paralysis of the miotic centre through the decreased CO₂-tension of the blood.

D. V. Giri.


(7) According to Bailliart and Magitot the tension of the eye is found to vary normally with the systemic blood pressure. In a large proportion of cases of chronic glaucoma, the general blood pressure is raised. In certain cardio-renal cases, however, the ocular tension is low in spite of enormously high blood pressure. In such cases, the sclerosis of small vessels with some degree of added arterial spasm reduces the volume of blood in the eye and so the tension. The circulatory trouble in glaucoma is one rather
of the exit than the entry of blood into the eye. In the normal eye the blood pressure in the central artery of the retina and its main branches is as follows: minimum 30 to 35 mm. of mercury, maximum 70 mm. The venous pressure is approximately equal to the ocular tension. Seidel is quoted as finding that the pressure in the anterior ciliary artery perforating branches is equal to 30 to 35 mm. minimum and 55 to 75 mm. maximum. (Bailliart and Magitot find the choroidal arterial pressure equal to the retinal arterial pressure, by observation of the circulation in an atrophic patch in the choroid.) Seidel in a similar manner found the pressure in the anterior ciliary vein equal to 10 to 14 mm. of mercury. Bailliart and Magitot consider that the choroidal venous pressure is about equal to the intraocular pressure, for, if the choroidal venous pressure were higher than this in the anterior ciliary perforating veins of the eye, these would become distended and so automatically lower the pressure. The walls of the intraocular veins are supported by the intraocular tension, whereas the anterior ciliary veins outside the eye are not supported. There is a corresponding pressure in the arteries of the choroid and in the retina.

In raised intraocular tension the minimal retinal arterial pressure is almost always raised. At the same time, the venous retinal pressure must rise to avoid obliteration, and therefore probably equals approximately the tension of the eye, as in normal conditions. In glaucoma the anterior ciliary and subconjunctival veins show raised pressure by their frequent marked dilatation and by their rapid filling from the limbus peripherally, after compression by the eyelid. Within the eye the raised tension prevents marked dilatation of veins which have raised blood pressure. Further, the dilatation of veins on the iris surface in long-standing glaucoma, and especially in cases with central retinal vein thrombosis, signifies raised intravenous pressure in the ciliary vein.

To summarize, in raised intraocular tension, there is a moderate increase of arterial blood pressure and often a marked increase of venous blood pressure. The ocular tension is not affected by obstruction of the central artery of the retina, nor, at least at first, by obstruction of the central vein. Is this true of the choroid? This is considered the kernel of the situation. It is probable that obstruction to the choroidal circulation has a definite effect, as the volume of the choroidal circulation is infinitely greater than that of the retina. It is possible that the rise in arterial pressure is compensatory and secondary to slowing of the venous circulation. The following observations are tabulated:

(a) Obstruction of the vortex veins causes marked hypertension, as also does obstruction of the anterior ciliary veins.
In animals bled to death, perfusion restores the tension of the eye to normal (Weiss, C.).

Carotid compression reduces the tension.

The introduction of adrenaline into the retrobulbar tissues, and to a less degree into the subconjunctival tissues, reduces the tension (Fromaget).

On the other hand, irritants, sodium chloride, mercuric chloride, cause a rise of tension owing to the resulting vasodilatation (Wessely).

Amyl nitrite by causing capillary dilatation also raises the tension (Wessely, Leplat, Bailliart, and Bollack).

Contusion of the eye by resulting hyperaemia of the choroid, also causes a rise in tension, not only in the injured eye, but also in the other (Leplat).

Section of sympathetic vasomotor nerves raises the tension, and stimulation of these nerves lowers the tension, concurrently with changes in the state of the vessels (Bailliart and Magitot).

Several cases are reported in detail of primary glaucoma in which pathological examination of the eye was possible during the condition of glaucoma owing to the supervision of death from some intercurrent cause, such as pneumonia or accident. Pathological examination showed various signs of inflammation in the eye, with perivascular inflammation and sclerosis of episcleral veins and vessels of iris and choroid. Reference is made to obstruction by experimental ligature of the vortex veins.

Conclusion.—Glaucoma may, therefore, be caused by venous obstruction from phlebitis or by overgrowth of the vein wall occurring particularly in the ciliary circle. The result of this venous obstruction is resistance to the emptying of the capillaries. This is of greater importance for the choroid than for the retina as the retinal circulation is a mere fraction in volume compared with that of the choroid. Vaso-dilatation follows, presumably owing to nerve impulses, in order to overcome the venous obstacle. As a result of this, the capillary reservoir dilates and ocular hypertension supervenes. Similar hypertension may occur in general arterio-sclerosis with general rise of blood pressure. When the ocular tension becomes equal to the arterial diastolic pressure, arterial pulsation appears in the retina. The observation of arterial pulsation is of practical importance. When the ocular tension becomes equal to the diastolic arterial pressure of the central artery, an indication for operation is obtained. The central artery blood pressure in the normal eye is approximately equal to half the blood pressure in the humeral artery.

Humphrey Neame.
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(8) In the case recorded by Gros glaucoma resulted from fine capsulo-corneal adhesions and when these were divided the tension returned to normal. The eye had been injured by the explosion of a detonator, lenses wounded, cataract successfully extracted in the eye under consideration, unsuccessfully in the other.

ERNEST THOMSON.

II.—DISEASE OF RETINA

(1) Shoji, Y. (Okoyama, Japan).—Congenital unilateral non-syphilitic retinitis pigmentosa. (Rétinite pigmentaire congénitale unilatérale non-syphilitique.) Arch. d'Ophtal., July, 1926.

(1) Retinitis pigmentosa congenitalis is almost invariably bilateral, and although certain observers have refused to entertain the idea of uniocular examples of the disease a number of cases have been reported, to which reference is made in this paper.

Shoji has now published complete notes of a case in which the evidence appears to be unequivocal.

The patient, a healthy Japanese youth, came under observation when aged 13 years. For about a year he had noticed that in the dusk the vision of the left eye was feeble than that of his right: Central vision, however, was R. 0.2; L. 0.8. Fields of vision: R. normal; L. concentrically narrowed for white, red, and green. Ophthalmoscopic examination: R. fundus normal; L. numerous small stellate pigment deposits in the retina peripherally. Charts of the fields and drawings of the right and left fundi at this date are given. The Wassermann reaction was negative. Family history: consanguinity in the grandparents. Parents healthy, no miscarriages.

After the lapse of ten years during which no treatment had been adopted, the patient reported increased defect of his left eye, and loss of distant vision. Examination at this date showed central vision in the R. = 0.5 with -2D. sph.; in the L. = 0.9 with -0.75D. sph.

The field of the R. normal for white and colours; the field of the L. concentrically contracted to less than 10° all round. Lightsense tested with Foerster's photometer (after 15 mins. dark adaptation) R. 2 mm.; L. 22 mm. With Nagel's adaptometer dark adaptation was normal in the right, very feeble in the left, as shown by curves in diagram. Colour-sense in central field was normal in R. and L.
Ophthalmoscopically: The right fundus was healthy; the left showed diminution of the retinal arteries; retina uniformly dull; much black stellate pigment deposit beyond the immediate neighbourhood of the disc and macula. On the anterior lens capsule there were about thirty small brown dots thought to be persistent remains of the pupillary membrane.

The Wassermann reaction was negative throughout.

J. B. Lawford.


(2) In this additional contribution concerning senile exudative retinitis Coppez and Danis devote especial attention to the relationship between this disorder and circinate retinitis, to which they drew attention in 1923. Several observers have written on the subject since that date. Batten (Trans. Ophthal. Soc., U.K., 1923) published a series of drawings of macular lesions in arteriosclerosis, some of which Coppez and Danis believe to be examples of senile macular exudative retinitis. Two cases showed lesions characteristic of circinate retinitis. Feingold (Trans. Amer. Ophthal. Soc., 1924) reported seven cases under the title proposed by the Belgian authors in 1922. He holds that senile exudative retinitis should be clearly differentiated from retinitis circinata and from angioid streaks in the retina. Junius and Kuhn in an illustrated monograph on cases designated by them “disciform degeneration of the macula lutea” report ten cases, three of which presented the signs of circinate retinitis.

Such statistics as exist indicate that circinate retinitis is a rare disease. Fuchs reports eleven cases in 70,000 patients; de Wecker fifteen in 150,000; Silex three in 23,000. It occurs in persons over 50 years of age and is characterized by slow and progressive failure of sight. Ophthalmoscopically, there are two chief signs: (1) an elliptical white corona, its long axis horizontal, encircling the macular region; it is composed of small rounded white spots posterior to the vessels, and isolated or confluent. The corona is usually broken on the temporal side. (2) A macular lesion which is, however, not constant; it consists of a zone of greyish or yellowish discoloration occupying the macular area; it varies considerably in size and in the definition of its borders, and generally shows central haemorrhage or pigmentation. It does not reach the corona. The retinal vessels seldom show any alteration but in some instances are tortuous. Anatomical examination of circinate retinitis has been made once only, by von Ammon. He reported that the white deposits in the retina
were external to the inner granule layer and that where the exudation was situated the rods and cones and the outer granules had disappeared; the other layers of the retina were thickened, as were Müller’s fibres.

de Wecker and von Ammon believe that the white deposits are the remnants of former haemorrhage; Fuchs disagrees and considers that they are analogous to the exudation in albuminuric retinal lesions and are composed of transuded albumen.

There are numerous analogies between senile exudative retinitis and circinate retinitis. Both arise without appreciable cause in elderly people; their evolution is slow and progressive; the vascular lesions are discrete; haemorrhages are frequent; the seat of the lesions is the posterior pole of the eye.

In this paper Coppez and Danis publish notes, with coloured plates, of two cases of senile exudative macular retinitis accompanied by the usual appearances of circinate retinitis. One of these examples was seen on one occasion only; the other has been under observation for ten years, and the drawings show the fundus changes at an interval of two and a half years. Examination of the latter case by means of the binocular ophthalmoscope and in red-free light showed that the haemorrhages are situated in the external layers of the retina or in the choroido-retinal space; the white spots are internal to the haemorrhages but are external to the retinal vessels; in the area of the macular exudation there are small deep blood extravasations in addition to pigmented spots; the nerve fibres are probably atrophied as their fibrillar arrangement is invisible in red-free light.

J. B. Lawford.

(3) Velter, E., and Blum, J. (Paris).—A case of ocular tuberculosis originating in the retina.) (Sur un cas de tuberculose oculaire à point de départ rétinien). Arch. d’Ophtal., March, 1926.

The case here reported was under clinical observation for a period of three or four months: the eye was then excised and submitted to histological examination. Although the evidence is not wholly without flaw there seems good reason for Velter and Blum’s contention that the tuberculous lesion began in the retina. Accompanying the report are to be found one drawing of the ophthalmoscopic changes and two micro-photographs of the pathological appearances.

The patient, a male, aged 36 years, had pulmonary and other lesions considered tuberculous in nature. When first seen the left eye exhibited no signs of disease in the anterior part; the media were clear and the fundus easily illuminated. The papilla was hyperaemic, an elongated patch beginning close to the uninvolved
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at the posterior pole of the eye. In the portion

of the choroid thus altered no trace of follicular tuberculous lesions

was found; it was covered by a degenerating fibrinous layer containing

scattered giant cells. In the anterior segment of the globe were recent lesions of tuberculous character in the choroid. Neither the ciliary processes nor the iris were obviously tuberculous: the latter was very vascular and thickened.

In cases of ocular tuberculosis in which the initial lesion is choroidal, invasion of the retina is frequent and often considerable, but the concomitant lesions of the uveal tract prove the primary seat of disease to be in that tissue. In the present case the order appears to be reversed; the retina shows a massive invasion by tubercle while the choroidal lesions are those of simple inflammation except in the anterior part where the lesions are recent but characteristically tuberculous.

J. B. Lawford.


(4) Lavrand and Desprets relate in detail the case of a woman, aged 35 years, who suffered from asthenopia, headache, and loss of vision in one eye, in whose fundus were found choroidal-retinal pigmented lesions of various ages. The patient was in various ways not in a healthy condition, but there was no direct...
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evidence as to the aetiology. Basing themselves on previous clinical experience and in spite of the absence of symptoms of frontal, maxillary, or sphenoidal sinusitis it was decided to explore the anterior ethmoidal cells. A hitherto undetected osteitis was found on both sides. Active treatment of this resulted in cessation of the headaches and the asthenopia. Visual acuity rose from barely 1/20th to 1/4th or 1/3rd.

ERNEST THOMSON.


(5) Harry and Jonas Friedenwald bring forward some interesting evidence in support of the theory that keratic precipitates may, in some cases, be derived from a focus of choroidal infection, without there being an iritis or cyclitis. Moreover, some of the cells passing forwards to the keratic precipitates may be deposited on other structures en route. Thus we have (1) the formation of pupillary globules; (2) globules hanging on to the fibres of the vitreous; (3) cells on the surface of the ciliary body without underlying inflammation; and (4) perivascular stripes in the retina. The evidence comprises the cases in which there were globular masses on the margin of the pupil. In four of these a patch of exudative choroiditis was found, in five there were extensive vitreous opacities, and the last was a case of iritis. In one case of acute exudative central choroiditis the eye was removed on account of the possibility of the presence of a growth and a full pathological examination was made. The presence of the choroiditis was confirmed and there was necrosis of the overlying retina. The interesting feature, however, was the occurrence of small lymphocytes, large cells with oval eccentric nuclei, and vacuolated protoplasm in positions far removed from the original focus of disease. Many of these cells were laden with the products of phagocytosis. The authors therefore consider that some of them at any rate are cells in process of being carried away by the lymph currents and therefore that the presence of keratic precipitates and of globular masses in the iris may be associated with a lesion much further back than the ciliary body.

F. A. WILLIAMSON-NOBLE.


(6) Bettremieux refers to a communication by Weekers in which the latter had pointed out, apparently as a new point, that
in detachment of the retina there is sometimes a rise of tension at the beginning. Bettremieux states that it was recognized some time ago by himself, Boucheron, and Dransart that in detachment there are two periods of ocular tension, one at the commencement when the tension is raised, and a later period of lowered tension. The early hypertension is sometimes appreciable by the finger but frequently is only manifested in the course of an iridectomy by the occurrence of a jet of liquid analogous to that in a case of moderate glaucoma. These slight increases of tension may occur several times in the early days of a retinal detachment when fresh exudations are occurring. According to Boucheron the indication is to operate early when there is a certain degree of hypertonus, and the author has come to the same conclusion. But Bettremieux prefers his own operation to iridectomy. This operation is that of non-perforating pericorneal sclerectomy. The reader will find abstracts in *The Ophthalmoscope* for 1908, pp. 400 and 818, and probably elsewhere either in that journal or its successor, dealing with the Bettremieux operation.

**Ernest Thomson.**

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### III—MISCELLANEOUS

(1) Coppez, Henri (Brussels).—Examination of the aqueous humour for the presence of iron, in relation to the diagnosis of intraocular foreign bodies. (Sur la recherche du fer dans l'humeur aqueuse considérée au point de vue du diagnostic des corps étrangers intra-oculaires.) *Arch. d'Ophthal.*, October, 1926.

(1) Despite the various aids to diagnosis commonly employed (giant magnet, radiography, etc.), the nature and even the presence of minute foreign bodies within the eyeball occasionally escape detection. In some instances, five of which Coppez quotes from published records, late evidence of the foreign body is forthcoming in the development of siderosis.

It is, for several reasons, desirable to obtain positive evidence of an intraocular foreign body, metallic in nature, in cases in which the means cited above have failed: among them is the fact that the majority of such cases being industrial accidents, lack of definite information might prevent the artisan obtaining compensation to which he was entitled.

In 1921, Coppez had recourse to chemical analysis of the aqueous, and reported his first case, at the time, to the Belgian Ophthalmological Society. In the present communication he gives detailed notes of the original and of three recent cases. In all four the presence of iron in the aqueous was indubitably shown by chemical analysis, after a negative result by other methods of examination.
In one case in which the eyeball had to be removed for secondary glaucoma a splinter of iron was found embedded in the sclera at the posterior pole.

The procedure to be followed should be studied in the Coppez paper: meticulous care is necessary to avoid all possibility of error and to exclude that which might arise from the presence of haemoglobin in the aqueous. The analysis should be entrusted to an expert analytical chemist.

The subject of this communication is important and the author's facts and statements are interesting: in his concluding sentences he says:—"I feel able to state from these observations that the examination of the aqueous humour for the presence of iron is a good diagnostic procedure. It is especially noteworthy by reason of its value at a date so distant from the accident that the usual methods have become uncertain or completely ineffective. I advise its adoption therefore in all cases of traumatism in which the abnormal development of lesions, or the appearance of signs of siderosis suggest the presence of a foreign body previously unsuspected."

J. B. Lawford.

(2) van Lint (Brussels).—Palpebral Akinesia. (L'Akinésie palpébrale.) Arch. d'Ophtal., December, 1926.

(2) In 1914 van Lint published an article in Ann. d'Ocul., Vol. CLI, 1914, on "Transient palpebral paralysis during operation for cataract," in which he stressed the danger from spasmodic contraction of the lids during extraction of cataract and showed how this danger could be obviated by the injection of novocaine-adrenaline in the course of the facial nerve fibres before they penetrate the orbicularis. While control of the lids is helpful in extracapsular extraction it is almost indispensable in intracapsular extraction in which the larger wound facilitates loss of vitreous.

Since that time palpebral akinesia (the term given by Rochat) has been widely adopted by ophthalmic surgeons. But though the majority employ this method there are still some who have not adopted it for various reasons. The present communication is specially addressed to these latter in the hope of convincing them that with an adequate technique happy results are constant. The reviewer believes that comparatively few surgeons in this country have adopted this practice and for that reason gives van Lint's directions almost in full.

van Lint uses a hypodermic syringe holding 4 c.c. with a strong steel needle 3 cm. in length. The solution injected (4 c.c.) is novocaine 2 per cent. with 2 to 3 drops of adrenaline added. Anaesthesia of the surface of the eyeball is begun and immediately after this the injections are made, i.e., about 15 minutes before operative measures.
In order that the injection may be successful it is necessary to prevent motor impulses from the centres reaching the orbicularis, by impregnating the terminal filaments of the facial nerve with novocaine. These filaments reach the muscle, grouped on the bone of the external and inferior borders of the orbit. The injection must therefore be made in this position and to the requisite depth. Iodine is applied to the skin: the needle is passed into the skin perpendicularly and down to the bone, at the intersection of a horizontal line, passing 0.5 cm. below the inferior edge of the orbit, and a vertical line passing 0.5 cm. outside the external margin of the orbit. One drop is injected: the needle is partially withdrawn and turned in the direction of the nose along the horizontal line already designated, and at each half centimetre point an injection is made, always feeling the point of the needle on the bone, until the inner end of the lower lid is reached. About 1.5 c.c. of the solution is injected during this procedure.

For the injection at the lower half of the temporal border of the orbit, the needle is not completely withdrawn from the first site but its point is directed upwards near the bone and half a cm. from the orbital margin. One c.c. is injected here by the same intermittent manoeuvre as before. The needle is then withdrawn. It is re-inserted from above downwards at a point on the frontal ridge at its intersection with a vertical line along the outer margin of the orbit. The remaining 1.5 c.c. of the solution is injected here. This is in brief the technique recommended, and the author says: "the results are constant, sometimes greater, sometimes less, but always sufficient to render the patient incapable of closing his lids forcibly. If the injections are deep and not excessive in amount no oedema of the lids is induced.

van Lint has advocated and practised this method consistently and is convinced of its great utility. He says, epigrammatically, "Celui qui opère la cataracte sans l'akinésie palpébrale a la sensation de faire de l'acrobatie, de marcher en équilibre sur une corde tendue. Celui qui fait l'akinésie marche d'un pas assuré sur la terre ferme."

J. B. Lawford.

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BOOK NOTICES

Transactions of the American Ophthalmological Society.

The sixty-second annual meeting of the American Ophthalmological Society was held at Hot Springs, Va., in June, 1926; the present volume, in addition to the papers read and the discussion thereon, includes some features of more than domestic interest in