Some Neurological Aspects OF OPHTHALMIC CASES.

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I NEED hardly say that I feel greatly honoured by my election to the presidency of this section. I must, however, confess to a feeling of not a little diffidence in taking up the duties of the post. To be the first physician invited to preside over a section so largely surgical as this is no small honour, but it seems to me to carry with it great responsibility and may require great indulgence on your part. I do not think I am at fault in thinking that my election is to be regarded as a recognition of the desirability—nay, the necessity—of close coöperation between the ophthalmic surgeon and the physician; this necessity was impressed upon me very early in my neurological work, and was clearly recognised by the last generation. It is rather interesting in this connexion to remember that when Moorfields Hospital was first established in Charterhouse-square in 1804 it had as its staff a surgeon, Dr. Saunders, and a physician, Dr. Farre. Apparently the surgeon was not allowed to prescribe medicine, and the physician's duties consisted chiefly in ordering ointment and aperients, although he saw out-patients as the surgeon did. The elder Dr. Farre was succeeded as physician by his son, and he in turn by Dr. Martin, whose immediate successor was Sir Stephen Mackenzie, who was a colleague of mine for some years. The long service which the physicians were able to give is noteworthy. The elder Dr. Farre was on the staff for 50 years, his son for 37, Dr. Martin for 28, and Sir Stephen Mackenzie for 21. When Sir Stephen Mackenzie was appointed an era began in which cases seen by a surgeon were referred to the physician for medical report. This naturally led to investigation—especially ophthalmoscopic investigation—and Sir Stephen Mackenzie's work on the ophthalmoscopic changes observed in anæmias are familiar to all of us. It was in 1899 that I had the good fortune to be associated with him as physician to Moorfields, and it is interesting to know that every ophthalmic hospital in London now has a physician on its staff.

I wish to allude in a somewhat cursory way to some of the subjects in which ophthalmic surgeons and physicians have a common interest; and I should like to consider this under three headings: (1) some ocular palsies; (2) visual failure; and (3) defects in the visual fields.

Ocular Palsies.

I have been struck with the varied conditions under which ocular palsies occur. It is said that sixth-nerve paralysis frequently occurs as a result of cold. I have always had grave doubts of this, because I have seen more than one case of isolated sixth-nerve paralysis supposed to result from cold, and following exposure more or less definite, in which the subsequent course of the case showed that the sixth-nerve paralysis was really the initial sign of some general condition—in one case disseminated sclerosis, in another tabes, and in yet another encephalitis lethargica. And when one considers how rarely, if ever, sixth-nerve paralysis is associated with ordinary Bell's paralysis (facial paralysis)—admittedly the result of exposure to cold air, draughts, &c.—it is hardly likely to be the case that sixth-nerve paralysis without paralysis of seventh often results merely from cold. Personally I have never seen sixth-nerve paralysis associated with ordinary facial paralysis, but, as I have said, I have frequently seen isolated sixth-nerve paralysis the initial

sign of what afterwards became a widespread affection of the nervous system.

Isolated third-nerve paralysis is a fairly common affection. In nine cases out of ten it is probably syphilitic in origin. It may occur without other signs of nervous disease, and clear up completely under appropriate treatment. It may also occur in cases of tabes or general paralysis, and there also it may completely disappear under treatment. It may be assumed to be the result of gummatous formation in the nerve sheath, for the presence of a chronic degenerative disease, like tabes or general paralysis, does not at all preclude the possibility of the development of manifestations of active syphilis. In some cases of intracranial growth third-nerve paralysis is an initial sign, but it is not common to get this alone; other nerves are usually involved as well, a fact easily understood when the anatomical relations, both of the nerve and of its nucleus, are remembered. And it should be kept in mind that in some cases of pituitary tumour such paralysis is present, although the classical signs of intracranial growth may be absent. I need only mention those curious cases of so-called migraine ophthalmoplegique—severe headache, usually one-sided—with paresis of the third nerve, which under the influence of repeated attacks becomes permanent paralysis. These cases are obscure in origin, for they go on for many years and may yield only rare post-mortem information. But in some of them actual involvement of the third nerve, either in growth or in inflammation, has been found.

Perhaps the most common affection of ocular movements is from a mixed affection of ocular nerves. Such an affection is common in diphtheritic paralysis. It is also frequent in encephalitis lethargica, but in some of these cases I am convinced that the affection is very slight; it is then usually transitory, and the patient may make a complete recovery. Myasthenia gravis frequently comes under the notice of ophthalmic surgeons, and in this disease the ocular nerve affection is usually striking, because it is often so complete—double ptosis and almost complete paralysis of ocular movements. It is also striking because of its occasional, if temporary, complete or partial disappearance, and thus tragic mistakes have not infrequently been made of regarding such cases as "hysterical" because of this extraordinary variability. I think one cannot be too careful in regard to the diagnosis of hysteria in cases of ocular paralysis. In more than one instance I have known cases of myasthenia gravis mistaken for cases of hysteria, and I have known the same mistake made with reference to cases of disseminated sclerosis in which ocular paralysis, sometimes of only one sixth nerve, has been the earliest sign. Graves's disease is one of those conditions in which weakness of ocular movements is frequently present and sometimes very persistent; and I have known it present long after the characteristic signs of Graves's disease have disappeared. I have also known—although I think this must be unusual—a patient with Graves's disease come for advice because of diplopia without any complaint of other symptoms. I need only just mention cases of cavernous sinus thrombosis, familiar, I fear, to most ophthalmic surgeons, as another group of cases in which ocular palsies occur.

Visual Failure.

I should now like to say a few words about those cases of visual failure associated with widespread affection of the nervous system. Cases of impaired vision naturally seek advice at an eye hospital, and if there are any here who did me the honour to come to my out-patient clinic when I was still seeing out-patients at Queen-square they may remember the extraordinary number of patients attending there who were suffering from tabes or general paralysis with optic atrophy. I was fortunate to have these cases; they were mostly referred to me by my colleagues at Moorfields. When I see a case of optic atrophy in a patient who has signs of tabes—loss of knee-jerks and lightning pains, and without ataxy—I am always apprehensive that such a patient will sooner or later develop the signs of general paralysis. I have seen this so often that perhaps I needlessly anticipate it.

For such a development is not invariable. The case may really be one of tabes, but it is remarkable how rarely ataxy is superadded, and the converse is true of ordinary cases of actual ataxy in walking, optic atrophy rarely appearing in such cases. But I think ophthalmic surgeons will agree with me that tabetic optic atrophy—so-called grey or primary atrophy—nearly always leads to complete blindness, usually in a very brief period of time. Let me here remark on the curious difference between this form of optic atrophy and that pallor of discs which is so very common in cases of disseminated sclerosis; I think it is now generally acknowledged that the cause of disc pallor in disseminated sclerosis is an antecedent retrobulbar neuritis. There is frequently a history of temporary obscuration of vision, the result doubtless of a patch of inflammation in the nerve behind the eyeball; but it is sometimes impossible to get a history of such temporary obscuration, and one must then suppose that the primary affection was very slight and of short duration. But disc pallor supervenes, usually much more of the temporal side of the disc, but not infrequently general in character. The striking thing to me is that in such cases you may often have extreme disc pallor with 6/5 of vision. And in contrast to the optic atrophy of tabes such atrophy never proceeds to blindness. At all events, I can say that I have never seen a patient with pale discs in disseminated sclerosis completely blind. I have known blindness in one eye following an unusually severe attack of retrobulbar neuritis, but blindness of both eyes never. One has also to remember that optic neuritis may be present in disseminated sclerosis, no doubt identical in causation with the neuritis which does not show itself at the disc, and in rare cases this may be the only obvious sign of the disease. I can recall a case sent to me by Mr. J. B. Lawford with slight optic neuritis in each eye. The patient was a young lady of good physique, living under very healthy conditions, who had sought his advice because of some impairment of vision. The only other sign of nervous disease when I first saw her besides the optic neuritis was an extensor plantar response on one side, but within 18 months she had developed all the classical signs of disseminated sclerosis, and she died less than two years from the onset of her eye trouble. I need only just allude to that curious fact, now, I think, familiar to all of us, that optic neuritis is so often present, even in cases of intracranial tumour without any detectable impairment of vision and without any complaint of visual failure on the part of the patient. It is a fact of, I think, immense importance and of great interest, and its discovery is perhaps the most valuable contribution made by a physician to ophthalmology.

Defects in Visual Fields.

I should now like to say a few words about the defects in visual fields which are probably equally familiar to ophthalmic surgeons and physicians, although I am bound to say that physicians do not seem to be quite so quick at recognising their existence, possibly because they are not so familiar with their importance. I remember on one occasion sending into hospital a patient who had been suddenly seized with weakness of the left leg and left arm, and who I found also had left homonymous hemianopia. After her admission to hospital I saw her with the house physician. By that time the weakness of the arm and leg had almost passed off, and the house physician, with due diffidence, I must say, suggested that the condition had been, and was, a functional one. I asked him about the fields of vision. He had not thought of them, and I was easily able to convince him that definite homonymous hemianopia was still present. I may say that I know that this still persists in the patient, although it is now several years since her attack; this leads me to remark how rare it is for hemianopia to disappear. I have seen two cases in which complete clearing up of homonymous hemianopia took place. One case, sent to me by Mr. Percy Flemming, was that of a girl aged 15 in whom hemianopia appeared after an attack of scarlet fever. We both verified the presence of the condition, but Mr.

Flemming saw her about a year later and found that it had entirely disappeared. The other case was one sent me by Mr. J. H. Fisher. This was an old but very clever lady who had not only left hemianopia, but also repeated slight Jacksonian fits, and remained in a precarious condition for several weeks. The fits ceased, but the hemianopia was still present; finally this also completely cleared up, although the clearing up was preceded by curious hemiopic hallucinations such as have been described in some cases. The hallucination in this case was of a little girl looking over the patient's left shoulder as she read—a definite but pleasing hallucination in her blind fields. This gradually faded away—much to the patient's regret—as the field was restored. I may mention in explanation of her regret for the pleasant lost hallucination that this lady was the happy mother of many daughters and of several sons. After this attack she lived for several years in good health.

I think it is very important to be on the outlook for quadrantic or other sectional defects. They are quite easily missed, at all events by physicians, and I have known cases in which the visual difficulty complained of by patients was put down to "liver," or some equally vague cause—cases in which careful examination with the perimeter would have revealed the presence of a partial homonymous field defect.

I need only briefly refer to the field defects in pituitary cases, usually, of course, of both temporal fields. It is strange how often a patient with temporal hemiopia in one eye presents himself or herself when the other eye has become completely blind. It is to be presumed that the patient has only discovered the visual trouble when one eye has become blind or the seriousness of whatever visual inconvenience has been experienced has not been earlier recognised. But we can often obtain from such a patient a clear history that the nasal field of the now blind eye had been present when the temporal field had gone. X rays now help us greatly in the diagnosis of such cases. I need not refer to other features in the symptomatology of these cases, but I think many of you must have been struck, as I have been, with the curious variability in their response to treatment by organic extracts. I have known more than one case in which extraordinary improvement has taken place after treatment with thyroid. I have known others in which pituitary extract has been equally efficacious, and Mr. Fisher has told me of a case in which first thyroid and subsequently, after a relapse, pituitary was efficacious in clearing up the symptoms. The question of operation in some of these cases is an extraordinarily difficult one. If there is any chance of the condition being cystic, operation has some chance of success; if it is a glandular hyperplasia—and, of course, I have known many cases quite untouched by such treatment—treatment by organic extracts would be called for and operative measures would probably be unsuccessful. Yet if the patient were in urgent pain, as in some cases I have seen, I should feel strongly inclined to advise an exploratory operation in the hope of giving at least relief. It is rather remarkable in these cases how rarely optic neuritis is present. It is nearly always a primary atrophy, I suppose from pressure. Personally I have only seen one case with symptoms of pituitary tumour with optic neuritis; this case I saw a good many years ago through the kindness of Mr. Richardson Cross, and I understand the patient afterwards became quite well.

It would be rather fascinating to go on and consider the suggestions recently made by Mr. Fisher as to the connexion between Leber's atrophy and pituitary changes. Some of you may remember that I brought forward evidence which seemed to lend support to his views. It would also be interesting to consider his somewhat revolutionary ideas as to the causes of migraine, although I think any evidence I would offer in regard to this would certainly not support his very original views on this subject. But I feel I have said enough, and I would only in conclusion venture to express the hope that physicians and ophthalmic surgeons during this session of our section may learn much from each other.