On the value of eye-symptoms in the localisation of cerebral disease / by E. Nettleship.

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ON THE VALUE OF EYE-SYMPTOMS IN THE LOCALISATION OF CEREBRAL DISEASE.

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THIS subject, which might, I feel sure, have been much more appropriately introduced by a physican, presents itself to a specialist somewhat in the following form.

How far can one, who is engaged in treating ophthalmic cases, contribute to the diagnosis of intracranial or intraspinal disease? For my own part, on the present occasion, I propose to keep almost entirely to the question of intracranial disease, though I would by no means wish to see diseases of the spinal cord excluded from the discussion. Whether we include diseases of the cord or not, the whole subject naturally falls into two divisions, according as disturbances of vision and sensation, or affections of ocular movement, cause the symptoms we have to examine; and in each division, we must distinguish, as far as possible, between eye-symptoms due directly to peripheral changes, and those caused by disease of the oculo-motor or visual centres.

A. *Visual and Oculo-Sensory Disturbances.*—It is not necessary to occupy much time in discussing double optic neuritis (papillitis): for double optic neuritis very seldom helps us to decide where a brain-lesion is; and it lies beyond our present subject to ask what aid this condition gives us in deciding the nature of the disease. The occurrence of optic neuritis with meningitis points to the meningitis being basic; for it is, according to all observers, rare in meningitis of the convexity. In regard to tumour, it is worth asking whether papillitis occurs more commonly, or comes on earlier, when the tumour is at the base, or in the anterior part of the brain, than when it is situated in the cerebellum, or posterior parts of the cerebrum. I do not know whether any statistics on this point exist; it is obviously a question on which the collection of good evidence would be full of difficulties.—Uniocular papillitis sometimes occurs in cases of coarse brain-disease, and has some localising value—generally coming on, so far as recorded cases tell, in the eye opposite to the lesion. It is of importance that all cases should be carefully observed and recorded; and it would be well to include, as possibly bearing on the subject, cases in which the papillitis, though eventually double, takes place in one eye long before, or much more severely than in, the other; also cases of double and equal papillitis, where sight is much more affected in one eye than the other. I have seen a case (Miss K.), in which a tumour in the postero-inferior part of the right anterior lobe, adherent to the bone, had caused severe pain, chiefly on the right side of the face and head; then she had simultaneous double

papillitis, with early, extreme defect of sight in the right eye, but no failure of vision in the left till a year later. Here, contrary to the rule, the neuritis was worst on the side of the lesion. In another case, the only lesions were softening of the under surface of the left frontal lobe, with adhesion of the softened part to the dura mater, and atrophy of the left optic nerve; the symptoms had been severe pain in the left side of the head and face, followed by fits, with convulsion of the opposite arm; then, about two months later, recurrence of pain in the left head, with rapid blindness of the left eye without changes, and papillitis of the right without failure of sight; the left disc slowly became atrophied, the right recovered. (Alice S., originally in my hands, then under Dr. Bristowe's care; *post mortem* examination by Dr. Greenfield.) Although, in the first case, the papillitis was worst in the eye on the side of the lesion, and in the second was present only in the opposite eye, it will be observed that, in both cases, sight was affected most on the same side as the lesion. Dr. Gowers has recorded two cases bearing on the subject. In one (*Medical Ophthalmoscopy*, Case 17), a man had fits, with temporary weakness of the right arm, followed by pain on the right side of the head and face; then papillitis of the right eye, passing off, and leaving the disc healthy, but sight defective; later, simple atrophy of both discs. In Gowers's second case (Case 25) there was left hemiplegia, from embolism of the right middle cerebral artery, and consequent softening of the corpus striatum; there were redness and haze of both discs, this change being more marked and lasting much longer in the right eye (*i.e.*, on the side of the lesion).

Cases of single optic neuritis from brain disease, with or without affection of sight, have to be distinguished from two other well-known varieties in which the disease in not situated further back than the optic foramen. (1) We meet with cases in which one eye fails with pain on the same side of the head, usually in the temporal or occipital region, sometimes also "behind the eye," and there may be pain in moving the eye. The pain and the amblyopia may both be slight, or both may be severe. In some cases the eye becomes quite blind, and when this occurs I have never yet seen it improve materially. At first the disc looks healthy or sometimes shows some haze. In a few weeks, unless the defect of vision passes off, it becomes more or less atrophic. Neither vomiting nor cerebral symptoms occur, and the pain, though sometimes very severe, never becomes general; it is often called "neuralgic" or "rheumatic." I have never seen the second eye suffer. In one case of this kind, my patient had previously suffered from Bell's paralysis of the opposite facial nerve (*Lancet*, 1880, i, 765). Another patient, Miss B., had had a severe attack of sciatica, followed by numbness and weakness of the limb. Mr. Hutchinson published cases of the same kind many years ago, and drew a comparison between some of them, and cases of infantile paralysis (*Ophthalmic Hospital Reports*, iv, pp. 381 and 120). It seems to me more likely that a rheumatic inflammation of the optic nerve occurs, probably in its course through the optic canal. (2) In the other group of cases, we find severe papillitis with very great venous engorgement, with failure of sight, sometimes of high degree, and, as in the previous group, pain on the same side without cerebral symptoms. Probably the inflammation in these cases is always communicated to the optic nerve sheath from the orbital structures; orbital disease, either cellulitis or tumour, can, indeed, often be proved. I believe that the signs of œdema and venous stasis are usually more marked in the cases of this group, than in papillitis from intracranial disease.

In the present state of knowledge, it would seem, from what has been said, that papillitis, with other symptoms of brain-disease, tells us something of the seat of the disease if it be single, or if papillitis in one eye be accompanied by failure of sight, without immediate change in the disc, of the other; and perhaps we may add, cases of

double papillitis in which either the visible changes, or the sight, become much worse in one eye than the other.

Passing from inflammation to primary atrophy of the optic nerve, we here also fail to gain much help in localising brain-disease. I believe that atrophy following pressure on the chiasma, whether by tumour or by fluid distension of the third ventricle, presents nothing pathognomonic, either in its course or appearances. I would like to ask here whether optic atrophy is often met with in chronic hydrocephalus, as is sometimes stated? I do not remember ever making the diagnosis.

It is certainly possible in some, though by no means in all, cases of atrophy not preceded by papillitis to distinguish between "spinal atrophy" (where the disc is often opaque and greyish, and the lamina cribrosa concealed), and atrophy secondary to injury, pressure, or destructive inflammation far above the eye, in which the lamina cribrosa is often exposed, the disc somewhat cupped, and its colour less grey than yellowish. But these differences are far from constant, and give no aid in the localisation of brain-disease. Nor is it always possible to say whether atrophy has been preceded by papillitis or not.

In regard to the mode of failure of vision in optic atrophy, and its possible bearing on our present subject, the state of our knowledge leaves much to be desired. For example, colour-blindness does not seem to occur in any constant relation, either to loss of vision, or loss of visual field, in optic atrophy. Some cases have very recently been recorded, which seem to point to the consciousness of colour depending rather on special endowment of some part of the brain than on any differentiation of the retinal or optic neural structures. Three of these are cases of loss of colour-sense in one half of the field of vision with no loss of acuteness of sight in the affected part (Bjerrum,* Samelsohn,† H. D. Noyes.‡) Noyes's case presented partial optic atrophy with Argyll-Robertson pupil and shooting pains; in one eye there was high amblyopia, with almost complete colour-blindness; in the other colour-blindness in one lateral half of the field only, acuteness of sight being normal over the whole field. But cases are not wanting which seem to show that colour-defect, even when limited to one half of the field, may be due to changes in the optic nerve: thus in the same paper, Noyes relates another case of hemiachromatopsia of the nasal half of each field, with glaucomatous cupping of the discs, the temporal halves of the fields, as in most cases of glaucoma, being normal; and I have myself recorded two cases of injury to the optic nerve, in one of which colour-perception was enfeebled, whilst in the other, with about the same visual acuteness, it was perfect. (*St. Thomas's Hospital Reports*, xi, p. 113, Cases I and VII.) Steffan (Gräfe's *Archives* 27, 2, p. 11) has given a case of colour-blindness, with very slight defect of visual sharpness, following an attack of giddiness, in an old man; the sight became quite perfect, and colour-perception improved, but some defect for green remained as long as four years after the attack. This case, which has been a good deal quoted as proving a colour-centre, certainly deserves attention, though it is not convincing. Colour-blindness is nearly always very marked in optic atrophy associated with locomotor ataxy, yet it is probable that in these cases the changes begin at the ocular end of the nerve. It is clear, therefore, that failure of colour-perception, in its bearing on cerebral localisation, needs further study; and the point is one towards the solution of which we, as specialists, ought to be able to contribute.

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* Bjerrum *Hospitals-Tidende* R. 2, B. 8, p. 41 (referred to in a paper by Dr. Berry, *Edinburgh Medical Journal*, February 1882).

† Samelsohn (of Cologne), *Centralblatt fur Medicinischen Wissenschaften*, 1881. No. 47 (referred to in Knapp's *Archives of Ophthalmology*, xi, 217).

‡ Noyes, Knapp's *Archives of Ophthalmology*, xi, 210, 1882.

I wish now to ask attention to a different subject.* All ophthalmic surgeons know the cases often called "spurious glioma," in which a young child loses an eye with iritis and the clinical appearances of either detached retina or lymph in the vitreous body, followed by shrinking of the globe and secondary cataract. This clinical group includes several pathological processes. Some of the cases are, no doubt, instances of severe infantile syphilitic eye-disease; a few may possibly be the results of injury. On dissection, we may find intra-ocular hæmorrhage, inflammation of the vitreous body, or detachment of the retina secondary to irido-choroiditis (Nettleship, *Pathological Transactions*, xxxi, 1880; Brailey, *Guy's Hospital Reports*, 1881, p. 497). The interesting point for our present purpose is, that severe cerebral symptoms sometimes occur just before the eye becomes blind with inflammatory symptoms. The two following cases are taken from amongst several others. Dr. B. brought me his little girl, aged eighteen months. The left eye was congested, and very soft; there was no anterior chamber; the iris was adherent to the lens-capsule. Three months previously, she had been taken suddenly ill, with quick breathing, dilated pupils, "glazed looking' eyes, and unconsciousness; there were no convulsions. She was seen by a high authority upon diseases of the nervous system. The temperature was 103-104° Fahr. for a fortnight; and two or three times it was thought she would die. Very early in the illness, the left pupil was smaller than the right, and the eye red, and soon afterwards a "fiery" reflection was noticed from deep in the eyeball; there was no proptosis. The child remained ill for three or four months. Soon after the onset, whooping-cough developed, and the relation between it and the severe cerebral symptoms and ocular inflammation was not clear. In another case, a little girl (Julia B., 3½ years), who had had bronchitis and measles one year before, and whooping-cough six months before, began to suffer one month ago from pain in the head, chiefly in the left forehead; a few hours later, convulsions set in, and she became, and is said to have remained for a week, totally unconscious; on the third day, the left eye was swollen and inflamed, and the iris discoloured; the child continued to have severe pain "in the eyeball" for another week, and then quite recovered. When I saw her, the eye was soft, the periphery of the iris retracted, and a yellowish-red reflex, devoid of vessels, was seen behind the clear lens. In the first of these cases, the eye-disease seems clearly to have been secondary to the cerebral attack; in the second case, the pain is said to have been all along localised on the side of the bad eye, and the question perhaps arises whether the convulsions and unconsciousness could have been merely symptomatic of the local affection of the eye. I do not myself think so; but the question is open for consideration. In regard to these cases of pseudo-glioma with brain-symptoms, we want to know what changes, if any, occur within the skull. I do not know whether these children ever die. If meningitis occur, is there anything unusual in its seat or its nature, and why does the child show this peculiar eye-disease instead of suffering from double optic neuritis? We may here remember that a form of choroiditis or panophthalmitis sometimes occurs in epidemic cerebro-spinal meningitis, and rarely after various infective diseases. The questions of pyæmia and of cerebral thrombosis have also to be considered in connection with such cases.† It may here be observed that, in the rare cases where a patient has died with purulent meningitis after panopthalmitis, or suppuration in the orbit, it has often been impossible to find any continuity between the orbital and the meningeal disease.‡

* A more detailed paper on the subject of this paragraph was read by the author before the Ophthalmological Society on Oct. 12 last. (Feb., 1883.)

† Hutchinson, *Ophth. Hosp. Rep.*, viii, 146; also the author in *Med. Times and Gaz.*, 1880; p. 63.

‡ Harlan, *Trans. American Ophth. Society*, vol. ii, page 542. T. P. Teale, *Brain*, vol. ii, 423.

I pass to another subject. It is not uncommon to meet with cases of blindness without visible changes, in infants. I formerly thought that the prognosis was necessarily fatal as regards sight. But, more lately, I have seen recovery of sight, apparently perfect, take place in several cases after the children had "taken no notice," and had had inactive pupils, for several months. Some of these patients have shown signs of slight chronic hydrocephalus; but others have, so far as I could judge, been free throughout from all signs and symptoms of brain-disease. Is not recovery from infantile amaurosis commoner than some of us have thought? and to what is the blindness attributable when there are no signs of hydrocephalus?—In another form of amaurosis in infants, we find extreme disorganisation of the choroid in the form of immense patches of atrophy, with grey membrane and accumulations of black pigment. In one of my own cases, the head was at first very small, but afterwards enlarged to an immense size.* Two cases of extensive choroidal atrophy in microcephalic infants recorded by Hutchinson (*Ophthalmic Hospital Reports*, v, 34, Cases 4 and 8) were most likely of the same nature. I believe that Dr. Barlow has found chronic hydrocephalus after death in one such case which ended fatally. Does the occurrence of this form of infantile choroidal disease, which is quite different from anything we commonly recognise as syphilitic, point to any particular variety of hydrocephalus?

We come now to cases of affection of sight without ophthalmoscopic changes, in which the cause is undoubtedly cerebral; hemiopia or hemianopia, defect of sight with hemianæsthesia, and other forms of cerebral blindness.—First, as to temporal hemianopia (loss of the outer half of each field), the symptom has, in rare cases, been proved to be due to tumour at the anterior part of the chiasma (Saemisch, E. Müller). That temporal hemianopia, however, does not necessarily point to disease in this situation, is shown by a case lately published by Dr. Baxter (*Brain*, January 1882), and which I had the opportunity of examining during life, where, *post mortem*, no lesion of any kind could be detected by the naked eye (except condensation of the vault of the skull).† It may be observed that, in some cases of spinal optic atrophy, the invasion of the visual field is remarkably symmetrical in position in the two eyes, showing exact bilateral symmetry in the seat of the lesion, whether this be in the centres or at the optic discs. In two such cases, I found the lower and inner quadrant of each field lost. A large number of cases of hemianopia have been collected and analysed in much detail by Dr. Wilbrand of Hamburg (*Hemianopsie und ihr Verhältniss zur topisch. Diag. der Gehirnkrank.*, 1881). Many of the more important cases will also be found repeated in all essentials in Ferrier's article on Cerebral Amblyopia and Amaurosis in *Brain*, No. xii; and in the Periscope of the *Ophth. Hosp Reps.*, vols. viii and ix.

The ordinary homonymous hemianopia (loss of the right or of the left half of each field of vision) has furnished and will continue to supply very valuable evidence of the locality of the lesion producing it, though the discovery of the cortical visual centres has widened the area within which such lesion may be expected to be found. 1. The fact that homonymous hemianopia occurs from destructive lesion of one optic tract demonstrates the semidecussation of the optic nerves at the chiasma in man. Cases of this kind, with good *post mortem* localisation of the lesion to the region of one optic tract, are rare; the best of them being, I believe, by Hjort, Dreschfeld, Gowers, and Hirschberg. 2. Several cases in which the lesion has involved the region of the corpora geniculata and the posterior part of the optic thalamus are also on record (Jackson, Dreschfeld). 3. And it has now been shown, further, in several cases, that lesions of the cortical or subcortical structures

* The first part of this case was published in *Ophth. Hosp. Rep.*, viii, p. 518.

† See, however, remarks on this case by Priestly Smith in *Ophth. Rev.*, ii., p. 11. (Jan., 1883.)

(posterior part of internal capsule) in the region of the angular gyrus and occipital lobe, may produce homonymous hemianopia. (Huguenin, Westphal, Pooley, Hughes, Baumgarten, Curschmann, Dreschfeld, and especially Haab.) It does not appear that we have at present any means of determining during life, by the characters of the hemianopia, in which of these three regions the lesion is seated. Ferrier (*Brain*, No. xii, 473) suggests that, in tract-hemianopia, the loss of field may be expected to come quite to the fixation point; whilst, in cerebral hemianopia, it may be expected to stop short of the fixation point by several degrees, leaving thus a small area of central vision perfect. If this distinction be trustworthy, we may, I think, say with some confidence that tract-hemianopia must be very rare, and cerebral hemianopia the rule; for in homonymous hemianopia a small area of central vision is almost invariably present. I have never been able to feel sure that it was absent. Ferrier quotes Gowers's case of tract-lesion as one in which the dividing line passed through the fixation point; but I have Dr. Gowers's authority for saying that, in the case referred to, no precise map of the field was made on the perimeter; and certainly no less rigorous test could be accepted on so delicate a point.

Other clinical evidence of the rarity of disease of the optic tract as a cause of hemianopia, may be adduced. Hemianopia, though it may be simple, is very often associated with hemiplegia. Indeed, Gowers (BRITISH MEDICAL JOURNAL, 1877, vol. ii, p. 729) says that temporary hemianopia is extremely common in the early period of attacks of hemiplegia; Ferrier suggests that this symptom may be caused by temporary pressure on the optic tract—an explanation which, though I cannot presume to criticise it, does not strike me as probable. It is important to note that what we call hemianopia is in numerous cases really a tetrato-anopia, loss of a quarter of the field or of a precisely bounded sector between a quarter and a half. Now, in some of these there is paralysis of the limb corresponding to the blind quadrant, *i.e.*, following Hughlings Jackson's law that in lateral hemianopia with hemiplegia the patient cannot see to his paralysed side, we have cases of tetrato-anopia in which he cannot see his one paralysed limb. I have seen a case illustrating this so far as the leg is concerned;* and I dare say physicians see many such. Loss of the upper quadrant alone also occurs;† but I am inclined to think that it is less common than loss of the lower quadrant. I do not know whether loss of an upper quadrant often occurs with isolated paralysis of the arm; but one case showing this coincidence has been published by Bernhardt, and will be found in abstract in *Brain*, part xviii. No single lesion involving the optic tract could account for paralyses so precise yet so limited and so physiologically associated as these.

Again, in homonymous hemianopia, it is not uncommon to find also some peripheral restriction of the remaining half of each field; this may be greater in one eye than in the other, but from such cases as I have been able to refer to (including several of my own) it does not seem that the eye with the greater restriction always bears the same relation to the presumed seat of the lesion.

Then the hemianopic loss of field is by no means always exactly equal in the two eyes; and this inequality, like the peripheral limitation just mentioned, does not seem to stand in any constant relation to the site of the lesion. Both these features in hemianopia cases deserve further attention. They could not easily be accounted for by lesion of the optic tract. But if, as the latest experiments seem to show (Ferrier and Yeo) lesions of the occipital lobe cause lateral hemianopia, whilst damage to the angular gyrus gives rise to crossed blindness, it is probable that variations in the seat and extent of disease

* This patient (Wm. C.) has been to many hospitals, and his case has lately been recorded by Dr. Berry (*Edin. Med. Jour.*, February 1882).

† See cases by Claeys in *Annales d'Oculist.* T. 80, 1878; and others.

affecting these contiguous parts of the visual centre, might account for variations in the resulting hemianopia. I have lately seen a case under the care of Dr. Bristowe, in which, together with symptoms of chronic cerebral disease, there was word-blindness and homonymous hemianopia, with no ophthalmoscopic changes; here the symptoms seemed to point to cortical disease.

Finally, the rarity of decided atrophic changes at the optic discs, in cases of homonymous hemianopia, even of long standing, points strongly against the tract being the seat of the lesion. The recently discovered cases of hemi-achromatopsia, already referred to, also seem to have a most important bearing on the localisation of the visual functions, in the cortex: it is not unlikely that these cases may be found to be common if sought.

Other pathological evidence is beginning to accumulate in favour of a cortical visual centre, and of its being seated in or about the occipital lobe. Thus, at least four cases are on record of double cerebral amaurosis, in which both occipital lobes were found implicated after death.* In one of them (Nothnagel's), hemianopia had occurred first. Again, according to Bastian, thrombosis of the posterior cerebral artery not unfrequently causes impairment of vision of the opposite eye, due, as Ferrier suggests, to interference with the cortical centre. A few cases are on record in which one eye was blind, and the other hemianopic in connection with chronic cerebral disease.† In only one of these cases, by Ross, has a *post mortem* examination been obtained; here a tumour was found pressing on the corpora quadrigemina, and most on the side opposite the blindness; the localising value of the eye-symptoms here, however, was lessened by the presence of double optic neuritis.

We should expect to meet with cases of uniocular amblyopia or amaurosis from cortical lesion, without ophthalmoscopic changes, and uncomplicated by hemianæsthesia, just as we meet, not uncommonly, with hemianopia, probably cortical, without hemiplegia. It would be very interesting to know whether such cases occur; and, if so, whether they are rare. I have seen two in which such an explanation seemed highly probable. Uniocular failure of sight has been seen in general paralysis, with lesion involving especially the occipital lobe (Fürstner, according to Ferrier‡).

It is most important, in reference to localisation, to note the state of the pupils, in all cases of damage or loss of sight from brain disease. Loss of reflex pupillary action indicates damage not higher up than the corpora quadrigemina, whilst blindness with active pupils points to disease in the cortex or subcortical structures (internal capsule).

Megrim is a malady, the study of which may help towards localisation; the association between its ocular symptoms, and other occasional symptoms of one-sided nervous discharge, or irritation, being worthy of more detailed investigation.

With regard to the fifth nerve, the only questions which occur to me are: 1. Whether shooting pains in the territory of this nerve, in cases of progressive optic atrophy, have the same value for the diagnosis of locomotor ataxy as lightning pains in the legs; and 2. If so, can we infer that the degeneration is beginning in the cervical region, instead of lower down, and has this any value in prognosis? If we may count such cases as probably ataxic, the number of uncomplicated optic atrophy cases will be diminished—since progressive atrophy, with no other subjective symptoms than shooting pains in the head, is not very uncommon. While on this point, I may mention that I have found that about 20 per cent. of my cases of progressive optic atrophy pre-

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* Nothnagel, Griesinger, Moore, Chvostek; quoted by Wilbrand, *loc. cit.*

† Gowers, *Medical Ophthalmoscopy*, Cases 30 and 55; Berry, *Edinburgh Medical Journal*, February 1882, Fig. 9; Ross, *Brain*, No. viii, p. 569.

‡ Ferrier, *Localisation of Cerebral Disease*, p. 132.

sented no other symptoms, either objective or subjective, pointing to disease of the cord or brain ; in about 80 per cent., some such symptoms were present ; (the total number of cases on which this statement is based was fifty-eight). Herpes zoster of the fifth nerve may be mentioned as a malady of which our pathological knowledge is incomplete. When, as sometimes happens, it is associated with paralysis of neighbouring cranial nerves (*e.g* , the third and the facial), we seem driven to assume a central, or, at least thoroughly intracranial, affection.

B. *Motor Disturbances.*—In considering the motor paralyses of the eyeball, in reference to localisation of cerebral disease, we should endeavour to distinguish between cases due to affections of (1) the nerve trunk ; (2) the lower centres (nuclei of origin) ; and (3) the higher (cortical) centres, for complex co-ordinated movements. I do not pretend to have mastered a tithe of what has been written upon this complex subject, especially upon the last-named division—that of the disorders of associated movements. The writings of Prevost, Hughlings Jackson, Priestley Smith, and Landouzy, upon lateral deviation of the eyes ; and those of Hutchinson, Gowers, Buzzard, Allen Sturge, Ormerod, and others, upon the varieties of ophthalmoplegia, are, no doubt, familiar to most of my hearers. I will, however, raise a few questions which seem suitable for further investigation.

Isolated Peripheral Paralyses (cases in which the muscles, supplied by only one nerve trunk, are paralysed). I suppose we have no means of diagnosing the position of a syphilitic node on the trunk of the fourth or sixth nerve ; and, as for the third nerve, it is only in the comparatively rare cases where a single muscle is picked out, that we may guess its nerve-branch in the orbit to be the probable seat of disease. Isolated paralysis of the third nerve, with simultaneous hemiplegia of the opposite side, may, as Hughlings Jackson long ago pointed out, indicate a lesion of the crus cerebri on the side of the paralysed third. Though a large proportion of the cases of single ocular palsy are due to syphilitic disease of nerve-trunks, whilst a certain number are warnings of locomotor ataxy, or general paralysis of the insane, we meet with others, especially, I think, in old persons, where paralysis of a single nerve occurs without any cause discoverable during life. Probably *post mortem* examination may, in future, clear up some of these.

It is well known that the relative frequency with which the third, fourth, and sixth nerves are paralysed singly is very different, paralysis of the fourth being much the least common. The anatomical relations of the sixth, in its long intracranial course, are supposed to account in great part for its proclivity ; but this is a point on which there still seems room for the collection of evidence. The comparative immunity of the fourth may be partly due to its containing fewer fibres than the other two, and being, therefore, proportionately less liable, *cæteris paribus*, to the commencement within itself of spreading disease, such as gummatous inflammation. Isolated paralysis of the superior oblique following injury to the head, is probably often due to fracture of the roof of the orbit, and consequent mechanical interference with the pulley, rather than to damage of the fourth nerve. In my experience, the relative frequency of the single oculo-motor paralysis has been, in seventy-seven cases—third nerve alone thirty-one cases, fourth alone nine, sixth alone thirty-seven.

Multiple Ocular Paralysis, when peripheral, is usually unsymmetrical. The seat of disease is presumably always either at and about the cavernous sinus, or about the sphenoidal fissure. There is generally severe localised headache ; the paralysis of the third takes all its branches equally, including those to the levator palpebræ and to the iris and ciliary muscle. The first and second divisions of the fifth nerve seldom escape ; but the third division is often free, and when it is

affected there is, in my experience, usually evidence of the disease having reached the zygomatic fossa ; the optic nerve also often escapes, but, when affected, there is usually papillitis rather than progressive atrophy. In ophthalmoplegia externa from nuclear disease, the symptoms, are bilateral ; the levatores palpebrarum are much less affected than the ocular muscles ; the fifth nerve seldom suffers ; the iris and ciliary muscle often escape, at any rate in the earlier stages ; the optic nerves not unfrequently suffer from progressive atrophy. Although symmetrical ocular paralyses are usually either nuclear or cortical, symmetrical syphilitic disease of nerve-trunks accounts for a few of them, and has been proved several times *post mortem*, at least for the third nerve. If in a case of paralysis of both third nerves alone, the irides and ciliary muscles escape, the cause is almost certainly central, the movements of these parts being under the control of centres separate from those which govern the movements of the external ocular muscles.

Central Paralyses (*Paralyses of Associated Ocular Movements*).—Our knowledge of these difficult cases is still very incomplete, both from the clinical and the pathological sides ; but it is rapidly growing. As in the analogous cases of disturbances of vision, we want to be able to distinguish between cases where the lesion is of the basal centres (the "nuclei of origin") of the ocular nerves, and others where it is cortical (motor centres). In regard to the latter, I believe that, as yet, comparatively little has been proved, though there are some remarkable cases on record in which one or more of the associated movements of the eyes have been lost, with evidence of comparatively mild brain-lesion.* Cases are known, besides Priestley Smith's remarkable one of lateral deviation, of loss of power to look upwards, loss of power to converge the optic axes, and loss of power to move either eye outwards. How far such comparatively simple paralyses are due to cortical, and how far to nuclear disease, is very much a matter for future investigation ; but analogy points to lesion of some part of the cortex.

The cases of ophthalmoplegia externa (Hutchinson, *Transactions of the Royal Medical and Chirurgical Society*, 1879), in which several movements are gradually lost, or in which the eyes have become almost fixed, are probably always due to disease involving the "nuclei of origin." Thus, though symmetrical, the symptoms are often for a time more advanced in one eye than in the associated muscles of the other ; and the motor palsy is sometimes associated with progressive double optic-nerve atrophy, or with paralysis of both fifth nerves—*i.e.*, with paralyses of nerves closely related at their basal nuclei, but presumably not associated in the cortical centres.

One form of single paralysis occurring in certain cases of apoplexy—viz., uncomplicated ptosis—has received considerable attention in France, especially from Landouzy (*Archives Générales de Médecine*). Ferrier thinks it possible that a separate centre for the elevation of the upper lids is situated towards the upper part of the anterior lobe of the cerebrum. We may remark that the levator palpebræ is functionally so distinct from the proper ocular muscles, that we certainly need feel no hesitation in provisionally assigning to it a separate centre. Indeed, other facts besides the occurrence of "isolated ptosis" favour this view.† Thus, in the typical progressive ophthalmoplegia externa before alluded to, the levatores palpebrarum are far less affected than the other muscles ; there is often only slight ptosis, though the globes are sometimes motionless. Observation may be expected to show that, of the various associated movements of the eyes, some are lost more commonly than others. I am inclined to

* Priestley Smith, *Ophthalmic Hospital Reports*, vol. viii, 185, and vol. ix, 22 and 428 ; Gowers, *Transactions of the Ophthalmological Society*, vol. i, 117 ; Allen Sturge, *ibid.*, p. 165 ; Lang and Fitzgerald, *ibid.*, vol. ii, p. 230.

† See also some remarks by Allen Sturge, *loc. cit*, p. 184.

think that, hitherto, loss of upward movement has been noticed more often than loss of movement downwards. It will be remembered that upward movement is the most difficult and wearisome of all the ocular movements. I would suggest that attention to the order of recovery of movements or of muscles may be useful. I have for a long time thought (and probably the fact, if it be one, is well known) that, in paralysis of the third nerve, the levator was the first to recover. In central paralysis, this might perhaps be accounted for by the movements of the upper lid being more automatic than those of the eyeball; but this explanation could hardly apply in cases due to disease of the trunk of the nerve. Some interesting remarks on this subject, the order of recovery in paralysis of nerve-trunks, will be found in a case lately published by Dr. Ormerod.*

Paralysis of one nerve, often quite incomplete and temporary, but sometimes permanent, is not an uncommon precursor of locomotor ataxy. I would ask whether it is common to meet with complex paralysis under the same circumstances. I had under care, some time ago an ataxic man (Isaac L.) with incomplete paralysis of the external rectus, superior rectus, and levator palpebræ of the same eye, and dilatation of both pupils; he was under care for many months, and no other oculo-motor symptoms appeared. Such a combination might be assigned either to sclerotic change distributed in an unusual manner in the nuclear region, or to peripheral disease of the motor branches similar to the peripheral progressive atrophy of the optic nerve in this disease.

Before concluding, I should like briefly to allude to one other subject upon which observation is needed—*uniocular diplopia* from cerebral disease. Several cases have been recorded in this country and abroad, in which patients with brain-disease averred that they saw double with one eye. The symptom is so unintelligible, that a very natural scepticism has often been expressed on the subject. It is a point which can be settled only by very careful future observation, and especially by looking for the symptom in cases free from complication with ocular paralysis.

In concluding, I wish to express my thanks to my friends Dr. W. A. Fitzgerald and Mr. J. B. Lawford for their assistance in collecting the material for many parts of this paper.

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* Ormerod, "Case of Symmetrical Disease of Third Nerves," etc. *Brain*, vol. v p. 260.