conditions. He appealed to members of the congress to make slit-lamp observations so that the true frequency of this condition might be established.

Mr. Frederick Ridley (London) described an "active" substance in the tears. He suggested that in glaucoma there might be a lack of some substance antagonistic to histamine-like bodies. His own work showed in normal tears a substance similar to histamine; it was not destroyed by boiling or by blood or serum. It was not acetylcholine. It produced the wheal and flare reaction in the skin and caused a rise in intra-ocular pressure through vascular dilatation.

Dr. Ida Czukrasz (Debrecen) described Hungarian plastic methods particularly those employed by Prof. Blaskovics and Prof. Kreiker, showing photographs of the excellent cosmetic results obtained in replacing large areas of diseased skin in the region of the lids.

ABSTRACTS

I.—CONJUNCTIVA


(1) Several cases of accidental infection in doctors have been observed by Wilson. In all of them the disease began after a few days with moderately acute symptoms such as redness and oedema of the conjunctiva, oedema of the limbus and slight discharge. In other persons, however, the disease begins imperceptibly, and is usually in Egypt obscured by chronic mixed infections.

Incipient Trachoma

In the initial stage there appear the phenomena of a subacute inflammation. In the lamina propria of the conjunctiva the superficial capillaries become dilated, the endothelial cells lining the smallest vessels enlarge and show signs of proliferation, there is an increase in the number of lymphocytes normally occurring in this situation, especially in the neighbourhood of the small vessels, and the number of histiocytes is greatly increased; the histiocyte has many other names—mononuclear cell, endothelial leucocyte, endothelioid cell, monocyte, polyblast, etc. A few plasma cells are present right from the beginning, but polymorphs are inconspicuous except in the epithelium itself, where they are to be found in small
The extent in the epithelium capsule. The of pockets of polymorphs in the crypts of the epithelium.

The epithelial layer shows increased activity of the mucin-forming cells, cell division is active in the deeper layers, and the number of layers becomes increased, the superficial cells becoming flattened.

Papillary hypertrophy occurs: this is due to the traction effect of fine fibrous bands which anchor the tarsal epithelium to the lamina propria (Goldzieher). It increases to such an extent that any follicles which form in the tarsal conjunctiva are entirely obscured, and are never recognizable.

In what Wilson calls the prototrachomatous stage the plasma cells increase in number especially in the papillae; the actual proportion of the different kinds of cell being — lymphocytes 30 to 35 per cent., plasma cells 20 to 25 per cent., histiocytes 45 to 50 per cent. and a few macrophages.

The first signs of trachoma follicles are small aggregations of the above mentioned cells at the upper edge of the tarsus and in the retro-tarsal folds; they tend to be flattened on the tarsus and spherical elsewhere. The follicles become scattered over the whole tarsal conjunctiva; thereby differentiating trachoma from other conditions of folliculosis, whether of the conjunctiva or of other mucous membranes.

The Trachoma Follicle

The trachoma follicle is the response to a definite infective process. It is best observed on the tarsus, since follicles are not normally found here. It has neither an endothelial lining, nor a capsule. The author has no doubt that the centre of the follicle is composed of histiocytes which have become localized as a reaction to the treatment; there are also here a few Leber cells or inflammatory macrophages such as occur in ordinary lymph nodes. In the older follicles the central cells stain poorly since they are undergoing autolysis. Nearer to the periphery there are a few cells with dark-staining nuclei, which are probably lymphoblasts; plasma cells are rarely seen. Here there is active cell-proliferation.

The opinion of Pulvertaft and MacCallan that the "bleb-like excrescences” of Tr. IIA are due to mechanical blocking of the ducts of Meibomian glands is in Wilson’s opinion untenable. These are best developed just above the upper edge of the tarsus and in the retro-tarsal folds where no Meibomian glands exist. Cysts do occur in the late stages of trachoma due to a blockage, but never to the extent of producing an actual protuberance of the conjunctiva. The follicles may disappear entirely in the general resolution of the inflammatory process, but this is by no means common. The larger superficial follicles, especially at the upper border of the tarsus
rupture spontaneously or with the slightest trauma. The deeper ones, surrounded by a dense infiltration of cells, especially in cases of marked papillary hypertrophy, never rupture but gradually disappear in the process of cicatrization. Cicatrization never commences within the follicle but the follicle is invaded from without.

The Follicles of Follicular Conjunctivitis

In follicular conjunctivitis the Halberstaedter-Prowaczek Körperchen, so constantly to be found in trachoma, are never present. There is very little papillary hypertrophy, since there is no diffuse infiltration of the subepithelial layer as in trachoma. The follicles become absorbed without the cicatrization which is characteristic of trachoma.

Trachoma Stage IIb

Follicles are invariably present in trachoma but they may be invisible clinically owing to the dense infiltration of the sub-epithelial layer. This raises the epithelium in huge papillae, between which there are deep epithelial crypts, the pseudo-glands of Henle or Berlin-Iwanoff. Within the crypts there may be collections of cells indicating a secondary infection; these may become blocked to form pseudo-cysts filled with epithelial débris. They may undergo calcareous degeneration giving rise to the concretions (P.T.D.) so frequently found in the cicatricial stage (Tr. III).

The cells which constitute the infiltration are almost entirely plasma cells; many of these degenerate and form an eosinophilic globule, with the nucleus pressed to the periphery (Russell bodies). The tarsus may be only slightly infiltrated. The epithelial layer is thinner with flattened cells.

Trachoma Stage III

Cicatricial changes begin at a very early stage of the disease, so that Tr. I passes rapidly to Tr. III and Tr. IV. In these cases there may be no thickening of the lid, but usually this does occur as the result, not of thickening of the tarsus itself, but of addition to the conjunctival surface of the tarsus of dense fibrous tissue, which replaces the infiltrated sub-epithelial layer. As trachoma heals, irregular strands of fibrous tissue are formed and islands of infiltration often remain for long periods within the mesh-work.

Other structures besides the conjunctiva may share in the fibrosis. Occasionally some of the accessory lacrimal glands (Krause’s glands) may show signs of disorganization, fibrosis and atrophy. The tarsus may show a certain amount of infiltration, when cyst-formation from blocked ducts may occur as a result of the general fibrosis. Such damage as occurs to the Meibomian glands is by an extension of the inflammatory process via the ducts. Changes occur in the
epithelium which becomes epidermoid. In the more severe cases the conjunctiva becomes xerotic.

In the latest stages of severe trachoma degenerations may occur, hyaline, hyalo-amylloid or osseous.

Conclusions

The pathology of trachoma is that of a chronic inflammatory disease which is entirely independent of any so-called constitutional dyscrasia. The type of pathological reaction suggests a virus infection and this is probably represented by the elementary bodies of the Halberstaedter-Prowaczeck Körperchen.

The disease is very protean in its clinical and pathological appearance but presents certain essential characteristics. Papillary hypertrophy, follicle formation on the tarsal conjunctiva and corneal lesions invariably occur and the disease terminates in scar formation.

The author particularly notes that he has omitted any consideration of corneal and limbal trachoma, one of the most important features of the disease.

A. F. MacCALLAN.


(2) The use of antimony in other granulomatous types of disease suggested that it might be a valuable remedy for trachoma.

Derkac gives the results of 50 cases which he treated with intra-venous injections of tartar emetic. The drug was given as a sterile freshly prepared 1 per cent. solution. Normal healthy adults received a dose of 2 or 3 c.c. on alternate days, and weaker patients only 2 c.c. every 3rd or 4th day. Children over 10 years of age were given an initial dose of 0·5 c.c., increasing every 3rd day by 0·2 c.c. up to 1 c.c. It was considered unsuitable for children under 10 years of age. After a course of 10 doses there was an interval of 10-14 days.

There was a rapid disappearance of pannus, and a beneficial effect upon the conjunctivitis and ptosis. The best results were obtained when local application of copper stick was combined with dosage with antimony.

D. R. CAMPBELL.


(3) Castelli draws attention to the value of peroxide of zinc in the treatment of trachoma; this substance was first used by
Pignatari in the Italian Hospital in San Paulo, Brasil. Though he published accounts of its success, the drug was taken up by few, and has been unduly neglected.

Its application is very simple; after local anaesthesia, for which in Pavia percaine is used because of its prolonged action, the powdered peroxide is brushed over the everted conjunctiva with a sable brush. The eye is bandaged and kept closed for half-an-hour; then the powder that remains must be carefully removed by flushing the fornices copiously with warm normal saline, aided by small swabs on glass rods; it is specially important that no powder be left at the bottom of any ulcer that may exist, since this may become a source of irritation and perhaps of danger to the eye.

HAROLD GRIMSDALE.


(4) Meloni regards trachoma as a manifestation of the adenoid condition, which spreads from the naso-pharynx to the conjunctiva. This specially affects individuals who are lacking in calcium and potassium, whose blood is insufficiently alkaline. He regards trachoma as double in nature. The primary adenoidal condition has added on to it, infection by various pathogenic organisms which take advantage of predisposed conjunctiva to flourish there. It is this part which is contagious; Ruata has attempted to treat the disease by combating these latter infections by procuring immunisation of the conjunctiva by means of vaccines of all the various strains of bacteria found to flourish in the trachomatous conjunctiva; this vaccine, "lacmin," is applied either by painting or by instillation. To lessen the acidosis, the author has treated some of his patients by "endoneutralin" and has found the Ph increased in all cases.

HAROLD GRIMSDALE.


(5) Cuénod and Nataf maintain that the virus of trachoma is related to the rickettsia group or is carried by members of this group. The inclusion bodies of Prowazek and Halberstaedter and the analogous small granules which are present in great numbers in the smear of trachomatous follicles are rickettsoid bodies. These bodies are to be found regularly in the intestines of lice collected from trachomatous patients, while they could be discovered only in 5 per
cent. of the lice of healthy people. The louse is credited with transmitting the trachoma virus. At least, it is a real store-house of it. As the same parasite is known to transmit typhus, the analogous geographical distribution of both diseases is a strong argument in favour of Cuenod and Nataf's views. Typical experimental trachoma could be produced in monkeys (macacus inuus) by conjunctival or subconjunctival inoculation of the intestine of lice, which had been infected with trachoma before. Likewise, the authors succeeded in producing a "wonderful" trachoma by a similar inoculation of a human eye. The period of incubation lasted ten days. The virus of trachoma could be subjected to multiple and alternating passages in the testis of the guinea pig and in the intestine of the louse without losing its pathogenic qualities.

HUMPHREY NEAME.


(6) Federici holds that the use of nitrate of silver, in accordance with Credé's method, is too dangerous to be attempted by any person without special skill. He has seen many cases of ophthalmia neonatorum recover without any damage which, though being brought to him on the fifteenth day of the disease or later, have received no treatment except washing with milk, and on the other hand has seen much damage following inexpert and excessive treatment. He advises that for prophylaxis the method of Tornatola should be adopted; the lids, lashes, and neighbouring parts should be cleaned with sterile cotton wool, and then washed with sterile water, a solution of hydrogen peroxide, orboric acid. Then the lids may be gently opened and some drops of 20 per cent. argyrol instilled; the lids must be held open for some minutes. Where infection is suspected these drops may be repeated several times. Similar precautions should be taken in the adult when infection is possible.

If infection has occurred, the sac must be washed out frequently with 3 per cent. boric acid solution, and drops of argyrol instilled. In addition compresses of 1 per mille salicylic acid, at the temperature of the room should be put on for half-an-hour three times a day.

If there is oedema of the lids, and free discharge of pus, he advises injections of milk daily or every other day.

When the disease is subsiding, leaving the tarsal conjunctiva hypertrophic, nitrate of silver may be painted on the everted lids, any excess being washed away at once. He inserts in the cul de sac an ointment of 1 per cent. picric acid.

HAROLD GRIMSDALE.
(7) Cecchetto (Verona).—Should Crède’s method of prophylaxis be generally used in ophthalmia neonatorum or not? (Il metodo Crède, quale profilassi e cura della congiuntivite dei neonati e consigliabile o no?). *Rass. Ital., d’Ocul.*, September-October, 1937.

(7) Cecchetto has seen such bad results following the indiscreet use of nitrate of silver as treatment for ophthalmia neonatorum, that he would forbid its use entirely. He holds that infection during birth is very rare and that if the lids are carefully cleansed with sterile water, later infection is equally rare. If the eyes show signs of redness and discharge within a few days of birth, the bacterial content of the discharge is ascertained, and the appropriate remedies used. He holds that silver nitrate increases the virulence of the gonococcus.

Harold Grimsdale.


(8) According to the Egyptian Ophthalmic Hospital Report for 1936 more than two thousand cases of spring catarrh complicated by trachoma, signified in MacCallan’s classification as “Trachoma Stage IIIb” were examined among the million new patients at the hospitals.

The incidence of this conjunctival hyperplasia at these hospitals was under a hundred annually from 1919 until 1931, after which it increased irregularly until it is now 2,059.

No explanation for this marked increase has been offered. Clinicians state that they now recognize many more cases than in former years.

Maxwell Lyons, who is working at the Memorial Laboratory of Egypt, recognizes an inherent sensitivity to explain why one person becomes afflicted with the disease and another, of the same age, sex, habits and environment escapes.

The average age of onset is between 14 and 15 years. The proportion of male to female patients affected is as 3 to 1.

In the great majority of cases in Egypt the disease begins about the middle of the summer, persists throughout the remainder of the hot weather, subsides during winter, recurring each subsequent summer, generally at a date earlier than that of the first attack. Between attacks there is a marked tendency for the lesions to clear up, but about 20 per cent. of the sufferers retain the disease in a less active form throughout the winter months.

The patient’s sensitivity seems to have an allotted span, about six years, after which spontaneous cure occurs.
The fibrinous exudate, in which an excess of eosinophile cells are entangled, is well-known.

The lesions are not due to a proliferation of pre-existing connective tissue, but to an invasion of the normal conjunctiva by new fibrinous material and wandering cells, originating from the capillaries of the conjunctiva.

The author discusses the calcium content of the blood in this disease, and also the effect of the exhibition of calcium. He considers that there must be an intangible and indefinite abnormality of the capillaries. The lesions of spring catarrh are merely local manifestations of a general bodily departure from the normal.

A. F. MacCallan.

II—MISCELLANEOUS


(1) This is the admirable Mackenzie Memorial Lecture delivered in Glasgow by Paton on October 29, 1937. After a brief account of the life of the greatest British ophthalmologist of pre-ophthalmoscopic days in which special attention is paid to Mackenzie's views on amaurosis, Paton pays tribute to von Graefe's original observations on the subject of optic neuritis. He goes on to describe several most interesting cases which have come under his own observation during the past 30 years. He emphasises the fact that optic neuritis is only very rarely caused by inflammations in the nasal sinuses or even in the orbit itself. "The nerve seems to possess a very strong protection against infection reaching it through its sheaths." But, in one of his cases here described he considered that the connexion seemed to be definite. In another case dental sepsis appeared to have a distinct bearing on a case of retrobulbar neuritis.

The chief part of his paper, however, is concerned with the demyelinating diseases of the central nervous system which he divides into three sections:

1. "Disseminated sclerosis, where the patches are discrete and sharply outlined, and may occur in the optic nerve, or anywhere in the central nervous system where myelinated fibres exist. This is responsible for the great majority of cases of retrobulbar neuritis.

2. Neuromyelitis optica, or Devic's disease, where the patches are diffuse and affect mainly the white matter in the spinal cord, causing an ascending myelitis, and, in a number of cases, affecting also the optic nerves and optic chiasma.
3. Encephalitis periaxialis diffusa (or Schilder's disease), where the demyelination is diffuse and commences first in the centrum ovale of the cerebrum, and, seemingly, most frequently in the occipital lobe. In this disease, also, the optic nerves and chiasma are also attacked in quite a definite number of cases."

Most interesting cases of each of these three groups are described in this paper which should be read in the original by all ophthalmologists and neurologists.

The paper is illustrated by the fine portrait of Mackenzie which appeared in our first volume and by four pages of charts and pathological sections to illustrate the various points discussed.

Many of these cases of optic neuritis associated with diseases of the nervous system are most dramatic and we can almost imagine Mr. Paton on meeting such examples speaking to himself in the words of Bailie Nicol Jarvie when he met his cousin Rob Roy in the Glasgow tolbooth. “Ah!—eh!—oh! my conscience!—it’s impossible! and yet—no!—conscience!—it canna be!—and yet again—Deil hae me!—that I suld say sae!—ye robber—ye cateran—ye born deevil that ye are, to a’ bad ends and nae good ane!—can this be you?"

"E’en as you see, Bailie," would be the “laconic answer” of the disease in question.

R. R. J.


(2) King describes the cellular classification of the melanomata elaborated by Callender and Wilder and that depending on the amount and distribution of reticulin. The cells which compose the melanomata of the eye fall into four main groups (1) the spindle cell type with two sub-groups (a) in which the nucleus has a fine delicate reticular chromatin network and (b) in which the nuclear chromatin is coarse and stains deeply; (2) the epithelioid type, composed of large polygonal cells of variable sizes, believed to originate from endothelial cells; (3) the fascicular type in which the cells are elongated, oval or spindle and arranged in columns along a central capillary or lymphatic; (4) the mixed cell type composed of spindle epithelioid and fascicular cells present in varying proportions in different areas of the neoplasm.

Reticulin has freely anastomosing fibrils, at some sites the branched cells of the reticulo-endothelial system are wrapped round the fibrils, it stains poorly with ordinary stains but is well differentiated by silver preparations. Reticulin may be revealed in specimens which have been embedded in celloidin for a considerable
time by preliminary sensitisation with uranium nitrate. The author has found it advisable to bleach all sections before using the silver stains.

The distribution of reticulin is variable (1) no fibrils at any site, or only in the stroma between lobules of tumour cells; (2) areas of neoplasm with and without fibrils sub-divided into (a) neoplasms with a preponderance of areas without fibrils (b) tumours in which areas with fibrils preponderate (e) neoplasms in which areas with and without fibrils are approximately equal; (3) fibrils between individual tumour cells throughout the whole mass.

The prognosis of the melanomata depends on their cytological character and the amount and distribution of their reticulin content. The spindle celled neoplasms give the most favourable prognosis, then the fascicular, the epithelioid and the mixed celled about equal. The presence of the so-called epithelioid cells alone or with other cell types gives the highest mortality rate. When all areas of the neoplasm contain reticulin fibrils no metastases occur and the patients were alive after five years; when some areas were without fibrils metastases followed in 36 per cent. of cases, and 68 per cent. died within five years and when no fibrils were present except in the inter-lobular stroma all the patients had died. It seems that reticulin must operate as a mechanical barrier against the neoplastic cells entering the blood vessels. It is therefore essential that every specimen of melanoma of the eye should be examined for its reticulin content and distribution for the purpose of prognosis.

H. B. STALLARD.


(3) It has been disputed whether recurrent retinal haemorrhage in young persons is due to a primary tubercular infection of the vessels, or to a primary thrombo-angeitis obliterans.

Suganuma describes the case of a girl, aged 21 years, with recurrent intra-ocular haemorrhage in one eye, which later developed scleritis and tuberculous iritis and had to be enucleated on account of secondary glaucoma. She had apical phthisis. Section showed several circumscribed tuberculous spots in the neighbourhood of the retinal vessels without any thrombosis of the latter. There was definite tubercular infection in the anterior part of the eye.

D. R. CAMPBELL.

(4) In opening the discussion on myopia at the Oxford Ophthalmological Congress Bishop Harman commented on the hereditary factor in myopia and the effects of social conditions such as deficient diet, overcrowding and bad hygiene which lead to an increase of this refractive error. It appears that Sweden has reduced the incidence of myopia in school children by 20 per cent. through improved lighting conditions and better and further facilities for outdoor exercise. The incidence of short-sight is less on the modern side than on the classical side where dictionaries are extensively employed.

He states that myopes between 20 and 60 years of age deteriorate more rapidly in occupations involving close work if the principles of ophthalmic hygiene have not been carried out during the years of school life than would otherwise be the case if these precautions had been taken.

Arnold Sorsby criticised the nineteenth century view of myopia as being due to a mechanical increase in the axial length of the globe. He believes that low and medium myopia is non-pathological and non-pathogenic but the higher degrees some of which progress to macular degeneration and retinal detachment are pathological.

In the discussion that followed Beatson Hird stated that there was insufficient evidence to regard calcium deficiency as a factor in progressive myopia. In a study of 3,500 consecutive cases of myopia he found retinal detachment in 0·7 per cent., 72 per cent. of whom were over 40 years of age and in four out of 25 persons thus affected both eyes were involved. Macular degeneration was present in 3·8 per cent., 63 per cent. of whom were over 40 years of age, and 7 per cent. had over 10 D. of myopia.

Williamson-Noble said that there were two types of myopia, one which was associated with growth of the whole globe and the other was due to stretching of the posterior segment of the globe when it was unsupported by the extra-ocular muscles.

Harrison Butler supported the conception that heredity plays a part in myopia and that this refractive error is aggravated by adverse social conditions.

H. B. Stallard.

(5) Motelese and Berardi (Florence).—Heredity in ocular disease. *(NTS SULL' EREDITA NELLE MALATTIE OCULARI.)* *Boll. d'Ocul.*, November, 1937.

(5) The authors have examined the refractive errors in a number of families, including the parents and the issue. They conclude that
corneal astigmatism (not including that due to scarring) is hereditary and may be transmitted by either parent to the children without distinction of sex. They are not able to say whether astigmatism is a dominant since there are observations for an insufficient number of generations.

It will be noted that the form of astigmatism and the degree frequently differed between the parents and the second generation. Thus in some cases the astigmatism was "with the rule" in one generation and "against the rule" in the other. Sometimes the amount varied much, thus in Family XXXII the parents are nearly emmetropic; 0·50 D. cyl. only; one son has an −4·50 D. Since it is a matter of every day observation that families have a "likeness," it might be expected that there would be similarity in the shape of their eyes.

HAROLD GRIMSDALE.


(6) Campos has investigated the mechanism of the production of papillary stasis, by ligaturing the optic nerve in different positions, and noting both the ophthalmoscopic appearances following, and the microscopical changes in the state of the nerve. There are naturally great differences between the early changes seen when the vessels are included in the ligature; the immediate stoppage of the circulation produces intense venous stasis, and numerous haemorrhages round the disc; later there is oedema from interference with the lymphatic flow. Roughly the time necessary for the development of papilloedema varies with the distance of the ligature from the globe. The nerve on both sides of the ligature shows oedema, extending much further on the distal side than centrally; and when the ligature is placed far behind the point of entrance of the vessels into the nerve, the oedema often stops abruptly there. The nerve sheaths also, show changes; the intervaginal spaces are largely obliterated partly by the swelling of the nerve, partly by inflammatory changes in the sheaths themselves. If an opening be made in the sheath, the swollen nerve will be seen to bulge into the gap, and papilloedema will come on as usual unless the ligature is very far back and the opening large; in this case the appearance of papilloedema will be delayed and its intensity lessened.

The author gathers from these observations that there must be a current of lymph from the eye centrally along the nerve, and that the interference with this flow has an important part in the production of papilloedema; he compares the results of his experiments with that of Dimissianos, who found that by injecting paraffin into
the orbit so as to compress the nerve where it contained the vessels, he produced papilloedema. It is easy to explain the onset of papilloedema when the central veins are compressed; there are difficulties in determining the cause when the venous flow is little affected. Campos thinks that many factors may enter into the production; the compression of the vessels where they pass through the sheaths of the nerve may be aided by the oedema of the nerve spreading from the brain, or brought about by the stoppage of the lymph flow in the nerve.

HAROLD GRIMSDALE.


(7) Raab describes the variability of the “recovery-point” in convergence tests; as convergence is increased there comes a point at which the image becomes blurred and then diplopia occurs. By weakening the prisms (i.e., lessening the convergence) the two images are “re-fused”—this is the recovery point. The author finds that (1) The position of the recovery point depends on muscle tone, which shows individual differences; (2) The position of the “blurring point” which is the higher limit of relative convergence, indicates that increase of tonus is possible; (3) Certain individuals show a primary and secondary “recovery point.” The position of the primary depends on the persistence of over-accommodation convergence; the second depends on muscle tone. (4) Two types of convergence insufficiency can be distinguished—a simple hypotonic form, and a hypotonia compensated by accommodation. (5) The curves for the “blurring” and “recovery points” do not usually cross one another.

D. R. CAMPBELL.


(8) Jess describes the use of a tampon to produce temporary pressure outside the sclera after diathermy round the site of a retinal hole, and removal of the subretinal fluid by oblique puncture with a hypodermic needle. The tampon is inserted inside Tenon’s capsule, and is secured by sutures which are led out through the conjunctiva. It is left in situ for fourteen days and removed under a local anaesthetic.

D. R. CAMPBELL.