I—IRRITATION OF THE CERVICAL SYMPATHETIC


Wölflin here reviews the facts already recorded, and adds some fresh observations about a condition to which little attention has hitherto been paid in ophthalmological literature as compared with paralysis of the cervical sympathetic.

The symptoms of irritation are (a) ocular, and (b) vascular. In the great majority of cases these are associated, while less frequently only those of the one or the other group may be present.

The ocular symptoms, in the order of their importance and frequency, are mydriasis, widening of the palpebral fissure and exophthalmos.

Mydriasis is always observed; it may be the only ocular symptom present. In the author’s cases the pupil was not dilated ad maximum and reacted to light and on convergence, though more slowly than normally. He doubts the statement in Römer’s textbook that absolute rigidity of the pupil may occur.

Widening of the palpebral fissure is nearly always present. This sign is best observed in the dark room by throwing the light with a mirror rapidly from one eye to the other, the difference between the two being then increased. When observed for some time in diffuse daylight, the variation in the distance of the upper lid margin above the limbus is often striking.

Exophthalmos is the least reliable sign, and when present is usually not marked. If it is not accurately measured its presence may be falsely inferred from mere inspection of the width of the palpebral fissure. While the other two signs each owe their origin to the action of one muscle, the processes concerned in the production of exophthalmos are more complicated, and the explanation of it is rendered more difficult by the fact that the results of experimental research have not been uniform, enophthalmos having been observed by some. Other symptoms of less regular occurrence are contraction of the conjunctival vessels, as well as those of the retina and uvea, most marked in the retinal arteries and veins; an increased flow of tears, a rise in the intraocular tension, and an increase in the dilatation of the pupil on pressure over the superior cervical ganglion.

The vascular symptoms are very variable. Typically they consist of pallor of the corresponding side of the face and lowering of the temperature, but the conditions may be the reverse of these,
accompanied by increased perspiration. In rare cases they are found on the opposite side of the face. In the experience of the author these symptoms are less constant and pass off more rapidly than is the case in paralysis of the cervical sympathetic.

The causes of irritation consist of a peripheral group (where compression of the cervical sympathetic is due to cervical tumours, enlarged lymphatic glands, etc., commencing apical tuberculosis, pleuritic bands, and trauma), and a central group, arising in a subcortical vasomotor centre in the mid-brain. In cases of central origin the vascular symptoms alone are usually present, and are elicited by various states of emotion, and in angina pectoris and allied conditions.

The diagnosis is usually easy, though sometimes more difficult than that of paralysis, if attention is directed to the oculo-pupillary symptoms alone. The prognosis is better than that of paralysis. In the majority of the central group of cases improvement or complete recovery ensues. In those of peripheral origin it depends on the early removal of the exciting cause.

As to treatment, apart from operative procedure, only galvanisation of the cervical sympathetic has hitherto been recommended.

THOS. SNOWBALL.

II—THE MICROSCOPIC STUDY OF THE LIVING EYE


Gallemaerts and Kleefeld give an appreciative description of the corneal microscope of Czapski as used with the Gullstrand type of lighting, that is, the use of the Nernst lamp with a slit. The microscope is a binocular one, fitted with Porro correcting prisms and an adjustment for interpupillary distances of 56 to 76 mm.; various power eyepieces and objectives giving magnifications of 8 to 103 times, with fields of from 13 to 1.8 mm. in extent. The microscope is mounted on a stand resting on a table provided with a chin and head rest, with all the necessary adjustments. The luminous source is a horizontally-placed Nernst lamp fitted on a mobile stand, the lamp being placed in a tube whose far end is occupied by a vertically-placed adjustable slit with rounded corners. Between the light and the slit is a first condensing lens, on the far side of the slit is a movable disc pierced with three openings, a rectangular one identical with the longest diameter of the slit, the other two circular. By rotation of the disc each of these can...
be brought successively into the axis of the tube. The light of the filament is condensed by a second aspherical lens of 7 cm. focal length and 50 mm. aperture, or of 6 cm. focal length and 43 mm. aperture, the more rounded surface being turned towards the lamp and the lens being furnished with a rectangular diaphragm with rounded corners similar to those of the slit. This second lens is placed in the line of the light emitted by the lamp, and can be moved to and fro on a 16-inch long arm projecting from beneath the lamp and moving with it. On this arm between the lamp and the lens can be placed coloured glasses or a parallel-sided cell with such fluids as fluorescein, eosin or methylene blue. The light should be directed on the eye to be examined at an acute angle with the direction of the microscope, and by the various special adjustments provided one can focus from the surface of cornea right down into the vitreous as required. The authors advise waiting for dark adaptation to occur before using the microscope in the dark room, as is necessary. The lighting is so regulated by the examiner that he obtains a slightly elliptical, uniformly lit, luminous band on the surface to be examined. When the higher powers are used more intense illumination is brought to bear on the eye by simple focussing. The size of the image is measured by an ocular micrometer, the depth by the vernier scale or graduated wheel attached to the substage.

The microscopic study of the living eye has been carried out very fully by Léonard Koeppe, who has endeavoured to bridge the gulf between ordinary naked-eye examinations and those of microscopic sections in this way and has recorded his observations in Vol. XCI et seq. of v. Graefe's Archives. W. C. Souter.

III—GLAUCOMA AND EPIDEMIC DROPSY

Maynard, Lt.-Col. F. P., the late, I.M.S., (Crewe).—Glaucoma and epidemic dropsy. Indian Med. Gazette, August, 1921.

This is a report on 104 cases of glaucoma associated with epidemic dropsy in Calcutta. All but three of the cases were seen in an epidemic which occurred in 1909; and they included 22 cases recorded in the Indian Medical Gazette of October of that year. A few of the cases have been followed up for ten years or more.

Epidemic dropsy has been confused with the dropsical form of beri-beri. But Colonel Maynard agrees with Braddon, Manson and others, that it is a distinct disease, not due to vitamine-deficiency in diet. Anaemia is characteristic, mostly with oedema, particularly of the legs. Many of the cases begin with fever and
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diarrhoea. There may be severe heart trouble, with risk of sudden death, though the mortality in some epidemics has been low. Maynard found the blood-pressure high. Several members of a household are usually attacked.

Of the patients affected with glaucoma 80 were male and 24 female. The average age was 34 years, only 19 patients being over 45 years old. This early age is noteworthy. The average time of onset of the glaucoma was about 5 months from the beginning of the disease.

The increase in tension was usually moderate, without shallowing of the anterior chamber or noticeable dilatation of the pupil. Congestion of the eye and pain were not very frequent, but the cornea was steamy in 49 cases, and coloured haloes were complained of in 73 cases. Contraction of the field of vision and pathological cupping of the disc were each present in about half the cases. The cupping developed rapidly.

Fifty-four eyes were operated upon, 34 by iridectomy, 18 by trephining, 2 by Lagrange’s operation. Trephining was the most effectual.

H. HERBERT.

IV—CONGENITAL WORD-BLINDNESS

Wallin, J. E., Ph.D.—Congenital word-blindness. Lancet, April 23, 1921.

Wallin is Director at the St. Louis Psycho-Educational Clinic and Special Schools. There is not a great deal that is original in his article as regards word-blindness itself, but he gives with particular clearness the position of word-blind children as pupils at school, and brings out their position as compared with the feeble-minded. While 85 per cent. of his cases were classified as subnormal in intelligence, only 5.2 per cent. were classed as feeble-minded. The article concludes with the practical point that word-blind children who are not feeble-minded should be assigned to special reading-disability classes, but the author admits that in St. Louis the financial difficulties have prevented any action being taken in the matter.

In the Lancet for April 30 there is a letter from Dr. Wood, of Rugby, referring to Wallin’s article. This letter is rather misleading in one respect. The writer says, “I wish he (that is Wallin) had paid more attention to the question of inheritance, which was first dealt with by Mr. Sydney Stephenson, for to my mind this line of investigation will lead to a better understanding of the disease. A striking thing about Dr. Wallin’s paper is the
close similarity between his figures of incidence and those of colour-blindness, and also the fact that the sex-incidence is the same. This similarity of incidence would suggest strongly that it is hereditary, and it would be informing to follow cases to see if the method of transmission in word-blindness follows the same course as that of colour-blindness.” These remarks would lead one, the reviewer thinks, to suppose that Wallin had specifically referred to colour-blindness, but this is not the case. Wallin speaks of word-blindness being apparently to some extent sex limited. He says that word-blindness is about four times as prevalent among boys as among girls, but the suggestion as to any similarity between the two conditions is entirely Wood’s.

Those interested in the educational aspect, more especially of the subject of word-blindness, would do well to read Wallin’s article.

ERNEST THOMSON.

V—THE AETIOLOGY OF SPRING CATARRH

Gabrïélidès, A. (Constantinople).—Etiology of spring catarrh.

(Étiologie de la conjunctivite printanière.) Bull. et Mémoires de la Société française d’Ophtal., 1920.

The author’s researches on the above subject have already shown that eosinophile cells are present in excess in the conjunctival secretion, the tarsal vegetations and in the peripheral blood. In a further paper he has demonstrated that the disease is often present in more than one member of the same family, pointing either to a microbic origin or to a family idiosyncrasy. He has failed, however, to discover a specific organism in the secretions or vegetations and cultivations from the blood have also proved negative. Examined from the clinical standpoint the histories of 15 families are given, in each of which families two or more members are affected, the peculiar fact is brought out that it is usually two brothers or a brother and sister who have the disease, the other members of the family remaining unaffected; in other words, it does not seem to be contagious, and therefore is probably neither microbic nor parasitic, being rather of the nature of a personal malady affecting those members of a family who are specially susceptible. In order to test this theory he proceeded to inoculate a monkey by rubbing the tarsal proliferations into the scarified conjunctiva. The result after long observation was negative. He then inoculated the blind eye of a girl, but with the same result. He finally repeated the experiment on a small boy aged 6 from his brother aged 8 who was suffering from the disease. The result was again negative,
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thus demonstrating that the disease is probably not due to an organism.

On the other hand, the eosinophile excess suggests that a toxic element is present. This excess occurs in many other diseases, including certain skin maladies, and the author thinks he can trace an analogy between spring catarrh and various skin affections, e.g., psoriasis, as in each case the action of the ultra-violet rays, and probably thermal rays also in the case of spring catarrh, is an important factor in provoking the onset of the disease. As a result of the irritation set up the cells secrete a toxin, against which the organism protects itself by multiplying eosinophiles. Viewed from this standpoint the affection of the tarsus is merely a symptom of a general malady which may sometimes be accompanied by enlarged glands, e.g., cervical or even axillary or inguinal, and occasionally there is such pronounced hypertrophy of the nasal mucous membrane that the disease has been described as "nasal conjunctivitis."

The author therefore arrives at the conclusion that spring catarrh is a manifestation of an auto-intoxication. The toxin probably exists in the serum and by stimulating the nerve endings provokes the pruritus which is a common symptom in this disease. As to the nature of the toxin and where it is produced he does not hazard an opinion.

CHARLES KILLICK.

VI—HISTO-PATHOLOGY OF TRAUMATIC CHOROIDO-RETINITIS PROLIFERANS


Mawas has been surprised, like other ophthalmic surgeons, by the frequency of choroido-retinal lesions accompanying wounds of the face or skull, with apparent integrity of the organ of vision. These lesions start by a more or less abundant haemorrhage which clears up in a few weeks' time. Its absorption is followed by the typical picture of traumatic choroido-retinitis proliferans usually found at the macula or surrounding the disc. The author has tried to solve the problem as to how these lesions have been brought about, to discover the relationship between the primary haemorrhage and the subsequent changes, the source of the effused blood, and to what extent they are curable.

They can be grouped according to the following table:—(1) Lesions of attack. This is the period of onset characterised by
haemorrhage from the choroid and rupture of the retina. (2) Lesions of defence or reaction which may last for several weeks; and (3) Lesions of consolidation, which is the last phase, characterized by absorption of the blood, cicatrization, and the fusing of retina with choroid.

Mawas states that the blood is derived from the lamina chorio-capillaris. If the initial shock is not great the haemorrhage may be absorbed without any trace being left ophthalmoscopically, as it fails to penetrate the layer of pigment epithelium. Greater force, however, causes the epithelium to yield, though with difficulty, and the retina is detached in one or several places. Microscopic sections demonstrate that this primary rupture is situated at the macula where the retina is thinnest. Other retinal lesions, partly due to concussion and partly to dissociation produced by the red corpuscles, appear at the same time. These include necrosis of the layer of rods and cones and degeneration of the layer of ganglion cells, while the other retinal layers undergo a process of fusion. While these changes are taking place the haemorrhage is being replaced by a black pigment directly derived from it. The above phase constitutes the lesions of attack. The lesions of defence are due to an effort of the retina to protect itself by an intense phagocytosis of the haemorrhage and the necrosed elements, together with the proliferation of the pigment epithelium, the neuroglia and connective tissue, the result being the formation of the intensely white areas and patches of pigment, while in addition there is vascular congestion which produces an inflammatory-oedema leading to a fibrinous exudate and diapedesis. Such are the main features observed microscopically.

The last phase or lesions of consolidation consist of a penetration of the vitreous by "young elements" (Mawas' own words*) and fibroblasts, which are gradually replaced by a dense connective tissue, which takes the place of the destroyed retina and forms a union between the vitreous proliferation on the one hand and the choroidal lesions on the other.

* He does not say whence derived.—C. K.

CHARLES KILICK.

VII—SECONDARY GLAUCOMA


Abadie's article is somewhat discursive, but the reviewer wishes to bring forward an idea which seems to be original on Abadie's part. He says, "Contrary to what was formerly held, these glaucomas may come on without the least adhesion or the
least sub-iridal exudate. How are we to explain their appearance?" He proceeds to recapitulate the facts regarding the blood supply by reflex action, and points out that if the centripetal peripheral excitation becomes abnormal the centrifugal action caused by it becomes also abnormal and is followed by an abnormal circulation. "So when an infective process strikes the iris tissue it necessarily irritates in an abnormal way the circular sympathetic nervous plexus contained in it. This irritation of the sympathetic, transmitted to the ophthalmic ganglion or to the Gasserian ganglion, or possibly to other ganglia farther off, brings about reflex vaso-dilatation of the ciliary arteries, increased blood supply, increase of intraocular tension, and the glaucomatous state." Then again, after dealing with the effect of treatment, preventive and otherwise, Abadie asks, "Why does iridectomy cause the disappearance, in such a remarkable way, of these secondary complications (in this case it must be remembered that the glaucoma is the secondary complication referred to), the prognosis of which, without iridectomy, is absolutely hopeless." He answers his question in the following words, which the reviewer has thought should be carefully considered by ophthalmologists in order that their correctness may be confirmed or denied: "In removing a part of the iris tissue by iridectomy we are taking away a part of the sympathetic nervous tissue which it contains. We are, in fact, performing an iridal sympathectomy. We are cutting the nerve circuit... Once this circuit is cut, things become normal, the peripheral centripetal irritation being no longer transmitted and the centrifugal vascular disturbance caused by it in turn disappearing."

ERNEST THOMSON.