Haemorrhage into the socket after enucleation

Another clinical feature which the author wishes to bring out is the frequency of haemorrhage into the socket after enucleation. Out of 17 eyes the subject of thrombosis, of which a note was made on their case papers, extensive haemorrhage into the socket occurred in ten cases. Of the seven remaining cases, in four there were no notes as to whether haemorrhage took place or not. In two cases which were definitely thrombosis of the central vein secondary to primary vascular sclerosis, there was complete absence of haemorrhage and the wound healed well. In the one remaining case of thrombosis of the central vein which followed a septic iridocyclitis, no haemorrhage took place after the removal of the eye.

Although these are the two principal clinical facts which the author wishes to bring out in collecting these seventeen cases of thrombosis of the central vein, there are several other facts which have been previously noted by other observers which may be of interest from an aetiological point of view. There was a history of injury in three cases, and old uveitis had been present in four cases. Both eyes were affected in four cases. In these four cases three of the patients suffered from albuminuria, and in the other case no record of the urine was made. Albuminuria was present in seven cases, and definitely absent from eight. The onset of glaucoma after the thrombosis varied between five days and one year. A detached retina in addition to the thrombosis was present in two cases, and in both cases the patient suffered from albuminuria. Sub-choroidal haemorrhage frequently followed iridectomy, and in the cases which were trephined, the trephine hole became subsequently blocked by exudation. Pathologically, in some cases the angles of the anterior chamber were found wide open, filled with coagulated albumin, and in other cases the angles were blocked by the apposition of the root of the iris and the back of the cornea. The new vessels on the surface of the iris were traced back to the ciliary circulation by serial section.

**SOME POINTS IN RETROBULBAR NEURITIS, WITH
SPECIAL REFERENCE TO PROGNOSIS***

BY

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UNDER the diagnosis of retrobulbar neuritis several different conditions are included. It should, I think, be limited to those

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*A communication to the Oxford Ophthalmological Congress, July, 1917.

cases in which there is, from some cause or another, an inflammation of the optic nerve, or its sheaths with involvement of the nerve, in some part of its course between the eye and brain.

For the present, however, it may be divided into :

(1) Retrobulbar neuritis proper, subdivided into acute cases and sub-acute cases.

(2) Hereditary optic atrophy or neuritis.

(3) Toxic amblyopia.

(1) *Retrobulbar neuritis proper*.—In March, 1897, a discussion took place at the Ophthalmological Society of the United Kingdom.* The opener, Mr. Marcus Gunn, remarked that the optic nerve was peculiarly liable to attack, due to its double character, viz., as an outlying part of the central nervous system, it partook of the susceptibilities of that system, and at the same time was liable to exposure in a similar manner to a peripheral nerve.

As regards the cause of the inflammation, he mentioned three groups :

(1) From inflammation communicated to it by neighbouring structures. He compared the optic nerve in the optic canal with the seventh nerve in its bony canal, and suggested the probability of rheumatic affections of the nerve sheaths, accounting for those cases directly caused by exposure to cold.

(2) Retrobulbar neuritis as a local manifestation of general disease, e.g., gumma, tuberculous deposit, inflammation of a gouty or rheumatic character, near the junction of the nerve with the sclerotic. He also emphasized the frequency with which a gouty history is found, these cases being distinguished by a tendency to recurrence, the sciatic and seventh nerve being sometimes affected as well. Sir W. Gowers, in the course of the subsequent discussion, also laid stress on the importance of gout as a cause.

(3) When the optic nerve suffers as part of the rest of the central nervous system, e.g., in insular sclerosis. This part of the subject was dealt with by Dr. Buzzard. He quoted Uhthoff's statistics of 100 cases of disseminated sclerosis, which gave roughly 50 per cent. with a varying degree of change in the optic nerves. Buzzard also found about 50 per cent. of cases with involvement of the nerve, in 70 per cent. the change being bilateral, though the amount of visual disturbance varied enormously.

Since that time the importance of acute retrobulbar neuritis as an early sign of disseminated sclerosis has been pointed out by many authors.

Recently Tarlet† undertook a research to determine how far it could be shown that already at the time of onset of the acute

* *Trans. Ophthalm. Soc. U.K.*, Vol. XVII, 1897.

† Abstracted in *The Ophthalmoscope*, September, 1916.

retrobulbar neuritis there was other neurological evidence of concurrent disease of the central nervous system, especially disseminated sclerosis. Of his 29 acute cases of retrobulbar neuritis, two-thirds were in females, and more than two-thirds between 15 and 30 years of age, the average age of onset being 27. In eleven of the cases nervous symptoms were present which, with the eye condition, made the diagnosis of disseminated sclerosis certain; in others the diagnosis was probable. He confirmed the view that acute retrobulbar neuritis is an early and important sign of disseminated sclerosis, and that in one-third to one-half of the cases, there is already, at the time of onset of the attack, other evidence pointing to the existence of disseminated sclerosis.

Shumway* draws attention to the same point, but at the same time points out that a probable diagnosis of disseminated sclerosis should only be made if toxic, hereditary, acute or chronic infective processes attacking the optic nerve, can be excluded.

The other cause of retrobulbar neuritis, which has come much into prominence since the above-mentioned discussion took place, is that caused by inflammation of one or other of the nasal sinuses, especially the sphenoidal or posterior ethmoidal.

Marcus Gunn in the original discussion, referring to cases caused by sinus trouble, said that both nerves were often involved, and changes in the disc were late. The vision was much affected at first, but with a tendency to recovery, though this was rarely complete.

In April, 1908, the greater part of *The Ophthalmoscope* was devoted to this subject. Amongst others, papers were contributed by St. Clair Thomson, Howell Evans, and J. Jameson Evans. J. J. Evans concluded that the possibility of nasal disease should be borne in mind in all cases of retrobulbar neuritis and especially in cases with unilateral or bilateral restriction of peripheral visual fields, and he observed that the relief of a nasal empyema did not necessarily lead to recovery of sight.

Birch-Hirschfeld (*Archiv f. Ophthalm.*, May, 1907) states that in inflammation of a posterior sinus, sphenoidal or posterior ethmoidal, impairment of vision or blindness due to involvement of the optic nerve is apt to occur, and is more common than usually thought, and that the ocular symptoms may be the first to draw attention to the trouble. He found that the primary impairment of vision seemed to be constantly a relative central scotoma with intact outlines of the fields. The principal points of difference from the clinically somewhat similar toxic or infectious cases of neuritis were: the unilateral affection, although bilateral cases are also recorded, and the relatively acute onset

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**Ophthalmic Record*, 1915.

and progress, the relative scotoma becoming absolute and the field secondarily contracted.

In order to form some idea of the prognosis in these conditions, I have looked up the notes of the last 62 cases that we have had at the Oxford Eye Hospital. Of these 32 occurred in females and 30 in males. Taking the female cases first, as regards age of occurrence:—

Under 10	=	1.
„ 20	=	4.
„ 30	=	11.
„ 40	=	11.
„ 50	=	3.
„ 60	=	2.

that is to say, that 22 out of the 32 occurred between the ages of 20 and 40.

I have divided up the cases into acute, by which I mean those coming on rapidly, with great loss of sight, pain on pressure and on movement, semidilated pupil with sluggish or “curtsey” reaction, the vision being usually reduced to counting fingers or at any rate not more than 6/60. And, secondly, slight or subacute, those cases in which vision is found to be reduced usually to somewhere about 6/18.

Out of 32 cases in females—The right eye was affected 12 times; the left eye was affected 13 times; both eyes seven times; but in one of these cases there was an interval of three years between the two attacks.

Twenty-three of the attacks were acute.

Nine of the attacks were slight.

Of the 23 acute attacks, 15 of the cases improved to 6/9 or 6/6 and this in spite of the fact that in some cases distinct optic atrophy was present. Two cases improved to 6/12. One case improved to 6/18. One case improved to 6/60 in three months. In four cases the result was unknown.

Thus the prognosis as regards recovery of sight after an acute attack in a female is quite good.

Of the nine slight cases, recovery was practically complete in six, one improved to 6/12, and in one case, a woman with severe haemorrhage at the monthly periods, there was no recovery, and in one the result was unknown. Thus in the slight cases, too, the prognosis is good. As regards the length of time required for recovery to take place, the period varies from 10 days to four months. It is rather curious that in 12 cases out of the 32 the time taken was almost exactly three months.

Finally, as regards the permanency of the recovery, one case improved to 6/9, in three years later vision had dropped to 6/18. Another case improved to 6/12 with V. still 6/12 twelve years later.

Another bilateral case was still R. 6/9, L. 6/12, seven years after the attack.

As to causation, in only one case was disseminated sclerosis a probable cause. Three cases were attributed to a bad cold in the head. In no case was sinus trouble definitely diagnosed, though by no means all were examined by a nasal specialist; one case was diagnosed as ? sinus. One case recurred in the second pregnancy, having had a similar attack in her first. In one case very septic teeth were noted. One case had marked anaemia. One case had had diphtheritic cycloplegia some little time before.

In the majority of the cases no suggestion of any cause could be found.

The disease as affecting the male sex seems to me to be a much more serious and severe complaint.

Of the 30 cases in men, both eyes were affected in 17 cases, as compared with the 7 in women. In 8 cases the right eye only was attacked, the left eye in 5.

20 of the cases were of the acute type; in 7 the onset was more gradual; whilst only 3 were of the slight variety. The ages at which the disease occurred are:—

Under 10	=	1.
„ 20	=	3.
„ 30	=	11.
„ 40	=	10.
„ 50	=	2.
„ 60	=	0.
„ 70	=	3.

That is to say, 21 out of 30 cases occurred between 20 and 40, resembling in this particular the disease as it occurs in women. Next as regards the prognosis, in women one found a large proportion of the cases recovered practically normal vision. Of the 20 acute cases 7 recovered to 6/9 or 6/6, 3 to 6/12, 1 to 6/24, 1 result unknown, whilst 8 cases showed no improvement, and were practically blind. Of the 7 cases with gradual onset, 4 showed no improvement, whilst 3 regained 6/9. Of the 3 slight cases, 2 regained 6/9, 1 result unknown. Then as regards causation.—One, aged 22, had symptoms of disseminated sclerosis present at the time of onset; both eyes were involved. One, aged 37, had developed staggering one year previously. One, aged 27, developed disseminated sclerosis five years later, both eyes being affected, with an interval of one year between the two attacks. One, aged 20, three years later developed some spinal trouble, the knee jerks being absent. One case was attributed to a cold in the head. One case occurred after operation on the nose and one after discharge from the nose, sinus cases (?) One case had had indigestion, (?) tobacco helping. One case had malaria. One case had

gonorrhoea. One case was the subject of "fits." One case was attributed to a fall. One case was attributed to a blow. Of the remaining 17 there was no cause found, and nothing to which the attack could be assigned. No history of any family eye trouble was elicited, neither was gout nor rheumatism a prominent symptom.

(2) *Hereditary optic atrophy*.—Habershon (*Trans. Ophthal. Soc. U.K.*, 1888) read a paper on this subject, and recorded numerous cases. The commonest form is that originally described by Leber. This type occurs almost exclusively in males, though frequently transmitted through the females. The age of onset is usually a few years after puberty, viz., 18 to 23. In cases occurring in women it tends to develop either at puberty or the menopause. The loss of vision is a central amblyopia, with a complete central scotoma. Habershon was inclined to attribute it to a family tendency, associated in some cases with excessive use of tobacco and sexual excesses.

Since that time not much had been added to our knowledge of the causation of the condition until 1916, when J. Herbert Fisher read a paper at the Congress of the Ophthalmological Society, in which he suggested that the lesion was caused by an inherited temporary disorder of the pituitary body, which implicated the visual pathways. He found support of his theory in the X-ray photographs, which seemed to show some abnormal condition of the pituitary body. This theory yet awaits confirmation; all one can say at present is, that it is a very suggestive one, and should be carefully followed up.

(3) *Toxic amblyopia or chronic retrobulbar neuritis*.—Collins and Mayou* state that the poison either acts directly on the ganglion cells of the retina or their synapses, or by the production of ischaemia which interferes with their nutrition. Other observers have regarded the changes in the ganglion cells as secondary to a retrobulbar neuritis.

The poisons which may affect the sight are either endogenous or exogenous, endogenous being found in connection with diabetes, uraemia, or the puerperal state; whilst the exogenous toxins are tobacco, methyl or ethyl alcohol, and numerous other substances.

The commonest poisons we have to deal with in this country are tobacco and alcohol.

A doubt has been expressed whether tobacco cases ever really recover. Collins and Mayou state that the recovery, which usually takes place on discontinuing the use of tobacco, may be delayed or not occur at all in cases in which there is much vascular sclerosis. Another frequent cause for failure, anyhow amongst hospital patients, is the difficulty in getting the patients absolutely to

* Pathology and Bacteriology, 1911, p. 197.

give up the poison. They reduce the quantity or give it up for a time and improve, and then they start again, and they find out the amount that they can smoke and just continue to do their work.

One fact about tobacco cases that is not usually mentioned is the great difference it makes in the general health when they really give up the drug. They do not recognize how bad they are in themselves until they give it up and then they feel different men and once more are able to eat a decent breakfast; the persistent inability to eat a good breakfast in a strong, healthy man being a very suspicious symptom, in my experience.

I have looked over notes of 55 cases of tobacco amblyopia, seven of them undoubtedly complicated by alcohol. My idea of a fully developed tobacco case is a full peripheral field with a central scotoma for red, extending from the blind spot to the fixation point, of definite oblong shape, and hardly extending above or below the fixation point.

When the scotoma is larger than this or assumes a circular form diabetes or other complication may be suspected. Of course in earlier cases one gets the para-central relative scotoma for red, starting from the blind spot and extending towards the fixation point, then gradually spreading until this is involved, becoming absolute for red and, lastly, for white too. This fact seems to favour the retrobulbar theory rather than a macular poison, as J. Jameson Evans has pointed out. Where there is contraction of the peripheral field as well, one may suspect alcoholic poison in addition to tobacco.

Prognosis

Of 39 cases out of the 55 that continued to attend for some months, 15 of them reached to practically complete recovery, their vision being 6/9 or 6/6, the time taken varying from 3 months to 11 months. In 12 of the cases recovery was fair, and the men, being probably content, attended no more. In 9 cases there was some slight improvement; in 3 cases no improvement at all, but they did not attend for very long. So, taking the complete recoveries and the fair cases as capable of complete recovery, one finds, roughly speaking, that 27 at least out of the 39 would recover, though I suspect that this is too low an estimate.

There is one case that puzzles me amongst those I have investigated. When first seen he was undoubtedly suffering from alcohol and tobacco poisoning, and his vision was 6/60 each eye; he was drinking half a gallon of beer a day, and smoking 1 oz. a day. Three years later he was smoking 1 oz. a week and drinking three pints a day, but his vision was still 6/60 each eye. Again

7 years later his vision was R. 6/18, L. 6/9, but, according to my house surgeon's notes, he was smoking $1\frac{1}{2}$ ozs. a day and drinking three pints of alcohol. So this man seems to have practically recovered without entirely giving up the poisons and to have resumed excessive use of them without suffering from it.

That patients can resume smoking without bringing on the trouble again, I know from cases that I have had in my private practice, and I believe that tobacco, as a rule, only gets its chance to cause trouble when associated with complete running down of general health, often associated with insufficient food, mental worries, or financial troubles, all tending to produce a depressed state of the body, and rendering it more liable to the action of the poison.

TRANSLATION

THE OCULAR COMPLICATIONS OF LETHARGIC ENCEPHALITIS*

BY

V. MORAX

IN view of the number of cases of so-called "Botulism" with ocular complications reported in this country of late years the following translation may interest readers:

At the meeting of March 22, 1918, of the Medical Society of the Paris Hospitals, Dr. Netter drew the attention of his colleagues to a peculiar nervous affection which, in addition to other disturbances, was principally characterized by a state of prolonged somnolence: whence the name "lethargic encephalitis" under which it was described by Economo in 1917. By reason of the interest and importance of the ocular complications observed in sufferers from this affection it has seemed to me useful to give a short account of the general symptoms of the disease and to enumerate the eye complications.

Netter's clinical description is as follows: "The disease, which usually runs a febrile course, begins with headache and sometimes with vomiting. Somnolence, which becomes more and more pronounced, appears rapidly. To begin with there is merely drowsiness; this is followed by true sleep from which one can awake the patient, who replies reasonably and walks without hesitation, but soon passes into slumber. At a more advanced stage

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*Translated from *Annales d'Oculistique*, July, 1918.