ABSTRACTS

I.—OPHTHALMIC ABIOTROPHIES


(1) As Sir James Paget wrote many years ago (Lectures on Surgical Pathology, 4th edition, 1896, pp. 70 and 72): "It is natural to become feeble and infirm, to wither and shrivel, to have dry, dusky, wrinkled skins, and greasy, brittle bones, to have weak fatty hearts, blackened, inelastic lungs, and dusky thin stomachs, and to have every function of life discharged feebly, and as it were waryly; and then, with powers gradually decreasing to come to a time when all the functions of bodily life ceasing to be discharged, death, without pain or distress, ensues. The changes of natural degeneration in advanced life have a direct importance in all pathology, because they may guide us to the interpretation of many similar anomalies, which, when they occur in earlier life, we are apt to call diseases, but which are only premature degenerations, and are to be considered, therefore, as methods of atrophy—as defects, rather than as perversions, of the nutritive process—or as diseases only in consideration of the time occurrence." This is an anticipation of Sir William Gowers's theory of "Abiotrophy" or degeneration of tissues due to defective vitality. Collins classes as hereditary degenerations hereditary post-natal cataract, hereditary ocular palsies, retinitis pigmentosa, symmetric familial pigmentary macular degeneration, family amaurotic idiocy, Leber's optic atrophy, Doyne's family choroiditis, and lattice-like and nodular degeneration of the cornea. These widely different diseases are hereditary in the sense that they may be met with in several siblings of the same generation, and all, with the exception of symmetrical macular pigmentary degeneration and amaurotic family idiocy, in several different generations. The lesions are always bilateral. In none so far examined pathologically do the findings suggest inflammation, but are such as indicate degeneration in a certain definite group of cells. The occurrence of the degenerations in several successive generations of the same family renders it impossible to attribute them entirely to the absorption of any exogenous toxic substance or to the absence of any exogenous essential nutrient material, such as a vitamine. There seems to be no substance of one of the endocrine glands, upon which the structures involved in these diseases are dependent for their vitality; neither do we yet know of any toxic material developed endogenously either by bacteria or as the outcome of some faulty metabolic process, capable of poisoning the cells picked out
by these diseases. Each of the diseases above-named is taken up successively by Collins and an attempt made from such pathological evidence as is available, to determine the structure in which the premature loss of vitality occurs, and the course and sequence of the clinical symptoms which follow.

S.S.


(2) The cases reported by McHenry were seen at the Polyclinic Hospital, New York, between September, 1915, and December, 1916. The observation of so many cases of a rare disease in a period of 16 months, leads the author to suggest that many examples of this malady are unreported, or are not diagnosed during the brief lifetime of the patients.

All nine cases occurred in the children of Hebrew parents; seven were female, one was male; in one (Case VII) the sex is given as female, but in the notes the child is spoken of indifferently as “he” or “she.”

The clinical notes are those of typical examples of “Tay-Sachs” disease. In each case the Wassermann reaction was negative: increased intracranial pressure was present in the 8 cases in which lumbar puncture was performed.

Photographs of three of the children before and after the onset of symptoms are given, and a useful bibliography is appended.

J. B. Lawford.

II.—COLOUR VISION

(1) Schanz, Dr. (Dresden).—Colour vision. (Das Sehen der Farben.) *Zeitsch. f. Augenheilk.*, December, 1921.

(1) Schanz points out that up to the present the rods and cones have been regarded as the light sensitive elements of the retina, but this cannot be the case, for light can only produce an effect when it is absorbed. The rods and cones cannot absorb light; the absorption takes place in the pigment layer of the retina, and we are justified in assuming that electrons are expelled from this pigment by the absorbed light. The rods and cones act like antennae and collecting these electrons conduct them to the central organ. Light of different wave lengths corresponds to electrons of varying speed. This explains the perception of pure colour as seen in the spectrum. Nature affords practically no pure colours. Our world is seen as a colour mixture, and there are countless colours which our eye cannot differentiate from spectral colours. The ear can analyse a complex of tone, but the eye is powerless to analyse a
COLOUR VISION

431

colour mixture. Colour mixture occurs when electrons of varying velocity are expelled simultaneously from the pigment epithelium and arrive at the central organ. There they can produce the same impression as pure colour which is caused by electrons of one velocity only. To explain this phenomenon we assume that electrons which travel together from the retina to the brain mutually influence each other as regards velocity; those of high velocity retard the slow and the slow accelerate the rapid. Mutual adjustment takes place and there will be a spot in the path where the speeds are equal. If this spot corresponds with the central organ then we shall get the sensation which would be caused by an electron speed which corresponds to that attained at the end of the journey by the combined electron complex. In each case the electrons reach the brain with the same velocity and elicit the same sensation. There is no other theory which explains colour perception in such a simple way. If the difference in wave length is too great the electron speed cannot obtain equilibrium at the central organ and no colour sensation is perceived. In this case we obtain the impression of white. The difference between the speeds of complementary colours falls into this category, and they are seen as white.

Schanz explains Purkinje’s phenomenon by assuming that the electrons from red light have less energy than those from blue and in consequence when the intensity falls red light fails to stimulate and appears black.

He assumes that in congenital colour blindness the pigment epithelium is not absolutely black and that in consequence colours of certain wave lengths are not absorbed.

Acquired colour blindness is due to lesions in the tracts which inhibit the passage of electrons. Those with least energy, namely, the red are first affected.

Schanz’ theory explains colour vision on the basis of physics and chemistry and does not, like those propounded by Helmholtz and Hering, fall back upon vitalism.

T. HARRISON BUTLER.


Schiøtz gives several pedigrees of colour blind families both of his own and of other observers with analytical notes. In all the cases of colour blind women that he was able to trace the father was found to be colour blind with one exception. In this case the colour blind woman was the only child and her mother had died when giving birth to her. The father was a Swede and the only member of his family living in Norway; he knew nothing of his wife’s family. Schiøtz thinks that the legitimacy of this daughter is very doubtful.
He concludes by stating that, as a result of his researches, he considers that there can be no doubt that congenital red-green blindness is always and without exception a recessive-sex-associated (Rezessiv-geschlechtsgebundene) hereditary peculiarity and that no exception to this rigid rule has yet been observed.

E.E.H.

III.—DISLOCATION OF LENS INTO ANTERIOR CHAMBER

Roche, C. (Marseilles).—Bilateral dislocation of lens into anterior chamber. (Luxation bilatérale du cristallin dans la chambre antérieure.) Arch. d'Ophtal., Sept., 1921.

Roche records a case of this rare affection in a child of five years and six months. The child was badly developed, both mentally and physically; large head, high palatine arch and lamellar teeth. Five days before consulting Roche the parents had noticed a sudden appearance of a bright spot in both eyes. This was evidently the dislocated lens. Roche advised operation, but this was not done for six months. The right eye was then dealt with by fixing the lens with a needle and tearing it up with a cystotome. Six weeks later the soft lens matter which had become completely opaque was evacuated. Healing was uneventful, and the parents were advised to bring the patient up the following month in order to have the left eye operated on. They did not do so for three months as the patient had been seriously ill with bronchitis. Eight days before returning the child had complained of severe pain and headache. When Roche saw the child the left eye showed all the symptoms of a very acute glaucoma. The lens was still in the anterior chamber and was opaque. There was no perception of light. The lens was removed with the scoop with much vitreous loss. The eye became soft and shrunk. The right eye retained good vision with a +10 D. lens, but, owing to the child's poor mental development, no accurate test of the visual acuity could be made. The iris of this eye showed a zone of atrophy midway between the greater and lesser circles which, in places, enlarged to what resembled a definite cicatrix. Roche inclines to the view that there had been some intra-uterine uveal affection which had affected the zonule. In spite of the fact that there was no history of parental syphilis and that the Wassermann blood test in the patient was negative, he considers that the general condition of the child pointed to that disease as the source of the trouble. The great importance of operation as soon as the condition is recognized is obvious.

E. E. H.
SECRETION OF THE AQUEOUS HUMOUR

IV.—LOCAL ANAESTHESIA


The method adopted by Liebermann, in Budapest, is the following:—A 2% solution of novocain, with 0.8% of sodium chloride, and 0.4% of potassium sulphate is made, and just before it is used 8 drops of a 1% solution of adrenalin is added to 10 c.c.m. of the novocain solution. Three injections are given through the skin, not through the conjunctiva. Before the needle is inserted a small intracutaneous injection is given with a fine needle. The first insertion is made just under the outer canthus close to the margin of the orbit with a needle exactly 4 c.m. long; this is driven in to its full extent in the direction advocated by Löwenstein in order to reach the ciliary ganglion. The second situation is that recommended by Braun close to the superior orbital margin, and a finger’s breadth above the inner canthus. For about 2 c.c.m. a sagittal direction is chosen, then the canula is directed more downwards and to the temporal side till the full 4 c.m. is reached. The third injection follows at a spot where the inner third of the orbital margin joins the middle third. It is given through the skin of the lower lid, and the direction of the needle is upwards and towards the temple. 1.5 to 2 c.c.m. of the solution is used each time or about 4.5 to 6 in all. The needle should be 0.6 to 0.7 mm. thick. After from about 20 to 30 minutes a painless enucleation can be performed. Liebermann states that it is a perfectly satisfactory method even with children of from 1½ to 2 years old. In the case of infants the needle must be pushed in for about 3 instead of 4 c.c.m.

T. Harrison Butler.

V.—SECRETION OF THE AQUEOUS HUMOUR

Hagen, Sigurd (Christiania)—The aqueous humour and its secretion in the human eye. (Om kammervandet og dets ansondring i det menneskelige øie.) Norsk Mag. f. Lægevidensk, 1921.

It has long been known that in the animal eye there is found a profuse secretion of fluid from the ciliary body, when the latter is influenced by irritants which cause hyperaemia of the ciliary vessels. The secreted fluid differs from the normal aqueous humour in that it is highly charged with albumen and fibrin. There are two special irritants which are generally employed and
are very effective: sub-conjunctival injections of hypertonic salt solution and evacuation of aqueous humour. Wessely showed, that after sub-conjunctival injection of salt solution in the eye of the rabbit, as a consequence of the activity of the ciliary body, there occurs an enlargement of the intraocular content, distinguished by an increase in intraocular pressure. By means of his manometer Wessely discovered that a few minutes after the injection the intraocular pressure was increased to a considerable degree; yet the increase was of quite short duration, and went again down below normal during the first hour after the injection.

Wessely believed that the effect on the human eye was essentially the same as on that of a rabbit.

The present author made the same experiment on the human eye and found that it reacts in quite a different way from the rabbit’s eye. After sub-conjunctival injection of a 5 or 10 per cent. strength salt solution he carried out a series of measurements of the intraocular pressure by means of Schiötz’s tonometer. As in the rabbit’s eye, there was found an increase in the tension, although very slow, so that the maximum was not reached until after one hour. However, it was then sustained for several hours. The author supposes that the increase in the intraocular pressure in the human eye also is caused by an increased intraocular content as the consequence of a secretion in the interior of the eyeball; but this secretion is less profuse, and proceeds much more slowly, but on the other hand is much more protracted.

The most effective irritant for the rabbit’s eye is evacuation of aqueous humour. The anterior chamber fills after a few minutes; but the newly formed aqueous humour differs from the normal in that it contains a considerable quantity of albumen and fibrin and therefore coagulates spontaneously.

It has been definitely proved that the newly formed aqueous humour in the rabbit’s eye is secreted from the ciliary body. By examining the intraocular pressure in the rabbit’s eye after evacuation of aqueous humour, there will be found a curve, which quite corresponds to that after sub-conjunctival injection of salt solution, which proves that the immediate new formation of aqueous humour in the rabbit’s eye proceeds from the blood-vessels.

It is generally accepted, as will appear from all the important text-books on Ophthalmology, that the new formation of aqueous humour in the human eye proceeds in the same way as in the animal eye. The author believes he has shown by his experiments that this is not the case. The newly formed aqueous humour in the human eye does not contain more albumen than the normal aqueous nor more fibrin and does not coagulate spontaneously.

By measuring the intraocular pressure in a physiologically normal human eye which had to be enucleated because of a small
intraocular tumor, it was shown that though the anterior chamber had filled itself after four minutes, the intraocular pressure was not increased to the normal level before 1 1/2 hours after the evacuation of the chamber. So long is the time taken for the ciliary body in the human eye to replace the evacuated aqueous humour.

From these experiments it may be concluded that in the human eye the immediate re-formation of the aqueous humour takes place from the vitreous body by filtration through the Zonula of Zinn, but the secretion from the blood-vessels (ciliary body) proceeds very slowly in the same way after evacuation of aqueous humour, as after sub-conjunctival injection of salt solution.

S. S.

VI.—AFFECTIONS OF THE OPTIC NERVE


(1) The chief features of this condition are described by Rönne as follows:—

1. The onset, subjective symptoms and course are practically identical with those of the more severe forms of retrobulbar neuritis.

2. The changes in the fields of vision are most frequently characterized by hemiopic (often bitemporal) defects with central scotoma.

3. The field defect has often a peculiar wandering character. This is very typical and is probably not present in any other condition. In some cases it is absent and is replaced by a significant but irregular change in the form of the visual field as may occur in gummatous basal meningitis.

4. Ophthalmoscopically papilloedema or optic neuritis is often present together with the other clinical signs of retrobulbar neuritis.

Eleven cases are described, five of which are from the literature, and numerous charts of the fields are given showing the characteristic changes. In two cases a microscopical examination of the optic nerves, chiasma and tracts was made. Inflammatory foci were found resembling those present in disseminated sclerosis. These conditions are illustrated by five excellent plates, one of which is coloured.
Rönné believes that these cases may be regarded as belonging to the category of "acute optic neuritis with myelitis," although spinal symptoms are by no means always present.

With regard to the localization of the pathological process in the chiasma, he expresses himself as follows:—"1. The affection of the optic nerves, which is often associated with myelitis and acute multiple sclerosis is a retrobulbar neuritis, without regard to the frequently accompanying optic neuritis or papilloedema. The ophthalmoscopically visible papilloedema is secondary to the process in the more proximal parts of the visual path. The affection is localized in the optic nerves, and especially in the chiasma; the latter position often strongly influences the field of vision and the course of the disease. 2. A similar retrobulbar neuritis with predominating localization in the chiasma may occur without accompanying spinal symptoms."

One of the cases (No. 6) had an aneurysm of the anterior cerebral artery which was interfering with the chiasma, and at the same time a glioma cerebri in the left frontal lobe. Rönné holds that this tumour rather than the aneurysm was the cause of the retrobulbar neuritis, and that the papilloedema which was present was secondary to the inflammatory condition in the nerve, though the connection between the retrobulbar neuritis and the tumour could not be explained. While it does not carry us very far, this paper is a step on the way towards the solution of the causation of retrobulbar neuritis. It is of interest to note that visual symptoms of the type described are often produced by hypophyseal tumours and other conditions.

H. M. TRAQUAIR.


(2) Hegner gives details of three cases of tangential occipital bullet wounds. In all three the bone was slightly splintered, but the brain tissue itself was not traversed. The first case presented a small paracentral scotoma occupying the apex of each left lower quadrant, the second a right-sided hemiopic scotoma involving the apices of both upper and lower right quadrants, and the third showed a defect in the lower inner part of each left lower quadrant extending to within ten degrees of the fixation point. Hegner concludes that the macular fibres are distributed to a very small circumscribed area at the posterior end of the occipital lobe, and considers that these cases provide evidence against the bilateral cortical representation of the macular area.

H. M. TRAQUAIR.

Knapp records a case of bilateral optic neuritis following influenza. A star-shaped figure was seen around the macula, resembling that found in albuminuric retinitis, but much more delicate and more silvery; it rather resembled the star sometimes found in optic neuritis associated with brain disease. There was a central colour scotoma. Improvement took place slowly, uninfluenced by treatment, and the ophthalmoscopic changes disappeared; but the vision fell appreciably (to 20/30) owing to macular lesions. The optic nerves may have been affected through the blood, through the cerebro-spinal fluid or through the nasal accessory cavities. The examination of the blood was negative in this case, whilst the changes in the cerebro-spinal fluid were those of a mild infection, the expression of a toxic reaction on the part of the meninges to the influenza poison. It was possible to exclude infection from the nose. Knapp believes that the optic neuritis was caused by a haematogenous infection, which at the same time produced a slight toxic reaction on the part of the cerebro-spinal fluid.

R. H. Elliot.


Seeing that it is by no means seldom that the ophthalmoscopic finding decides the diagnosis as to whether the disease one has to deal with is general or cerebral and indicates the therapy, at the outset Carl Behr emphasizes the importance of differentiating between oedematous papilla and inflamed papilla as early as possible in the course of development. He deprecates the indiscriminate use of “optic neuritis,” “papillitis,” and “choked disc” as though they were synonymous, and suggests that the employment of the terms “oedematous papilla (Stauungspapille)” and “inflamed papilla (Entzündungspapille)” would obviate confusion as to whether the ocular condition is due to infection or mechanical derangement of the lymph circulation in the eye.

He contends that the use of the two modern methods of examination—dark-adaptation and the stereoscopic view of the fundus by means of Gullstrand’s reflexless ophthalmoscope—enables one to arrive at an early and trustworthy differential diagnosis. In a
former communication he has shown that, as a rule, in inflamed papilla, even while the other functions are not appreciably deranged, there is a marked diminution of dark adaptation, whereas at the onset of oedematous papilla the dark adaptation remains normal.

Long before the ordinary ophthalmoscope reveals any change, a stereoscopic view with the Gullstrand apparatus brings into relief a glassy swelling involving some part of the edge of the disc, generally above or below or nasally, but never the whole disc simultaneously. Then the swelling spreads to other parts of the edge as in a hilly landscape. The retinal vessels ride over the swelling and are not enveloped by it. The lamina cribrosa remains visible, and as a rule even at a late stage the physiological cup does not become completely obliterated. As the swelling increases the unevenness of the disc diminishes, but the temporal side of the disc remains generally less prominent, even when the swelling is far advanced. Even when the prominence has attained its maximum, the transparency of the nerve tissue is maintained and thus the border of the optic foramen remains visible, although it is masked to the usual direct method of ophthalmoscopy. It is only at the stage when the presence of oedematous papilla is quite obvious even to the ordinary method of examination that the nerve-fibre tissue generally begins to get cloudy.

The inflamed papilla reveals itself quite otherwise to Gullstrand's apparatus. It never begins with a glassy transparent swelling. The cloudiness of the papilla begins very early and affects the structure not in parts but as a whole. The border of the optic foramen and the lamina cribrosa become veiled and invisible at a time when the swelling of the disc is at its minimum. Instead of the retinal vessels as they leave the lamina cribrosa riding over the swelling, they become overlaid and obscured by cloudy nerve fibres and exudate.

<table>
<thead>
<tr>
<th>Oedematous papilla</th>
<th>Inflamed papilla</th>
</tr>
</thead>
<tbody>
<tr>
<td>No disturbance of acuity and field of vision. Normal dark-adaptation.</td>
<td>In most cases, diminution of vision and scotomata in the field. As a rule marked disturbance of dark-adaptation.</td>
</tr>
<tr>
<td>Nerve fibre tissue long transparent, optic ring long visible, lamina cribrosa long visible, vascular funnel long maintained.</td>
<td>Clouded. Blurred or invisible. Veiled or invisible. Obliterated.</td>
</tr>
<tr>
<td>Vessels ride on the swelling.</td>
<td>All in parts overlaid by clouded tissue of papilla.</td>
</tr>
<tr>
<td>Perivascular space invisible.</td>
<td>Often recognizable as white sheathings of the vessels.</td>
</tr>
</tbody>
</table>

Carl Behr's theory of papilloedema is that it is a genuine oedema of the nerve fibres themselves, that it is altogether indepen-
dent of the blood vascular system, and that it is caused by centripetal obstruction to the flow of lymph. He discusses the other theories and shows how his theory explains the phenomenon better.

D. V. GIRI.

(5) Sharpe, William (New York City).—The pressure signs of certain intracranial conditions, observable in the fundus of the eye. Arch. of Ophthal., Vol. XLVI, No. 4, p. 320, 1917.

(5) Sharpe states that the effect of an increase of intracranial pressure upon the fundus of the eye can be easily demonstrated throughout its various stages by the experimental production of an internal hydrocephalus in dogs, and proceeds to describe his own experiments on the subject in some detail. He next insists on the necessity of keeping those terms which imply an inflammatory process, e.g., papillitis, apart from those which merely connote the effects of pressure, e.g., papilloedema and "choked disc." Pressure can and does cause congestion and oedema, but it does not cause inflammation, unless infection is present. Retinal haemorrhages are rare in cases of choked disc, due to even extreme intracranial pressure, when occurring alone, whereas they are very common in inflammatory conditions. Under pressure conditions Sharpe recognises the following stages: (a) blurring of the disc edges; (b) oedema hiding the details of the disc themselves; (c) measurable swelling of the disc (papilloedema); and (d) choked disc, where the papilloedema is greater than two or three dioptres. The signs of increased intracranial pressure as seen in the fundus must be checked by measurements of the pressure of the cerebro-spinal fluid, made by means of lumbar puncture with the use of a spinal mercurial manometer. If the pressure of the cerebro-spinal fluid is found to be increased, a more accurate diagnosis of the intracranial condition is reached; it is thus possible to exclude cases in which the optic discs are "normally blurred."

Sharpe next takes up certain intracranial conditions and discusses each in turn along the lines above laid down.

1. Intracranial tumour.—It is rare in cases of brain tumour for a choked disc to result unless the growth has either attained a very large size, or has blocked the ventricles; the prompt diagnosis of these cases by the recognition of the early papilloedema will make for the saving both of life and vision.

2. Brain abscess.—Inasmuch as the purulent detritus merely replaces the brain tissue, we do not meet with signs of intracranial pressure in these cases, unless the ventricles are blocked, or meningitis supervenes; it is rare for temporo-sphenoidal abscess to produce fundus changes even though the abscess may be very large.
3. Intracranial haemorrhage and oedema following fractures of the skull rarely produce choking of the discs, since the patient dies before this sign supervenes. It is less important to know the site and extent of the fracture than to ascertain the presence of increased pressure, since an early relief of this pressure by means of a decompression operation offers great advantages if effected before the patient reaches the dangerous stage of extreme medullary compression.

4. Cerebral spastic paralysis is due to three causes: (a) a lack of cerebral development; (b) a meningitis or meningo-encephalitis; and (c) an intracranial haemorrhage. In the first class there is no rise of intracranial pressure, nor is it present in cases of the second class which survive the inflammatory symptoms; whereas in patients with intracranial haemorrhage the increase of pressure is highly marked in the very young, and sufficient for recognition even in those who are older. The important point is that early diagnosis enables the surgeon to intervene for the relief of intracranial pressure.

5. Hydrocephalus.—The internal type of this condition, which is due to a blockage of the cerebro-spinal fluid in the ventricles, and which is accompanied by dilatation of the ventricles and high intracranial pressure, can produce a marked degree of choked disc, and its subsequent secondary optic atrophy. The recognition of the condition enables the surgeon to save the patient’s sight by means of a suitable drainage operation.

Sharpe shortly discusses some other conditions in which an ophthalmoscopic examination is of great importance in enabling a surgeon to recognize an increase in the intracranial pressure, including various forms of meningitis, and the different types of apoplexy.

The paper is well worthy of careful study, and the more so since it is pleasantly and interestingly written.

R. H. ELLIOT.


(6) Früchte sums up previous literature on this subject and gives notes of two cases from Axenfeld’s private clinic. The first case was a woman of 23 years who came complaining of a mist before the right eye. She had had an operation ten days before for ethmoidal suppuration in which an opening had been made into
the ethmoidal cells and also into the right sphenoidal sinus. The vision in both eyes was normal. Measurement of the field showed a blind spot thrice the normal size. This varied in size during the next few days, and eventually a complete Killian operation including the frontal sinus was performed. This was unfortunately followed by a paralysis of the superior oblique. The blind spot was reduced to normal and remained so. There was never any ophthalmoscopic abnormality. The second case was a man of 24 who had suffered from an injury to the head which was followed by loss of consciousness of short duration five weeks before being seen by Axenfeld. Immediately after the accident he noticed the sight was affected, but that, while the left eye speedily recovered, the right eye did not do so, and saw a black spot in front of it. The vision of the right was less than 5/60; that of the left 5/5. The field showed an absolute central scotoma extending out to about 15 degrees. There was no ophthalmoscopic change. Früchte believes that the most probable cause of the scotoma in this case was a haemorrhage in the central part of the nerve behind the globe. He thinks that pressure on the nerve may also play the most important part in the production of a central scotoma in cases of accessory sinus disease.

E. E. H.


(7) Lhermitte has published a very useful account of the pathology of encephalitis. The article is concerned chiefly with the work of other authors, and while containing nothing very new or original, will be found of considerable use to those interested in the subject. He first deals with the macroscopic and microscopic anatomy of the disease, and from this proceeds to what he calls the physiology, i.e., the correlation of the clinical symptoms with the lesions found. He concludes with an account of the experimental work done on the disease. He considers that the evidence as to the disease being caused by a virus that can be cultivated and not by a toxin, as earlier observers (Wernicke) had believed, is conclusive. Wiesner isolated a diplostreptococcus and injected a pure culture into an ape. The animal presented some symptoms of the disease. It was killed on the 12th day and showed characteristic lesions. Harvier and Levaditi, experimenting with rabbits, found that the germ passed the finest filters, and could not agree with Wiesner, but considered that the virus is analogous to the one causing acute poliomyelitis. Lhermitte is unable to offer any opinion on the matter.

E. E. H.