COMMUNICATIONS

SOME NEURO-OPHTHALMOLOGICAL PROBLEMS*

BY

ALAN MOONEY

Neurosurgical Unit, Richmond Hospital, Dublin

HAVING been associated with the Neurosurgical Unit of the Richmond Hospital for 25 years, I feel that the time is opportune to cast my memory back and recall some of my experiences which may be of interest.

If I succeed in making it clear to the neurosurgeons that the ophthalmologist has a useful contribution to make in the assessment of many of their problems, and to the ophthalmologist that he should be familiar with the neurosurgical implications of certain ocular findings, this paper will have served a useful purpose. To stress the fact that these problems are real—they are a frequent source of anxiety to me—I propose to give just one example of each, although many more are available. The ophthalmologist has an important place in the team, being often the last line of defence the patient must pass before entering the operating theatre. To emphasize this I propose to describe two cases: in the first I was at fault in advising an unnecessary operation and in the second a young girl became incurably blind because an operation was not performed in time.

Case 1, a woman aged 48 years, was complaining of failing vision. There was no toxic history and investigations were negative. This was in 1935 before the days of angiography. Visual acuity was 2/60 in each eye, with slight temporal pallor of each disc.

Perimetry showed bilateral central scotoma embracing the blind spot (Fig. 1). On my advice a right transfrontal exploratory operation was performed. The optic nerves were pale and to the outer side of the right nerve was a hard artery with a bulge on its wall. No tumour was found. The patient died 11 days later.

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Fig. 1.—Case 1, visual fields.
ALAN MOONEY

Recommending operation in this case was a serious error of judgment on my part. To-day angiography would be performed on a similar case, and in the event of its being normal, operation would, at least, be deferred.

With all the modern diagnostic aids which are available to-day a negative craniotomy is difficult to justify, but on the other hand it is equally indefensible to allow a patient with papilloedema to become blind without first excluding a space-occupying lesion.

Case 2, a girl aged 17 years, had been seen elsewhere 4 years previously with papilloedema of 2 dioptres and a diagnosis of optic neuritis had been made. When she was referred to the brain unit the visual acuity was less than 6/60 in each eye with left homonymous hemianopia and papilloedema of 4 dioptres with some pallor of each disc. The girl was ataxic with partial deafness in the right ear. A skull x ray and electroencephalogram were negative.

Ventriculography disclosed internal hydrocephalus, the third ventricle showing, and at operation a right acoustic neuroma was removed.

The patient is now blind from secondary optic atrophy.

This case brings up the question how long one can allow papilloedema due to a space-occupying lesion to be present without running the risk of blindness from secondary atrophy. Apparently it took 4 years to develop in this patient but I feel that this must be quite exceptional. My impression is that, if papilloedema is steadily increasing, especially when it reaches 4 dioptres, and a space occupying-lesion is suspected, operation should be performed without delay. I have never sought an answer to the time factor because, in order to provide it, one would have to wait until fibrosis had begun and then it is generally too late to save the vision.

CONGENITAL DISC

It is not unusual for a patient to be referred to the Neurosurgical Unit on account of headaches and papilloedema on the assumption that a tumour is present. If the discs are pathological and the patient is not hypertensive, this diagnosis would probably be correct. On the other hand, having regard to the important implications of this finding, the ophthalmologist must be quite sure that the patient has papilloedema and not a congenital anomaly.

Case 3, a woman aged 24 years, a postulant in a religious order, was admitted to the Richmond Hospital on account of severe headaches, vomiting, and blindness, with a history of head injury 4 years previously. It was reported that she had papilloedema of 2 dioptres.

When examined by us she said that she could not see light with either eye but the pupils reacted briskly to light. While the discs appeared swollen, the presence of colloid bodies at their margins and the fact that the florid appearance usually associated with papill-oedema was absent, suggested that we were dealing with a congenital anomaly. We also concluded that the blindness was psychogenic in origin. Ventriculography was negative.

A burr hole was made in the right frontal region where the headache was most intense. About 16 ml. subdural fluid was removed and the eyes were bandaged for 48 hours. When the bandages were removed the patient appeared to see normally and the headaches had disappeared. The appearance of the discs did not change.
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These "full" discs are by no means rare and are sometimes, but not always, associated with high hypermetropia. The presence of colloidal bodies at the disc margin, fibrosis on its surface, and the absence of both a florid, congested appearance and darkening of the veins after they pass over the disc margin should arouse doubts that the condition is pathological.

A certain amount of blurring of the upper disc margin is very common and when associated with headaches should be carefully assessed.

I do not believe that it is possible to give a definite opinion in a large proportion of these cases and my usual procedure is to state that the disc is within normal limits and should be re-examined in a week. If it is then unchanged I repeat the examination 2 weeks later, and if there is no alteration after a further 4 weeks I pass the disc as normal. Further examination is necessary only if the headaches persist after a further 3 months.

Although the vision was not at any time affected, the same point about the discs arose in the following patient:

Case 4, a girl aged 16 years, also a postulant, complained of headaches, and in France, where she was resident, papilloedema was diagnosed. This was subsequently confirmed in England where signs of early secondary optic atrophy were found, and a diagnosis of posterior fossa tumour was made. She was referred to the Richmond Hospital because she wished to return to Ireland.

On examination the discs presented the "full" appearance previously described, and neurological examination and ventriculography were negative.

The headaches cleared up when she returned home, the discs remained unchanged for over 6 months, and she has been well ever since.

HYPERTENSIVE DISCS

Papilloedema due to hypertension is not uncommon and occasionally patients with this complaint are referred to the Neurosurgical Unit for operation.

Case 5, a patient aged 19 years, was referred to us on account of headaches, vomiting, and dimness of vision. Bilateral papilloedema was noted on admission and the electroencephalogram suggested an intracranial lesion, probably in the mid-line or posterior fossa. Fundus examination showed bilateral papilloedema of 4 dioptres, apparently of long standing, with some pallor of the disc surface. Arterial constriction, venous congestion, and well-marked retinopathy were present.

The patient was obviously hypertensive (the blood pressure was subsequently found to be 220/150) with albuminuria. I was in some doubt whether the pallor of the disc surface was due to exudation or to early fibrosis, and, while expressing the opinion that the patient had hypertensive retinopathy, I did not completely rule out the possibility of a superimposed space-occupying lesion. This was excluded, however, without resorting to ventriculography, and 8 months later, after medical treatment, the fundi were normal, except for vasoconstriction, and the patient was symptom free. The blood pressure and urine were unchanged.

Similar findings due to a different cause were seen in the following case:

Case 6, a man aged 38 years, had had during the previous 4 months severe bouts of headache, sometimes associated with vomiting and weakness of the legs.

At the time of admission to hospital the blood pressure was 220/140 and there was a
trace of albuminuria. The electroencephalogram showed some diffuse abnormality with no focal features, the appearances being comparable with those found in malignant hypertension.

There was well-marked hypertensive retinopathy and papilloedema of 5 dioptres. The discs were very florid in appearance and, taken by themselves, were more suggestive of intracranial than of vascular hypertension. Ventriculography showed internal hydrocephalus with a patent aqueduct.

At operation a large tumour which proved to be a metastatic carcinoma was removed from the left cerebellar hemisphere (Fig. 2).

The foregoing cases underline the problem frequently presented by the "swollen disc". Is it congenital, hypertensive, or due to a space-occupying lesion? I do not propose to repeat observations made by ophthalmologists more experienced than I, but I must say that the more discs of this type I see the more reluctant I am to express an immediate opinion, since diagnostic errors may occur as a result of making only one examination.

When a patient is suffering from headaches, without neurological signs and with blurred discs, there is generally no urgency, and repeated fundus examination will often avoid the necessity for ventriculography or even craniotomy. It is much better that the ophthalmologist should admit that he does not know than that a patient should be submitted to either of these procedures in error through a hasty diagnosis.

PAPILLOEDEMA IN TUBERCULOUS MENINGITIS

We are confronted with a different, and comparatively new problem, when a patient with tuberculous meningitis develops choked discs, since papilloedema from this cause is amenable to neurosurgery.

Case 7, a woman aged 21 years, had been ill for 10 weeks with tuberculous meningitis, and there was papilloedema of 3 dioptres in both eyes with one choroidal tubercle. The oedema increased to 4 dioptres in the course of a week, neurosurgery was advised, and ventriculography showed internal hydrocephalus.
At operation the optic nerves were found to be surrounded by granulation tissue (Fig. 3). This was removed, a third ventriculostomy was performed, and 2 months later the discs were flat and have remained so since then.

The next case, which had a less favourable outcome, underlines the danger of assuming, as has been stated from time to time, that papilloedema from meningitis does not develop secondary atrophy.

**Case 8, a boy aged 7 years**, was admitted to the Richmond Hospital, after having been under treatment for tuberculous meningitis in a County Hospital during the previous 11 months. Fundus examination showed bilateral papilloedema of 4 dioptres in highly hypermetropic eyes. There were signs of beginning secondary optic atrophy and the vision was low. The skull was hydrocephalic and the sutures were wide open.

Operation disclosed fine soft adhesions round the optic nerves. These were removed and a short circuit done on a dilated third ventricle. Just before his discharge from hospital 4 weeks later the discs were flat but well-marked secondary atrophy was present and the visual acuity was practically nil.

There can be very little doubt that a third ventriculostomy, done in time, would have saved this child’s sight. The following case shows, however, that conservative treatment is sometimes justified.

**Case 9, a woman aged 20 years**, suffering from tuberculous meningitis, had double sixth-nerve palsy and 2 dioptres of papilloedema in highly hypermetropic eyes. There were eight choroidal tubercles in the right fundus and five in the left. The discs had not the florid appearance found in the presence of internal hydrocephalus due to other causes but presented a “serous” change such as I have sometimes seen with external hydrocephalus. A chest x-ray showed miliary tuberculosis.

Daily spinal punctures, together with the usual treatment of Delta-Cortryl, intramuscular streptomycin, P.A.S., and I.N.A.H. were advised.

The papilloedema subsided in 4 weeks, by which time the ocular palsies had also cleared up, and the patient has since made a complete recovery from the meningitis and miliary tuberculosis.

**Optic Neuritis**

To bridge the gap between the problem of the swollen disc and that of failing vision, the next case showed the not unusual condition of optic neuritis of unknown origin:

**Case 10, a woman aged 36 years**, gave a history of 5 days’ bilateral visual failure and of influenza one month previously.
When she was first seen there was no perception of light in either eye, the pupils were non-reacting, and there was papilloedema of \(1\frac{1}{2}\) dioptres with congested retinal veins. The discs did not appear florid. Other investigations, including angiography, were negative.

A course of systemic cortisone and chloramphenicol was given and the swelling subsided in 10 days; 5 months later the visual acuity was counting fingers at 6 metres in each eye and the visual fields showed bilateral central scotoma (Fig. 4).

The vision gradually improved and 1 year and 8 months later it was normal in each eye, the fields being within normal limits, and there was bilateral Grade 2 pallor of the discs with clear margins.

This bilateral condition is not rare in middle-aged patients, and generally a cause is not discovered. Some make an almost complete recovery while others remain blind, both on the same treatment. The rapid visual failure, the appearance of the swollen discs, and the negative angiogram should insure against the risk of these patients being submitted to neurosurgery.

**Case 11, a woman aged 48 years**, complained of headaches and failing vision in the left eye of 2 months' duration, and 4 months before that loss of the sense of smell and taste during a severe cold. The electroencephalogram was negative, and visual acuity 6/6 in the right eye and counting fingers at 1 foot in the left eye. Papilloedema of \(1\frac{1}{2}\) dioptres was present in the right eye, the left disc being normal. The right visual field showed a slight upper temporal quadrant defect, the left an inferior hemianopia with large macular loss (Fig. 5).

A left antero-posterior arteriogram showed stretching and displacement of the anterior cerebral artery round a space-occupying lesion in the inferior part of the frontal lobe (Fig. 6, opposite).

A lateral arteriogram showed downward dislocation of the siphon, the anterior cerebral artery being stretched and displaced backwards round the posterior aspect of a space-occupying lesion (Fig. 7, overleaf).
At operation a large left-sided meningioma growing from the cribriform plate was successfully removed.

While the diagnosis of retrobulbar neuritis might have been made in the case of the left eye, the presence of normal vision and the absence of scotomatous changes would hardly justify the application of the term optic neuritis to the right. Positive angiography removed any doubts about the diagnosis in this case just as the negative findings did in the previous one.

I should like to draw attention to the compressed siphon which, in 1948, we put forward as a possible cause of the central scotoma in these cases.
A high refractive error may obscure the presence of a lesion affecting the lower visual pathway.

Case 12, a man aged 48 years, had complained of failing vision for 2 years, the right eye having become rapidly worse during the past 6 months. He had always been "short-sighted" and had recently consulted a few specialists who had changed his glasses. The visual acuity with correction was perception of light in the right eye and 6/12 in the left. Each eye had a high refractive error, the right more than the left. The right fundus showed Grade 1 pallor of the disc with a shallow complete cup, and the left disc was normal. The intra-ocular pressure was normal.
Perimetry disclosed a left-sided temporal hemianopia, most marked in the upper quadrant (Fig. 8).

**Fig. 8.**—Case 12, visual fields.

A right antero-posterior angiogram showed the proximal part of the anterior cerebral artery to be tilted upwards and badly filled (Fig. 9).

**Fig. 9.**—Case 12, right antero-posterior angiogram.

A lateral angiogram showed the siphon to be lifted upwards and forwards (Fig. 10, overleaf). At operation a chromophobe adenoma was found beneath the chiasma and right optic nerve, extending lateral to the internal carotid artery, which was embedded in a deep groove (Fig. 11, overleaf). This middle fossa extension was bulky and over its upper pole were stretched the middle and anterior cerebral arteries. The tumour was partially removed.
Fig. 10.—Case 12, lateral angiogram.

Fig. 11.—Case 12, chromophobe adenoma beneath the chiasma and right optic nerve.
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Before the right eye became blind it would have been very difficult to make a correct diagnosis in this case unless perimetry was performed. It would be reasonable to expect a certain amount of amblyopia in each eye, especially the right, because the refractive error had not been corrected by means of suitable glasses in childhood. Defective vision caused by disuse was seen again in the following case:

Case 13, a female acromegalic, aged 52 years, was admitted complaining of headaches and failing vision. Her right spectacle lens was correct and with it the visual acuity in the right eye was normal. For the left eye she was wearing only a simple lens to correct a complex refractive error, and with a correct lens the visual acuity in the left eye was only 6/24. The discs were normal. The right visual field was normal and the left showed temporal hemianopia to small visual angles (Fig. 12).

There was ballooning of the sella with erosion of the floor and dorsum and undercutting of the anterior clinoids. The antero-posterior and lateral arteriograms were normal.

Deep x-ray therapy restored the left visual field to normal but failed to improve the defective central vision of this eye which was due to an uncorrected refractive error. The complaint of recent visual failure was due to the onset of presbyopia and not to any affection of the visual pathway.

This case also underlines a common experience with acromegals, namely, normal discs, minimal field changes, and a normal anterior cerebral artery in the antero-posterior arteriogram.

RETROBULBAR NEURITIS

Chronic retrobulbar neuritis is a diagnosis that should seldom be made.

Case 14, a woman aged 44 years, complained of failing vision in the left eye for 18 months and in the right eye for a few weeks. The visual acuity was 2/60 in the right eye and counting fingers at 2 metres in the left.

The right disc was normal, and the left showed Grade 2 pallor, with clear margins and normal vessels.
The right visual field showed incomplete inferior hemianopia with macular loss, and the left a temporal hemianopia with macular loss (Fig. 13).

A skull x ray disclosed nothing abnormal.

At operation a meningioma of the tuberculum sellae, the size of a walnut, was found to be displacing the left optic nerve (Fig. 14).

It had originally been suggested that was an early case of disseminated sclerosis, and a similar diagnosis came to my mind when I first saw the following patient:

**Case 15, a man aged 29 years,** gave a history of failing vision in the left eye of 9 days’ duration. The visual acuity was 6/9 in the right eye, and hand movements (in the peripheral field only) in the left. The left pupil reacted sluggishly to light. The fundi were normal.

He was admitted to hospital as a case of acute retrobulbar neuritis and treated with systemic cortisone, a skull x ray disclosed an enlarged sella with destruction of the floor and thinning of the dorsum.

Perimetry, done 6 days later, showed bitemporal hemianopia, the visual acuity being then 6/9 in both eyes (Fig. 15, opposite).

The left antero-posterior angiogram showed upward displacement of the anterior cerebral artery (Fig. 16, opposite). The lateral view showed that the siphon termination was elevated (Fig. 17, overleaf).

Radiotherapy effected a satisfactory visual improvement and 1 year later the visual acuity and fields were normal.
Fig. 15.—Case 15, visual fields.

Fig. 16.—Case 15, left antero-posterior angiogram.
Two lessons may be learnt from this case, the first being that pituitary tumours may simulate retrobulbar neuritis and the second that deep x ray is indicated where bitemporal hemianopia is present with normal discs.

There is considerable operative risk in these cases as the tumour is chiefly retrochiasmal and an attempt at even partial removal may disturb the hypothalamus sufficiently to cause death. No such risk is involved in modern radiotherapy and Case 15 shows that the result may be satisfactory if the treatment is given early enough. This case also demonstrates that a central scotoma may be caused by the opening out of the siphon as well as by its compression.

**OTHER MISLEADING SIGNS**

In Case 16 an unverified subchiasmal tumour simulated a retinal vascular lesion:
Case 16, a woman aged 32 years, stated that one year previously she had noticed that the vision of her left eye was defective. As there was pallor of the left disc, an irregular inferior nasal retinal artery, and a corresponding upper temporal field defect on the same side, a diagnosis of occlusion of the inferior nasal artery of the left retina was made.

Subsequent ophthalmic examination showed the visual acuity to be 6/5 in the right eye and 6/24 in the left. The right disc was just off normal colour, and the left showed Grade 1 pallor. Perimetry showed bitemporal hemianopia affecting chiefly the upper quadrants (Fig. 18).

A skull x ray disclosed an enlarged sella with erosion of the dorsum and floor. A left antero-posterior angiogram showed upward displacement and slight narrowing of the anterior cerebral artery in its proximal part (Fig. 19).
The lateral view showed backward displacement of this artery with slight uplift of the siphon termination (Fig. 20).

Both operation and radiotherapy were refused by the patient.

My impression is that where there is bitemporal hemianopia with pallor of one or both discs, surgery is advisable. Even when there is erosion of the sella, meningioma cannot be completely excluded, and if a tumour of this nature is given radiotherapy without improvement and operation is subsequently performed, there is some risk of monococular blindness, as was indeed our experience on one occasion. In the case of adenoma, removal of sufficient tumour to free the optic nerves and no more, followed by radiotherapy, appears to be the ideal treatment. A less conservative approach gains little or nothing and increases the risk of post-operative mortality. We have found that pallor of the disc without scotomatous
changes is usually due to lateral displacement of the optic nerve and is associated, in the majority of cases, with the lifting up of a badly-filled proximal part of the anterior cerebral artery.

The symptomatic onset of pituitary tumours takes various forms and led in Case 17 to a diagnosis of Adie's syndrome.

Case 17, a woman aged 29 years, gave a history of diplopia and severe right-sided headache of 18 months' duration. The right pupil had become enlarged and the vision of this eye blurred before our examination. Visual acuity was normal in each eye. There was right-sided semi-mydriasis and accommodation weakness. The discs were normal.

Perimetry showed that left homonymous hemianopia was present for small visual angles (Fig. 21).

A skull x ray showed gross destruction of the floor and dorsum sellae.

A right antero-posterior angiogram disclosed upward displacement of a badly-filled anterior cerebral artery (Fig. 22, overleaf).

The lateral view showed a widely-open siphon (Fig. 23, overleaf).

Radiotherapy resulted in the visual fields and accommodation returning to normal, but the pupil was unaffected. The pain and diplopia disappeared.

This case demonstrates that there can be a widely-open siphon without scotomatous changes and a badly-filled proximal part of the anterior cerebral artery without disc pallor. Possibly these signs, especially the pallor, only develop when the vascular displacement has been present for some time.

Case 18 presented some signs suggestive of pituitary tumour.

Case 18, a woman aged 66 years, gave a history of intermittent pain in the distribution of the second division of the left trigeminal nerve. When the pain was present the vision of the left eye became blurred. The vision of the right eye had been failing gradually for 2 years.

The visual acuity was hand movements in the lower nasal quadrant of the right eye, and 6/9 in the left eye. Grade 2 pallor was present in the right disc, the left disc being normal.

Perimetry showed complete loss of the right field except in the lower nasal quadrant. The left showed slight temporal hemianopia (Fig. 24, overleaf).

The right anterio-posterior angiogram disclosed a large sacculated aneurysm projecting across the mid-line, and displacing the anterior cerebral artery upwards (Fig. 25, overleaf).

The lateral view showed the aneurysm appearing to come off the termination of the internal carotid and beginning of the posterior communicating artery (Fig. 26, overleaf).

The patient refused operation and 1 year later had Grade 3 pallor of the discs. The right eye was blind and she could only count fingers at 2 feet with the left, there being complete temporal hemianopia.
Fig. 22.—Case 17, right antero-posterior angiogram.
Fig. 23.—Case 17, lateral angiogram.

Fig. 24.—Case 18, visual fields.
At the Congress of the Ophthalmological Society of the United Kingdom in 1956, an ophthalmologist referred to a patient who had been operated on for pituitary tumour when he was in fact suffering from chronic glaucoma. Another speaker, possibly a neurosurgeon, countered this by describing a patient who had been treated for chronic glaucoma and who subsequently died, a pituitary tumour being disclosed at autopsy. The latter experience must surely be most unusual, but the former is quite understandable as is illustrated by Case 19.
Case 19, a man aged 43 years, had been blind in the right eye for some years as a result of chronic uveitis and secondary glaucoma. The corrected vision of the left eye was normal, but there was marked contraction of the visual field with pallor of the disc. He was referred to the Neurosurgical Unit on account of the possibility of intracranial trouble. The visual acuity was nil in the right eye and normal in the left. The right eye was soft with an oculated pupil, and the left showed a few fine keratic precipitates, Grade 2 pallor of the disc with shallow cupping; the ocular tension was 30 mm. Hg, and rose to 40 mm. Hg 1·5 hours after the instillation of homatropine. The left visual field showed marked
nal contraction with general depression (Fig. 27). A chest x ray suggested sarcoidosis.

Cortisone, I.N.A.H., and P.A.S. with topical Pilocarpine and Neo-Cortef improved the uveitis and brought the ocular tension down to 24 mm. Hg.

My impression was that the appearance of the disc and field changes were due to periodic rises in the ocular tension due to uveitis, and as it was not a straightforward case of glaucoma I considered that it was a wise precaution to refer the patient to the brain unit for neurological investigation which, however, turned out to be negative.

TOXIC AMBLYOPIA

That the gradual visual failure may give rise to diagnostic error is demonstrated by Case 20.

Case 20, a man aged 43 years, who had been treated for tobacco amblyopia, gave a history of the left eye having been defective from childhood and failure of the right vision for 3 years. He had consulted two ophthalmologists who had advised him to stop smoking and prescribed tablets. When he was first seen the visual acuity was nil in the right eye and 6/60 in the left.

The eyes were hypermetropic, the left being worse than the right. A slight left internal concomitant squint was present with defective vision from disuse.

Perimetry showed temporal hemianopia with macular sparing in the left eye (Fig. 28).

Grade 2 pallor was present in the right disc, the left being just off normal colour. It was not possible to do a pre-operative angiography.

At operation a tumour closely associated with a pulsating swelling was found to be lifting up the right optic nerve (Fig. 29, opposite). As it was considered inadvisable to remove the tumour the operation was terminated.
Fig. 29.—Case 20, tumour closely associated with pulsating swelling found to be lifting up the right optic nerve.

A right antero-posterior arteriogram showed some lifting up of a badly-filled anterior cerebral artery and lateral displacement of the internal carotid (Fig. 30).

Fig. 30.—
Case 20,
right antero-po-
tero-po-
terior arterio-
gram.
The lateral view showed some elevation of the siphon termination (Fig. 31).

After a course of radiotherapy the visual acuity in the left eye was 6/36 with a more suitable correction of the refractive error. The visual field was unchanged.

Case 21 is the counterpart of Case 20:

**Case 21, a woman aged 63 years**, who complained of failing vision especially for near work during the previous 6 months, was referred to the Neurosurgical Unit as a probable case of pituitary tumour on account of defective vision, bilateral pale discs, and inconclusive fields.

A skull x ray showed an extremely thin dorsum sellae, and the visual acuity was 6/36 in the right eye and 2/60 in the left.

There was doubtful pallor of the temporal half of each disc and macular degeneration in the left eye.

The visual fields showed a bilateral centro-caecal scotoma (Fig. 32, opposite).

This lady came from County Kerry and on being asked if she smoked a pipe admitted to smoking 3 oz. of strong tobacco each week. She was advised to stop smoking and sent home. She could not return to Dublin but a letter received from her 6 months later made it clear that she had made a complete visual recovery.
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Fig. 32.—Case 21, visual fields.

PALE DISCS IN TUBERCULOUS MENINGITIS

In the early part of this paper I referred to the problem of papilloedema in tuberculous meningitis. The treatment of visual failure in these cases also presents difficulties.

Case 22, a woman aged 29 years, was admitted to Dublin Fever Hospital with headaches of 5 weeks' duration. A diagnosis of tuberculous meningitis was confirmed by guinea-pig inoculation.

She was given intra-thecal and intra-muscular streptomycin, I.N.A.H., P.A.S., and Delta-Cortryl. Within 10 weeks the visual acuity of the right eye deteriorated to hand movements, with subsequent recovery. While this recovery was taking place, the sight of the left eye failed, with simultaneous development of right homonymous hemianopia. Both these defects recovered within 4 weeks. While these visual changes were taking place the discs, from being normal, gradually developed Grade 2 pallor in the right eye and Grade 1 in the left. The patient made an uneventful recovery and the ocular findings before her discharge, 8 months after admission, were normal vision in each eye and Grade 1 pallor in the right disc, the left disc being just off normal colour.

The visual fields showed a slight right upper homonymous quadrantopsia (Fig. 33).

Fig. 33.—Case 22, visual fields. Note that 20/0 should read 20/00 Red.

The next case presents a contrast with the foregoing:

Case 23, a boy aged 2½ years, was admitted to Dublin Fever Hospital with tuberculous meningitis of 2 weeks' duration, and was given the same treatment as Case 22. The child was blind, with inactive pupils and normal hypermetropic discs. There were no choroidal tubercles. As there was no change in the eye condition after 8 weeks, I advised neuro-surgery and this disclosed an excess of cerebro-spinal fluid in the subdural space.
The optic nerves were covered by very firm adhesions and in places there appeared to be granulation tissue (Fig. 34). A third ventriculostomy was performed after the adhesions had been removed.

Fig. 34.—Case 23, optic nerves covered by firm adhesions and granulation tissue.

Examination of the granulation tissue showed it to be tubercular (Fig. 35); 3 weeks after the operation the vision began to return and it was apparently normal within a month. There was at no time any apparent abnormality of the discs.

Case 24, a boy aged 3 years, was taken into Dublin Fever Hospital with tuberculous meningitis, having been under treatment in a County Hospital for 3 weeks where he was comatose on admission. He was found to be very emaciated, blind, and deaf, and as he was considered too ill for surgery he was given the usual treatment for 3 weeks and was then transferred to the Richmond Hospital.

At operation considerable communicating hydrocephalus was found, with adhesions round the nerves which bled following their removal (Fig. 36, opposite). A third ventriculostomy was performed and the patient returned to the Fever Hospital on the tenth day. With the exception of the Delta-Cortryl, the pre-operative treatment was continued. There was no visual improvement for over 4 months, the discs remaining very pale. The vision and hearing then gradually recovered and the boy now appears to see and hear very well and is physically fit. The treatment is being continued.

It may be asked why the first of these three patients recovered normal vision
without neurosurgery in spite of the discs being pale, why the second required
neurosurgery to restore vision, the discs being normal in spite of the eyes being
blind for 8 weeks, and why the third boy's vision recovered 4 months after operation,
the discs being very pale all the time?

These are just three examples of the difficult decisions that have to be made in
tuberculous meningitis, not to submit the patient to unnecessary surgery and not
to run the risk of irrecoverable blindness by postponing operation for too long.

SUMMARY

If we add up these 24 cases, each to exemplify a different problem, what is
the answer? I will try to give it in the following summary.

The ophthalmologist is an important member of any neurosurgical team.
His function is to collaborate with the other members in order to prevent an
unnecessary craniotomy or to avoid delay when surgery is indicated.

Familiarity with the anatomical variations of the normal disc and discrimina-
tion between the hypertensive disc and that due to a space-occupying
lesion is essential. It is important to be aware of the danger in allowing
secondary optic atrophy to develop even in tuberculous meningitis and also
of the type of oedema that will subside without surgical interference.

Correlation of the visual acuity, the fundus appearance, and the perimetric
findings should point towards the proper diagnosis, even if angiography is
required to finalize it. I am afraid that, in this country anyhow, perimetry
must be regarded as a pleasant pastime not covered by the economics of
ophthalmology, but it is important, not alone as a means of localizing lesions
in the chiasmal region but also, in conjunction with the appearance of the
discs, as a guide to treatment in such cases as the following:

1. Bitemporal hemianopia with normal discs suggests a retro-chiasmal
   lesion unsuitable for neurosurgery.

2. Central scotoma in one eye with temporal field defect in the other
   suggests a space-occupying lesion in the chiasmal region, either
   elevating or depressing the carotid siphon, usually the former, on the
   same side as the scotoma.

3. Bitemporal hemianopia, predominantly non-scotomatous, with some
disc pallor suggests that some, at least, of the tumour is pre-chiasmal,
displacing the nerves. Surgery is indicated to verify the nature of the tumour and, if it is an adenoma, to excise just sufficient to free the nerves; it should then have post-operative radiotherapy.

There is no doubt that pituitary tumours may press the optic nerves and chiasma upwards against the anterior cerebral arteries, thereby producing transverse notching of these structures, but the evidence that this notching is responsible for the field defects is not convincing.

Narrowing of the proximal part of the anterior cerebral artery is frequently, but not invariably, associated with the prechiasmal extension of pituitary tumours. This association may have some influence on the development of disc pallor, and possibly also on certain perimetric findings, the anterior cerebral artery being compressed between the optic nerves and chiasma below and the base of the frontal lobe above.

We have seen the anterior cerebral artery recover its normal calibre after the removal of tumour material and this may have some connexion with the recovery in the visual fields and slight improvement that frequently takes place in the colour of the disc post-operatively.

I have not solved the problem of defective vision in cases of tuberculous meningitis; some patients recover good vision after neurosurgery, and others recover without surgery. A third category, usually late cases, fails to improve after operation. Possibly compression of the pial vessels by adhesions determines the loss of visual function, and re-establishment of the blood supply after removal of these adhesions or the arrest of their development following modern therapy may account for the visual recovery.

I found no connexion between therapy and visual failure in these cases.

As patients with tuberculous meningitis are surprisingly good surgical risks, even when pyrexial, I feel that they provide, in the present state of our knowledge, a special exception to the rule that exploratory brain operations should not be advised by the ophthalmologist.

The Future

When B.C.G. inoculation and the rigid isolation of open chest cases have their full impact on tuberculosis, the ocular complications of tuberculous meningitis will disappear from the chiasmal scene.

Other diagnostic problems continue and fresh ones will probably appear. In order to deal with these effectively, more and still more accurate diagnosis, which will both pinpoint the lesion and determine its nature, will be essential.

Success will largely depend on an intimate anatomical knowledge of the visual pathways and their blood supply in relation to field defects and disc changes. When dealing with lesions in the chiasmal region, detailed perimetry, routine air studies, more refined angiography, and a reconciliation of the resulting findings should go far towards accomplishing this end.

My thanks are due to my colleagues in the Neurosurgical Department of the Richmond Hospital, Dublin, for their cooperation, and to Stella Ross for the operation drawings.
SOME NEURO-OPHTHALMOLOGICAL PROBLEMS

Alan Mooney

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