On the clinical significance of double optic neuritis in children and young adults, and on the capacity for recovery from symptoms of tumour of the brain / by C.O. Hawthorne, M.D.

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ON THE CLINICAL SIGNIFICANCE OF DOUBLE OPTIC NEURITIS IN CHILDREN AND YOUNG ADULTS, AND ON THE CAPACITY FOR RECOVERY FROM SYMPTOMS OF TUMOUR OF THE BRAIN, BY C. O. HAWTHORNE, M.D.

ON THE CLINICAL SIGNIFICANCE OF DOUBLE OPTIC NEURITIS IN CHILDREN AND YOUNG ADULTS, AND ON THE CAPACITY FOR RECOVERY FROM SYMPTOMS OF TUMOUR OF THE BRAIN.

By C. O. HAWTHORNE, M.D.

The cases which form the basis of this paper illustrate some of the circumstances under which double optic neuritis affecting children and young adults occurs in clinical practice. In none of them, fortunately, is the cause of the neuritis made absolutely certain by post-mortem examination, but as they are records of clinical facts which demanded to be dealt with both from a diagnostic and prognostic point of view, they have a definite practical interest and value. An attempt has been made to increase this value by making the records as complete as possible, both by detailed examination of the patients, and by following the cases after the opportunity for continuous observation had passed away. In this respect it may be noted, that whilst some of the cases were first seen when the symptoms, including the optic neuritis, were of recent development, others came under observation long after —in some cases years after—the acute conditions had subsided. Hence, presuming these two groups include cases of the same order, the clinical picture they contribute to form is rendered more complete, the probable issue of the earlier cases being reasonably inferred from the known facts of those seen at a later stage. At all events, the records convey some of the considerations and difficulties which arise in the practical handling of such cases, and at the same time contribute some-

thing to our knowledge of the clinical history of optic neuritis.

The patients are all young, their ages at the date of the first observation ranging from 10 to 21 years, but the date of the occurrence of the optic neuritis was not in any case later than the seventeenth year. Of the total of nine, six are females and three males.

The first question which inevitably arises on the recognition of a double optic neuritis is this—Is the neuritis due to a tumour of the brain? the word tumour being used, in the sense Hughlings Jackson defined it, as equivalent to a "foreign body" or "adventitious product" within the skull. An affirmative answer to this question is sometimes easy; at other times such a result can only be reached if all the evidence is carefully collected and weighed. On the other hand, a confident negative answer is almost always difficult, and for the most part is only justified when some other admitted cause for the optic neuritis can be certainly recognized, or when, in the absence of any such cause, and in consequence of the lapse of time, the progress of the case, and the non-appearance of other evidence, the existence of a tumour is rendered highly improbable. The diagnostic position thus defined is the first practical issue from a study of the cases here under consideration.

Attention may now be directed to the evidence of cerebral disturbance actually present in the first seven cases, which is taken as justifying the view that the optic neuritis was in all probability dependent in each case on the existence of an intracranial tumour.

These seven cases naturally fall into two groups—(1) those seen shortly after the development of the optic neuritis; (2) those in which the optic neuritis had long since passed into a condition of optic atrophy.

The first case (see p. 130) presents evidences of cerebral disturbance in abundance. Obstinate vomiting, and pain in the head varying much in intensity, are sufficient of themselves, together with the optic neuritis, to make the diagnosis, but

here we have in addition, giddiness, retraction of the head, anosmia, and certain paralyses, not to speak of the mental phenomena and the slow pulse and subnormal temperature.

The second case offers a marked contrast to the abundant evidence provided by the first. With the exception of considerable pain in the head and optic neuritis there is no manifest evidence of cerebral disturbance, yet these will be allowed to be sufficient, and more especially as they receive confirmation from a study of the knee-jerk. The observation in this case was made under unusually fortunate circumstances, for whilst the knee-jerks were present when the boy was first seen, they disappeared during his residence in hospital, and they have never since been detected. There is thus no doubt that the patient lost his knee-jerks during the time when he was the subject of headache and double optic neuritis almost certainly due to cerebral tumour. In two other of the cases (Nos. 5 and 7) here submitted with a diagnosis of intracranial tumour the knee-jerks were absent. No doubt before such a conclusion in reference to the knee-jerk is justified the examination demands great care, as unless this is exercised the jerk, though potentially present, may not be elicited. In examining these cases the precautions insisted on by Jendrassik, Buzzard, and Gowers have been borne in mind, and whenever possible the tests have been repeated and confirmed by other observers. The result is to show that of the seven cases the knee-jerks were absent in two (Cases 5 and 7), in one (Case 2) they disappeared under observation, and in another (Case 1) they varied at different stages of the case, being sometimes present or exaggerated and sometimes absent or diminished.

The point of importance just now is the restriction in Case 2 of the evidences of cerebral disturbance to double optic neuritis, headache, and loss of knee-jerks, and the necessary inference arising therefrom, viz., that such loss may be a valuable confirmatory sign of intracranial disturbance. Yet it must be remembered that the loss of the knee-jerk did not occur until fully two months after the onset of symptoms; during the greater part of these two months the boy had no

complaint to make except of pain in the head, and it was not considered necessary to seek medical advice until sight was failing. The earlier phases of the case were marked by nothing more than headache and optic neuritis. It is highly probable, therefore, that neglect to use the ophthalmoscope at that date would have led to a serious error of diagnosis.

In Case 3 a further illustration is afforded of the slightness of the evidence, apart from optic neuritis, in some cases of cerebral disease, presumably tumour. Though the boy had never been confined to bed even for a single day, yet symptoms of intracranial disturbance had undoubtedly existed for six or seven months. Every morning during that period the boy vomited and had some frontal pain, but at other times he appeared quite well. When medical aid is sought the boy is blind from optic atrophy, the result of neuritis which had doubtless existed for months. Here again the first, and for long the only symptom, was vomiting, and that limited to one particular hour of the day. The vomiting and double optic neuritis were the only two events which occurred in a period of ten months, the vomiting being regarded by the parents as a trifling affair and only mentioned under some pressure. Yet it was the first, and probably for some time the sole evidence of an organic intracranial disease which has resulted in the permanent destruction of the patient's sight.

The three cases well display the cerebral events which may be associated with double optic neuritis as evidence of (presumably) intracranial tumour, and show how widely such events may differ both in their number and severity.

The next four cases came under observation for defective sight dependent on optic nerve atrophy, the conditions having been in existence for periods of time ranging from six months to ten years. In all but the last one the optic atrophy was manifestly the sequel to an optic neuritis, and the question may be raised whether this case (No. 7) ought to stand in line with the others. And all the more so, that whilst in the three other cases (Nos. 4, 5, and 6) there is a distinct history of a more or less severe illness with such symptoms as pain in the head and vomiting, the case now alluded to has no such history,

but, on the contrary, the failure of sight is reported to have come on quite gradually and without any other symptom or evidence of any kind. There are, however, certain reasons which may be urged in support of the suggested diagnosis. In the first place, the absence of evidence of neuritis at the present date does not prove that neuritis never existed. "I am certain," writes Hughlings Jackson,[1] "that the most striking appearances of double optic neuritis . . . will pass away so far as to render it impossible for a careful observer seeing the patient for the first time, when recovered, to be able to declare that the peculiarities discoverable about the optic disc are pathological." Similarly, Gowers[2] writes, "Great caution is necessary in inferring, from the appearance of discs long after the onset of the atrophy, that this was simple and not neuritic; . . . the characters of the latter may ultimately resemble very closely those of the former." The same point is emphasized by Buzzard.[3]

There is another statement resting on the authority of Hughlings Jackson[4] which may be advanced in support of an arrangement that places Case 7 in line with the previous six. It is this, "There is sometimes an acute illness as our first evidence of tumour . . . but it is important to bear in mind that optic neuritis may occur without any headache and without vomiting, or that headache may be slight and the vomiting none." It must therefore be regarded as possible, that the condition of the optic discs in this patient is not due to a primary atrophy, but is subsequent to a pre-existent optic neuritis which was unaccompanied by any other sign of cerebral disturbance, and that the evidences of neuritis other than the atrophy of the discs have disappeared. But even though the atrophy of the optic nerve were primary in character this would not exclude the diagnosis of cerebral tumour, though it is difficult to think of such a result except from a tumour situated so as to press on the optic

[1] *Medical Times and Gazette*, 1873, ii., p. 541.
[2] *Medical Ophthalmoscopy*, 3rd edition, p. 166.
[3] *Diseases of the Nervous System*, p. 148.
[4] *Transactions of the Ophthalmological Society*, 1880-81, vol. i., p. 72.

chiasma,[1] whereas in the case now under discussion, the presumption from the evidence is that the tumour is in the cerebellum. The point, however, need not be further laboured, as the evidence of intracranial disturbance is by no means limited to the condition of the optic discs. Further and sufficient proof is provided by the existence of nystagmus, by the absence of the knee-jerks, by the occurrence of "fits" of the *petit mal* order, and by outbreaks of temper which may be taken as evidence of depreciation of the cerebral tissues. Attacks of a similar character are noted in Case 6, where the diagnosis of tumour can scarcely be questioned (see also Case 2).

Taken together, the four cases of the second group confirm and extend the lesson enforced by the members of the first group. They show that the degrees of cerebral disturbance which accompany optic neuritis of intracranial origin may vary widely, and they further demonstrate how largely the results of such disturbance may pass away, though the injury to the organ of sight may be both permanent and pronounced.

It is possible that the question may be raised whether after all these cases are really cases of intracranial tumour, and especially in view of the evidences of recovery which they exhibit. And it may be admitted that reasoning on *à priori* grounds, or considering only the numerous undoubted cases of tumour in which the patients suffer severely, and which pass with comparative rapidity to a fatal termination, there is some difficulty in accepting the diagnosis here suggested. But it cannot be denied that in all the cases there is evidence of definite organic mischief within the skull, and the facts of experience do not seem to offer any choice other than tumour or meningitis. Taking the mere occurrence of recovery into consideration, it is certain that the diagnosis of meningitis, at least of tubercular meningitis, is not less difficult than that of tumour. Then in three of the cases the patients never had in the general sense of the term any illness which could reasonably be regarded as an attack of meningitis, and in those who were at any time seriously ill there were in two cases signs of

[1] Handford, *Brain*, 1892, vol. xv., p. 463.
Hinshelwood, *Glasgow Hospital Reports*, vol. i., p. 4.

a localized lesion in the brain, such as paralysis of a limb or a hemiplegic attack. Possibly, though certainly very rarely, such paralyses may occur in meningitis, but the presumption from such incidents is decidedly in favour of tumour. Again, it is to be remembered that in several of the patients the evidence of active intracranial disease extended over many weeks, and even months—a state of matters difficult to reconcile with a diagnosis of meningitis. And if the cases were cases of tubercular, and presumably therefore of basal, meningitis, the absence of disturbance of the functions of the ocular muscles is certainly remarkable. Further, in the one patient seen throughout the whole course of her very serious illness the temperature, so far as it was disturbed, had a range below the normal level, and two other members of the series seen with more or less acute symptoms were also free from evidence of fever. The degree of the optic neuritis is also of importance in the discussion of this question. Optic neuritis is undoubtedly present in a certain number of cases of meningitis, but it is almost invariably of slight, or, at the outside, of moderate development. But in all the present seven cases—with the possible exception of the last—the degree of optic neuritis either was, or had been, of the considerable or extreme degree usually found with, and indeed strongly suggestive of, intracranial tumour. The balance of probability, even when attention is confined to the facts of the present cases, thus strongly inclines to the diagnosis of tumour rather than to that of meningitis. Other considerations support this view. Thus it is a fact that a tumour, or even tumours, may develop in the brain and yet fail to give the slightest clinical evidence of their existence. Such growths, too, may be of considerable size, and may occupy parts of the brain which, to judge from general experience, are by no means tolerant of disturbance. The cerebellum, the frontal lobe, the occipital lobe, the temporo-sphenoidal lobe, even the motor tract, has each in turn been found to be the seat of a tumour which during life gave no evidence of its presence.[1] A step in advance of these cases is provided by a further series in which, with a tumour

[1] See Table I., p. 126.

of the brain (proved by post-mortem examination), there has existed some, but only slight, evidence of cerebral disturbance, the evidence being in some of these cases equivocal or non-suggestive of tumour.[1] A further contribution to the argument is found in records of cases in which severe symptoms of brain tumour have subsided, to be followed after an interval, and sometimes a long interval, by return of the symptoms and death of the patient, the diagnosis of tumour being confirmed by post-mortem examination.[2] Indeed, the recorded instances of verified cases of brain tumour form a series in which, commencing from those absolutely without symptoms, there may be traced all degrees of severity in the number and degree of the disturbances produced.

In reference to the question of recovery, Hughlings Jackson[3] writes that "it is not at all uncommon for a patient to get rid of all symptoms of tumour except that he remains blind or defective in sight," and in discussing the diagnosis as between meningitis and tumour adds, "But when recovery follows, what are we to say? . . . I still think them to be cases of tumour." T. K. Monro[4] has recorded a case which comes as near actual demonstration as possible of the recovery of a patient from the effects of a brain tumour with the exception of optic atrophy and blindness. The patient, a man of 63 years, died in the Glasgow Royal Infirmary from cancer of the stomach. He was the subject of optic atrophy, and was almost blind. For thirty-three years he had been an inmate of the Blind Asylum, the loss of sight being attributed to a severe illness which confined him to bed for almost twelve months when he was 16 years of age. The knee-jerks were normal. The post-mortem examination showed a myxomatous tumour almost replacing the left half of the cerebellum, with atrophy of the optic chiasma and nerves. Such a record materially strengthens the decision for a diagnosis of tumour in such cases as are now under consideration.

Thus the facts of the present cases, together with the pre-

[1] See Table I. [2] See Table II.

[3] *Medical Times and Gazette*, 1873, ii., p. 197.

[4] *Glasgow Medical Journal*, 1896, ii., p. 173.

sumption arising from a study of other cases, form a strong foundation on which the diagnosis of intracranial tumour seems securely placed. It would certainly be difficult to find anything like so strong a defence for a diagnosis of meningitis.

In reference to the disappearance of symptoms that have become fully established, it has to be borne in mind that the usual clinical effects of an intracranial tumour are due not so much to the pressure or mechanical displacement which the tumour produces as to the vascular and inflammatory disturbances which are excited in its neighbourhood. Given cessation of growth of the tumour, it is to be expected that such disturbances will subside, with a corresponding diminution in the symptoms due to them.

Presuming then that these seven cases are examples of the clinical exhibition of the effects of intracranial tumour, two conclusions follow. First, as already observed, the physician must be prepared to expect in different cases very different amounts of evidence—sometimes of such a nature and degree as to proclaim loudly the existence of intracranial disease, at other times slight in amount, and not manifestly cerebral in origin. It is in these latter cases especially that the ophthalmoscope renders such useful service, for whilst optic neuritis is not present in every case of brain tumour, it exists at some stage or other in the great majority, and not infrequently it is an early, if not the earliest, evidence. Even when existing alone, and more especially when it affects both eyes and is distinguished by much swelling of the papilla, it demands the consideration of cerebral tumour as a possible diagnosis, though, as will be shown later on, it may have exactly the characters just described and yet be due to some other and much less serious cause than tumour of the brain.

The second conclusion which follows from the acceptance of intracranial tumour as a diagnosis in the seven cases now under discussion is, that the prognosis of such cases so far as life is concerned is by no means hopeless. One patient has survived the loss of sight by eleven years and enjoys good general health except for an occasional quasi-epileptic attack, another

has a similar experience extending over four years, a third for three years, and the rest, though less impressive, are similar in the direction of their testimony.

Several parallel series to the seven cases now submitted are on record. So far back as 1866 Mr. Hutchinson[1] tabulated a series of twelve cases of optic neuritis in children following as a rule a more or less severe illness marked by delirium and other head symptoms, and he was able to show by the subsequent histories of the cases that frequently such children regain good health excepting as regards sight. Mr. Hutchinson[2] also reported a number of similar cases in 1879. The precise nature of the primary lesion he considered doubtful, but was disposed to distribute the cases among tubercular meningitis, basal arachnitis, cerebro-spinal meningitis, and some of the exanthemata, as the probable causal conditions. T. K. Monro[3] has published three cases of blindness following double optic neuritis other than the one already quoted, and regards each case as one of intracranial tumour. Indeed it seems difficult to avoid that conclusion in reference both to his and other similar cases unless the accepted clinical significance of the combination of pain in the head, vomiting, and double optic neuritis is to be seriously challenged.

The possibility of recovery from symptoms, and even severe symptoms, of brain tumour must necessarily be considered in determining the line of treatment to be pursued. The recognition of such a possibility cannot fail to infuse not only a more hopeful tone into the prognosis but also increased resolution into the treatment. Probably most will agree that when the diagnosis of intracranial tumour is accompanied by localizing symptoms, surgical interference should be anxiously considered; that in any case where this is not deemed advisable medical treatment must consist of attempts to deal with the more troublesome symptoms; and that in the event of recovery occurring the measures indicated are those

[1] *Royal London Ophthalmic Hospital Reports*, 1866, vol. v., p. 307.
[2] *Ibid.*, 1879, vol. ix., p. 124.
[3] *Glasgow Medical Journal*, 1897, ii., p. 367.

calculated to promote and secure the general vigour of the patient. Recovery however is a matter of degree, and at least two (1 and 5) of the present cases illustrate the dangers which may attend the patient even long after the original illness has subsided—a fact important to the prognosis, and also significant in reference to the anxious care with which these patients should be guarded. Hughlings Jackson[1] is especially emphatic upon the great necessity for caution in framing the prognosis in any case presenting symptoms of intracranial tumour. He points out, not only the liability to return of the symptoms after a period of quiescence, but also the very extended duration of the symptoms in occasional cases. Another danger on which he insists is that of sudden death, which may occur even when the patient appears to be doing well. The extent of this last risk receives striking illustration in a careful and elaborate analysis of 100 cases of brain tumour published by Hale White.[2] There is another point to consider in reference to treatment. All the patients referred to in this paper as examples of recovery from the symptoms of intracranial tumour have this very serious qualification of their recovery, namely, that each one is blind. Recognizing that this is a very likely issue, that it may indeed be a relatively good result of the case, the best perhaps to be hoped for, it is of great moment to consider whether any means can be adopted to secure the patient against so grave a defect. Hughlings Jackson has considerable confidence in large doses of potassium iodide with or without mercurial inunction. There seems no doubt at all of the fact that trephining the skull, altogether apart from the removal of a tumour, is sufficient, at least in some cases, to cause the subsidence of the optic neuritis. This measure has been advocated both by Victor Horsley and Macewen,[3] and Jas. Taylor[4] has also called attention to it. The question is one of great importance, for, from such cases as are recorded here, and from the statement that

[1] *Op. cit.*, p. 195 *et seq.*

[2] *Guy's Hospital Reports*, 1885, p. 117.

[3] *British Medical Journal*, 1893, ii., p. 1365.

[4] *Transactions of the Ophthalmological Society*, 1884, vol. xiv., p. 105.

the neuritis will subside under trephining, there would seem ground for hoping that in a certain number of cases of intracranial tumour where the situation of the growth cannot be localized, operative means may still assist in promoting the patient's comfort, and given a natural tendency for the growth of the tumour to cease, may assist him to many years of good general health and unimpaired vision. One difficulty in urging on the patient what he at least will consider heroic measures is that occasionally all the evidences of intracranial tumour, including optic neuritis, subside spontaneously.[1] Another arises from the fact that optic neuritis may occur apart from intracranial tumour, and whilst at the time of its development and existence it may be impossible to say tumour is not the cause—for undoubtedly optic neuritis may be for long the sole evidence of tumour—the subsequent history of the case renders such a diagnosis in the highest degree improbable.

This latter difficulty is illustrated by Cases 8 and 9 here recorded. In Case 8, not only was there double optic neuritis, but there was also paralysis of the right and possibly slight impairment of the function of the left sixth nerve, an event under the circumstances as significant of cerebral disturbance as well could be. Was such a combination indicative of tumour? In the early stage of the case it seemed impossible to deny that tumour might be the explanation, but the subsequent history renders such a diagnosis very improbable. There were no cerebral symptoms other than the optic neuritis and the ocular paralysis, but it was difficult to conceive of any explanation of these apart from intracranial mischief. No doubt the girl was anæmic, and anæmia is said to be one of the causes of optic neuritis. But anæmia, unless indirectly through the agency of a thrombosis or hæmorrhage, will scarcely produce a sudden diplopia and complete paralysis of a cranial nerve; and optic neuritis is itself a very rare and unusual circumstance in anæmic patients. An examination of every patient admitted to Sir Wm. Gairdner's wards for a period of five years did not reveal a single instance of the occurrence of optic neuritis with

[1] See Table II.

simple anæmia or chlorosis. Considering how very common cases of chlorosis are, how not only relatively but absolutely rare it is to find optic neuritis accompanying that condition, it becomes a question whether, when such association does occur, some new factor has not complicated the problem. The possible gravity of the significance of double optic neuritis certainly renders it most inadvisable to consider the mere existence of anæmia an adequate explanation of its presence, at least until all other known causes have been excluded, and even then, as already remarked, the extreme rarity of the association of the two conditions justifies some suspicion of the accuracy of their suggested relationship.

The case (No. 8) is an example of double optic neuritis existing with a circumstance strongly suggestive of intra-cranial mischief (namely, paralysis of the sixth nerve), yet almost certainly the case is not one of tumour. The difficulty of providing a satisfactory diagnosis is great. A very similar case is recorded by Gowers,[1] but the patient, in addition to optic neuritis and sixth nerve paralysis, suffered from attacks of severe occipital pain and vomiting, and the view taken of all these symptoms was that they were due to a transient localized meningitis. Possibly some help in understanding the present case (No. 8) is afforded by some observations recorded by Bristowe[2] noting the development of optic neuritis in anæmic patients, with evidence of thrombosis of some of the cerebral sinuses and veins. One patient, a girl of 19 years, when under treatment for anæmia, had a severe pain, first in the neighbourhood of the right ear, and later, a similar experience on the left side, with, on each side, tenderness and swelling suggesting thrombosis of the internal jugular vein. She was found to have double optic neuritis, and vomiting and headache were troublesome. After a few days she improved, but convalescence was interrupted by an attack of phlebitis in the leg. Bristowe's suggestion is that the attacks of pain about the ears meant thrombosis in the lateral sinuses, and he supports this by the record of a second case,

[1] *Transactions of the Ophthalmological Society*, 1880-81, vol. i, p. 115.

[2] *Clinical Lectures and Essays on Disease of the Nervous System*, p. 184.

This patient, a woman, æt. 23 years (six weeks pregnant), suffered for twenty-five days from headache, sickness, pain and tenderness over the left mastoid, and general drowsiness; during her illness she developed a right hemiplegia, which had nearly passed off, when she suddenly died, apparently from syncope. There was double optic neuritis. Post-mortem examination revealed thrombosis of the left lateral sinus and internal jugular vein and of some of the veins on the surface of the brain. No evidence of disease in the ear, bones of the skull, or meninges could be found, and there was nothing to suggest any cause for the thrombosis other than the anæmia. These are certainly very important and interesting cases, and they afford a possible explanation of the appearance of optic neuritis in some cases of anæmia. In the present case (No. 8), too, it is not difficult to imagine a thrombosis or resultant hæmorrhage so situated as to interfere with the function of one on both sixth nerves.

The occurrence of double optic neuritis in young women has been attributed to menstrual and uterine derangements, but the pathology of the combination is quite obscure. In connection with such cases, Dr. Bristowe's experience should not be forgotten. In a case reported by Broadbent,[1] in which headache, vomiting, and double optic neuritis had developed on sudden arrest of the menses, death ultimately occurred, and the post-mortem examination showed neither tumour nor meningitis—"the only morbid appearance in the brain was effusion into the ventricles." A second case, apparently of the same order, lost all her symptoms on re-establishment of the menstrual flow, but was blind from optic atrophy.

The eighth case of the present series is certainly not a riddle easily read, but it is at least a demonstration that double optic neuritis, even when combined with an event so suggestive of intracranial disease as paralysis of a cranial nerve, does not necessarily mean a diagnosis of tumour, or at least that such a combination is not inconsistent with a rapid and practically a complete recovery. But how is such a case to be distinguished at the outset from cases such

[1] *Transactions of the Ophthalmological Society*, 1880-81, vol. i., p. 108.

as the earlier members of the series here submitted. For if double optic neuritis and one such symptom as headache (Case 2), or vomiting (Case 3), is sufficient to justify a diagnosis of cerebral tumour, or, at least, to demand a very anxious prognosis, the same must be true of double optic neuritis when associated with such an event as a sixth nerve paralysis. The fact seems to be that there is no absolutely sure ground on which to base a distinction other than that provided by an exact knowledge of the progress of the case. When there is double optic neuritis there is ground for anxiety; whether it means definite organic disease within the skull or not it is in many cases difficult to say. But it is certain that even when there are other suggestions of cerebral disturbance the issue may be most satisfactory, whilst, on the other hand, the neuritis may stand almost alone as a sign of such disturbance, and yet the patient's eyesight, or even his life, be in great peril. In individual cases there may be particular features which will help the distinction between the two classes thus defined, but, on the whole, it is from a study of the development and progress of the case that the most reliable guidance will be obtained. How different, for example, is the character of the movement in Case 8 from what occurred in the earlier members of the series. It is not the mere existence of optic neuritis, of headache, of vomiting, etc., that gives cause for alarm, so much as the persistence, aggravation, or expansion of any one or combination of these symptoms. Further, the knowledge of the progress of the case on which an estimate and outlook are to be based must not be too limited in its range, for there can be no doubt at all that the urgency of the symptoms in many cases of cerebral tumour varies from day to day in the most remarkable manner. In Annie K. (Case 1) this was repeatedly seen; every now and then during the time when a fatal termination seemed probable, a day or two would occur in which pain and vomiting would entirely cease. "You go to a patient one day," writes Hughlings Jackson,[1] "and find him curled up in bed suffering intense pain, and hear that he has been vomiting urgently. Next

[1] *Medical Times and Gazette*, 1873, vol. ii., p. 541.

time you go to see him you find him sitting by the fire reading the newspaper, apparently quite well. *Do not be deceived by these pseudo-recoveries.*" It is a steadily continued, rather than a sudden, diminution of the symptoms which justifies a lessening of the anxiety necessarily born of the discovery of double optic neuritis. This is well seen in Case 9, where there existed double optic neuritis with such a large amount of swelling that, taking the ophthalmoscopic examination alone, the diagnosis of tumour seemed almost inevitable. No doubt the complete absence of all other cerebral symptoms was a grateful sign, and the history—if it could be relied on—of very rapid loss of sight made one think, or rather hope, that some other interpretation was possible. But it was only the steady movement of the case in the right direction that lessened the anxiety of the prognosis and justified an expectation of still better things. What exactly the diagnosis here ought to be may possibly be a question, but the case emphasizes what has just been stated in regard to the questions of diagnosis and prognosis arising in connection with attacks of double optic neuritis. The diagnosis now suggested is that of retro-bulbar neuritis. No doubt such a condition usually affects one eye only, or occasionally one eye, and then after an interval, the second. There seems no reason why it should not attack both eyes at one and the same time. Again, the diagnosis of retro-bulbar neuritis usually includes no appreciable early ophthalmoscopic signs, or only evidences of slight neuritis. But the evidences of papillitis certainly vary widely, and here at the worst it is a matter of degree. The rapid and early loss of sight, the history of pain in the eyeballs, the condition of the pupil light-reaction, the fact of nearly complete recovery, and the restoration of the periphery of the visual fields before central vision was possible, are all in favour of a retro-bulbar lesion. That both nerves were affected at the same time, and that there was also considerable swelling of the optic discs, are interesting features which give individuality to the case. The rapid failure of vision in this case may be usefully compared with the gradual decline of sight in the cases regarded as dependent on organic intra-

cranial disease—probably tumour. In these latter cases it is not as a rule during the stage of swelling and exudation that sight is prejudiced, but rather when the inflammatory movement is subsiding and the newly framed fibrous tissue, gradually becoming more compact, is by pressure injuring the vitality of the nerve elements.

It is of course a matter of common knowledge that double optic neuritis, as in Case 8, may exist without any complaint of defective sight and without any recognized failure when the usual tests for the visual acuity are applied. This was taught by Hughlings Jackson more than thirty years ago. The present cases show three possible conditions of the visual acuity with double optic neuritis. First, vision may be practically unaffected during the whole course of the neuritis (Case 8); it may be almost or completely lost during the stage of exudation and yet may subsequently be entirely regained (Case 9); or, as in the majority of cases, though it may be little or not at all affected during the acute inflammation, it may in the end be entirely destroyed (post-neuritic atrophy).

The diagnosis in all these nine cases may possibly be questioned. As has been already suggested it may be asked whether any of the first seven is really a case of tumour of the brain. On the other hand, it may be urged that Cases 8 and 9 ought, equally with the earlier members of the series, to be placed under that diagnosis, and that they must be regarded as differing from the earlier cases only in presenting slighter clinical evidence and a more complete recovery. It may be impossible to maintain an absolutely confident negative to this latter proposition, just as it is impossible—in view of the fact that occasionally a tumour of the brain is found when no evidence of its presence existed during life—to be quite certain that any particular individual, though apparently in perfect health, may not be the subject of some intracranial growth. One must accept what seem to be the reasonable probabilities of the case. Thus, when in children and young adults (as in Cases 8 and 9) the evidence of intracranial disturbance other than optic neuritis is little or none, when there is relatively rapid subsidence of the optic neuritis

and of any other existing symptom, and when also during such evidences of improvement no fresh symptoms emerge, it is justifiable to regard the optic neuritis as probably non-indicative of tumour, and to speak of the prospects of the patient in fairly confident and hopeful terms. But when the conditions attending double optic neuritis are the opposite of those just stated, the existence of gross organic disease is highly probable if not absolutely certain, and the risks to life, or alternatively to sight, are considerable. Some assistance to prognosis, if not also to diagnosis, is gained by treatment, and if under complete rest, mercurial inunction, with potassium iodide—or in the case of anæmic girls, iron—internally, the symptoms whatever they be do not show with a fair degree of promptitude decided improvement, the probability of organic disease is great, and the prognosis is correspondingly anxious. The differential diagnosis of tumour and meningitis must then be discussed, and in the event of the former being accepted the question of surgical interference at once arises. Unless there are evidences of the exact locality of the tumour, operation with a view to its removal could hardly be proposed, and statistical evidence shows very decidedly that in the great majority of cases of intracranial tumour removal is out of the question. Further, any proposal for an operation must be considered in the light of the fact that in a certain number of cases patients make a good recovery, with or without impairment of sight, under medicinal treatment, or, indeed, with no treatment at all; it must also be recollected that there may be more than one tumour, a state of matters not uncommon in young patients. The practical summing up seems to be that when optic neuritis, with or without other symptoms, is present in children or young adults, such medicinal measures as have already been indicated should be energetically applied. Should these fail and there exist localizing symptoms, as, *e.g.*, attacks of Jacksonian epilepsy, the possibility of surgical removal of the tumour comes seriously into view. In the absence of localizing evidence and the failure of medicinal measures, it is a fair proposal to trephine the skull with a view to relieve symptoms. Undoubtedly

headache and optic neuritis may be relieved in this way, and the patient may thus be rendered more comfortable even if his life is not prolonged. If sight is to be benefited by trephining, the operation must not be too long delayed; it must be undertaken during the exudative stage and before the nerve fibres have been structurally damaged by the pressure of the newly organized fibrous tissue.

There is one other practical point which may be noted. It arises from the possibility of optic neuritis being associated with, or perhaps due to, menstrual or uterine disturbances. Such cases as those reported by Broadbent [1] demonstrate this possibility, and it is supported by various authorities.[2] Hence when the patient is a female, and especially if there is absence of normal menstruation, attention to this function ought to be included in the treatment. The coincidence of suppression of the menses with optic neuritis must not however lead too confidently to the conclusion that the one is the cause of the other and to a correspondingly cheerful prognosis. In Case 1, in which the clinical evidence of tumour is complete, the patient ceased to menstruate at a very early stage of her illness, and the function for a long period remained in abeyance. Presumably the general functional disturbance resulting from the tumour interfered with menstrual activity just as it did with other of the bodily functions. Thus whilst it may be said there is evidence to support the view that optic neuritis may in some way or other result from menstrual irregularity, it must also be allowed that a tumour of the brain may in some indirect manner lead to suppression of the menses.

[1] *Transactions of the Ophthalmological Society*, 1880-81, vol. i., p. 108.
[2] *Ibid*, 1880-81, vol. i., p. 61.

[Tables.

TABLE I.

CASES OF TUMOUR OF THE BRAIN WITHOUT SYMPTOMS OR WITH SYMPTOMS OF ONLY DOUBTFUL SIGNIFICANCE.

Nature and Position of Tumour.	Author.
Cancerous tumour of greater part of anterior lobe, secondary to cancer of the lung. Nothing cerebral observed except manner at times peculiar and replies to questions "odd."	Hughlings Jackson, *Medical Times and Gazette*, 1873, vol. ii., p. 195.
Tumour of each lobe of cerebellum. No disturbance of gait or other motor symptoms.	Hughlings Jackson, *Ibid.*
Psammo-sarcoma, measuring 2 by 1½ inches, growing from falx at junction with tentorium and compressing corpus callosum.	Goodhart, *Pathological Society's Transactions*, 1885-1886, vol. xxxvii., p. 16.
Mixed-cell sarcoma, of considerable size, in ascending parietal and supra-marginal convolutions; also a smaller tumour in occipital lobe.	D'Arcy Power, *Ibid.*, p. 54.
Myxo-chondroma, measuring 1½ inches in longest diameter, springing from the falx and loosely embedded in a depression on the median aspect of ascending parietal convolution.	Hadden, *Ibid.*, p. 71.
Carcinoma, 1½ inches in diameter, affecting dura mater and skull over right orbit. A prominence was observed over the right orbit but no symptoms of cerebral disease.	F. Taylor, *Ibid.*, p. 72.
Psammoma (two), each ½ inch diameter, springing from choroid plexus of each lateral ventricle and occupying symmetrical portions of each descending cornu.	Hadden, *Ibid.*, p. 74.
Tumour, size of Tangerine orange, in occipital and temporo-sphenoidal lobes.	Hale White, *British Medical Journal*, 1886, vol. i., p. 117.
Tubercular mass in cerebellum.	Hale White, *Ibid.*
Tumour in frontal lobe. Only symptom, headache, until shortly before death, when optic neuritis appeared. Death sudden.	Hale White, *Ibid.*
Extensive tumour of orbital convolutions, destroying one olfactory nerve and producing considerable depression in bone. No cerebral symptoms unless "shortness of temper."	Alexander, *Liverpool Medico-Chirurgical Journal*, 1888, p. 253.
Large spindle-celled sarcoma in substance of hemisphere. Patient had occasional fits for ten years—controlled by potassium bromide—but no optic neuritis or other evidence of tumour.	Hadden, *Brain*, 1888-1889, p. 523.
Considerable cystic tumour in anterior part of temporo-sphenoidal lobe. Patient for several years under observation for "epileptic mania." No other evidence of tumour.	Trowbridge, *Journal of Nervous and Mental Diseases*, 1891, p. 217.
Large tumour of pre-frontal lobe. Optic neuritis for a long time, no other symptom until thirty hours before death.	Schweinitz, *Journal of American Medical Association*, 1893, vol. xxi., p. 607.
Numerous tubercular tumours both in sensory and motor regions. Patient, a child, quite free from head symptoms until a day or two before death, when some evidences of meningitis.	Middleton, *Lancet*, 1893, ii., p. 137.

TABLE II.

CASES OF RECOVERY, MORE OR LESS COMPLETE, FROM SYMPTOMS OF BRAIN TUMOUR.

Symptoms.	Sequel.	Author.	Notes.
Girl, 11 years, headache, vomiting, optic neuritis.	Recovery complete except blindness.	Hughlings Jackson, *Med. Times and Gazette*, 1865, vol. i., p. 626.	Patient unable to stand but could walk well with very slight guidance.
Woman, 19 years, headache, paresis of abducent and facial nerves on each side, with double optic neuritis.	Four months after, free from pain and generally well, but some degree of strabismus and facial paresis.	Wilks, *Brit. Med. Journal*, 1870, vol. ii., p. 61.	The diagnosis of tumour suggested but with some hesitation.
Man, 23 years, intense headache, convulsive seizures, optic neuritis.	Recovery to extent of walking about ward and reading newspaper, then sudden death (hæmorrhage from gliomatous tumour).	Hughlings Jackson, *Med. Times and Gazette*, 1873, vol. ii., p. 195.	Illustrates risk that attends even "recovery" from cerebral symptoms (see page 117).
Woman, 59 years, severe paretic and mental symptoms followed by headache, vomiting, and optic neuritis.	Illness extending over 5 months interrupted by periods of marked improvement—one of these lasting for 3 weeks. Death: glioma in white substance of posterior lobe.	J. B. Yeo, *Brain*, 1878, p. 273.	
Woman, 23 years, probable signs of tumour of cerebellum.	A year later general improvement, also some improvement in vision.	Gowers, *Trans. of Ophthalmological Society*, 1881-82, vol. ii., p. 34.	Paresis of upward movement of eyes was a special feature. Tendon-jerks exaggerated.
Woman, 19 years, extreme double optic neuritis with perfect vision for five months.	Then rapid and complete failure of sight, with optic atrophy—no other symptoms.	West, *Ibid.* 1882-83, vol. iii., p. 136.	Patient had been subject to chlorosis.
Headache and optic neuritis.	Recovery under administration of iron.	Hale White, *Brit. Med. Journal*, vol. i., p. 117.	Stated to be a case of anæmia.
Woman, 17 years, diplopia, strabismus, optic neuritis, weakness of lower limbs, absence of knee-jerks.	At end of six months quite well, vision normal. Recurrence of symptoms after 18 months, but again recovery.	James Anderson, *Ophthalmic Review*, 1886, vol. v., p. 121.	

TABLE II.—*Continued.*

Symptoms.	Sequel.	Author.	Notes.
Woman, 18 years, headache, vomiting, tinnitus, optic neuritis. Patient anæmic; no syphilis.	Sodium iodide and mercurial inunction ordered. Patient completely well in two months. Six months later reported, "general health excellent, vision perfect."	James Anderson, *Ibid.*, p. 126.	
Man, 36 years, headache, hemiplegia, delirium, coma, optic neuritis.	Recovery complete under mercuric chloride and potassium iodide.	Suckling, *Birmingham Medical Review*, 1889, p. 159.	Recognized by author as probably a case of gumma though no history or evidence of syphilis.
Woman with headache, vomiting, marked optic neuritis. No history or evidence of syphilis.	Recovery complete under use of potassium iodide.	James Taylor, *Lancet*, 1894, vol. i., p. 133.	Author expresses his view that success of treatment is no proof of syphilitic causation.
Young man with headache, vomiting, intense optic neuritis, and epileptiform attacks.	Recovery apparently complete; two years later reported in perfect health.	Ransom, *Brain*, 1895, p. 534.	Renal disease, syphilis, plumbism, could all be excluded.
Woman, 39 years, headache, diminished mental power, convulsions, optic neuritis; no evidence or history of syphilis.	All symptoms disappeared under mercury and potassium iodide. For many years health good though occasional return of symptoms in a slight form and always yielding to treatment.	Althaus, *Trans. of the Clinical Society*, vol. xxxix., 1896, p. 39.	
Boy, 7 years, headache, vomiting, optic neuritis, paresis of left oculo-motor nerve, knee-jerks absent.	Disappearance of all the symptoms except blindness from optic atrophy. Fifteen months later general health reported to be good.	T. K. Monro, *Glasgow Medical Journal*, 1896, vol. ii., p. 173.	
Man, 63 years, history of severe head symptoms at 16 followed by blindness; knee-jerks normal.	Good health until shortly before death from cancer of stomach. P.M. = myxomatous tumour in cerebellum.	T. K. Monro, *Ibid.*, p. 176.	

TABLE II.—*Continued.*

Symptoms.	Sequel.	Author.	Notes.
Woman, 16 years, serious illness of three to four weeks' duration, severe headache, vomiting, optic neuritis, and ocular paresis. Temperature subnormal, pulse slow, knee-jerks almost abolished.	Recovery complete except for very serious defect of vision (optic atrophy).	Alexander Napier, *Ibid.*, p. 372.	Author regards the case as one of tumour complicated by a transient attack of meningitis.
Girl, 8 years, serious illness with headache, vomiting, constipation, and loss of vision.	When seen at 17, well except for blindness and slight paresis on one side—occasional epileptic seizures since puberty.	T. K. Monro, *Ibid.*, 1897, vol. ii., p. 367.	
Woman, 17 years, some months of headache followed by vomiting and double ptosis; optic neuritis on examination and diminished knee-jerks.	Improvement for some weeks, then relapse, followed by almost complete recovery. Vision said to be "good" but evidence of post-neuritic atrophy.	T. K. Monro, *Ibid.*, p. 369.	

Mr. Hutchinson's cases of double optic neuritis in children associated with severe illness and various head symptoms, and followed by recovery except for partial or complete blindness, are recorded in the *Royal London Ophthalmic Hospital Reports*, vol. v. (1866), p. 307, and vol. ix. (1879), p. 124. In the same publication, vol. vi. (1869), p. 43, Mr. Hutchinson reports the case of a patient who suffered from pain in the head, ptosis, convulsions, and sudden blindness, and who in the course of six weeks, though still blind, was otherwise quite well. The same author [*Reports*, vol. xii. (1889), p. 65] has described two cases of double optic neuritis occurring without explanation in young men; one of the patients was seen eleven years later when he was the subject of double optic atrophy, but his general health was quite undisturbed.

Whatever may be the explanation of these cases, they manifestly have a very close clinical relationship to those recorded with this paper. The earlier ones afford abundant confirmation of the statement that double optic neuritis even when associated with severe cerebral disturbance by no means demands anything like a hopeless prognosis as regards the life of the patient, though the risk of permanent damage to vision is great. The last two cases further show that this danger to vision may also exist even when no cerebral events accompany the optic neuritis; in the parallel case (No. 9) of the present series the patient regained almost perfect vision.

Further cases illustrative of a capacity for recovery from severe symptoms of cerebral disease will be found in the late Dr. Bristowe's *Clinical Lectures on Diseases of the Nervous System*. And of course on all points concerning the diagnosis and prognosis in cases of intracranial tumour invaluable assistance is to be gained from the several papers of Dr. Hughlings Jackson. See *e.g. Royal London Ophthalmic Hospital Reports*, 1863-65, vol. iv., p. 389 *et seq.*; *Medical Times and Gazette*, 1873, vol. ii., p. 139 *et seq.*; *Transactions of the Ophthalmological Society*, 1880-81, vol. i., p. 60; and *British Medical Journal*, 1888, ii., p. 59.

CASE 1.

Severe illness characterized by symptoms of cerebellar tumour and followed by recovery except for total loss of sight (optic atrophy). Return of symptoms after two years' interval of good health, with subsequent complete convalescence. Patient reported free from all symptoms, except blindness, six years after first illness.

Annie K., æt. 16 years, admitted to Glasgow Western Infirmary July 28, 1893, complaining of vomiting, pain in the forehead, and unsteadiness of gait.

She enjoyed good health until two months before admission, when vomiting began, at first only on rising in the morning, but afterwards also throughout the day, both immediately after food and in the intervals between meals. In a week or so frontal pain became troublesome; the degree of it has varied much, but it is always present to a greater or less extent She has been told that her eyes have recently become more prominent, the amount of protrusion varying from time to time. Her sight has been so bad that on several occasions she has run against objects in the house and street. Still, three weeks ago she went out to domestic service, though she soon found herself unfit for this, chiefly, it would seem, on account of her defective sight. Shortly after returning home she began to be very giddy at times; to stagger, with an inclination to fall backwards, when walking; and more recently she has complained of pain in the right side of the neck. Menstruation, which previously had been regular, has ceased during the last two months.

Patient is a well-nourished, healthy-looking girl. She is somewhat lethargic, and is slow in answering questions, but quite intelligent. There is marked retraction of the head. When assisted to rise she becomes giddy, and tends to fall backwards, indeed she would fall unless supported.

The pupils are equal and contract fairly well both to light and in convergence. In each fundus there is marked papillitis; visual defect is considerable; no squint or nystagmus; possibly

slight exophthalmos on each side. No tenderness on percussion of the skull. The superficial reflexes are normal; each knee-jerk is very marked—almost certainly exaggerated; cutaneous sensibility seems everywhere normal, and there is no paralysis of the limbs.

Thoracic and abdominal viscera normal to physical examination, and urine normal except for some phosphatic excess.

The sense of smell is very defective; she has observed this herself. Hearing not definitely abnormal.

The condition of patient during her first month of residence varied very much. Occasionally she would seem fairly comfortable, but for the most part frontal pain was extreme and vomiting very obstinate. At times she passed into a semi-comatose condition and appeared very likely to die. The bowels could not be evacuated without enemata, and unless the catheter was regularly used the urine was passed in bed. Temperature almost invariably subnormal; pulse from 60 to 72. The swelling of each optic papilla became less, the light response of the pupils diminished, and the vision was reduced to mere perception of light. Tested on several occasions the knee-jerks were found to be absent.

On August 23rd some evidence of right facial paresis was observed, the upper as well as the lower muscles being involved; general condition somewhat improved.

On the 2nd of September she somehow or other fell out of bed, but apparently did not hurt herself. The nurse, however, noticed that during the night she did not move her left leg, and on examination the limb was found absolutely paralysed. The knee-jerk at this time was quite decided on each side. She was now again very ill, the retraction of the head very marked, mental faculties much obscured, and urine repeatedly voided involuntarily.

By the middle of September she was much better again, frontal pain and vomiting much less, retraction of head much less, power returning to left lower limb, and no advance of facial paresis. On the 19th the knee-jerk was found absent from the left side, and only a trace present on the right.

There were one or two occasions during November when

pain in head and vomiting were troublesome, but on the whole the patient made decided progress. She gained flesh and became bright and cheerful, though she was quite blind (optic atrophy); there was also marked horizontal nystagmus. The functions of the bowels and bladder became normal, and apparently complete voluntary power was regained over the left lower limb; the evidence of right facial paresis had also receded, and the sense of smell somewhat improved.

When she left the hospital on December 4th she could stand steadily and without giddiness, and could walk with very slight assistance. Examination showed a very marked left knee-jerk, the right being both slight and sluggish.

The most impressive feature of this case was the recovery of the patient after her condition for many weeks had seemed practically hopeless. The facts of the case show the diagnosis of intracranial tumour "writ large," and evidence is not wanting to show that the situation of the tumour is probably the right hemisphere and middle lobe of the cerebellum. A combination in aggravated form of vomiting, vertigo, and optic neuritis is in itself, though not conclusive, sufficient to suggest a cerebellar growth; marked retraction of the head, and possibly nystagmus, are also not without significance in the same direction. The difficulty in maintaining equilibrium and the tendency to fall backwards indicate disturbance of the middle lobe. Paralysis of the left lower limb associated with right facial paresis show that the pressure of the growth is mainly towards the right side, so as to involve part of the motor tract above the decussation, and the trunk of the facial nerve. This is in harmony with the usual experience of paralyses produced by pressure of a laterally situated cerebellar growth. It is noteworthy that the headache was entirely frontal, showing how little reliance is to be placed on the site of the pain as indicative of the situation of the tumour. The varying character of the knee-jerks and the presence of anosmia—a rare result of intracranial tumour—are also special features of the case.

Patient was again under observation from October 10th to November 27th, 1895. In the interval she had for the most

part enjoyed good health, but, recently, frontal pain, vomiting, and giddiness had troubled her. Under rest in bed, etc., these symptoms subsided, and she left the hospital in good general condition, and with no disturbance of gait other than might well be due to her blindness. Knee-jerks *in statu quo.* In June, 1899, patient's sister writes: "A. is never very strong, but she is free from her old symptoms, except the blindness."

The risk of recurrence of the symptoms after even a considerable period of quiescence, and the possibility of a second "recovery," are thus further features of clinical interest in the record of this case. (See also Case 5.)

CASE 2.

Absolute blindness, with marked papillitis and subsequent optic atrophy in right eye (and presumably also in left), with history of severe pain in head, and, later, disappearance of knee-jerks; patient seen in good general health eighteen months after onset of symptoms.

Martin T.,[1] æt. 10 years, complaining of defective sight and pain in head, May 1, 1898. Present illness commenced ten months ago. Pain in the head has at times been very severe, apt to come on in the evening and keep patient from sleeping. The loss of sight has been of gradual development. At no time has it been necessary to confine patient to bed, and there have been no convulsions and no complaint of giddiness. Vomiting has occurred occasionally, but always after taking milk. No history of a fall or blow on the head.

Patient is a pale, delicate-looking boy; general physiognomy not suggestive of hereditary syphilis; he is almost absolutely blind. The left cornea is hazy from interstitial keratitis, and evidences of the same condition exist in the right eye. The pupils show very defective light response. In the right fundus there is marked papillitis which is showing signs of retrogression. Midway between the disc and macula is a small patch of choroidal atrophy, and to the inner side of this

[1] Reported by permission of Mr. Ernest Clarke, F.R.C.S.

is a group of yellowish-white, highly refractive dots. The left fundus cannot be seen on account of the corneal haze, which, however, is not sufficient to account for the extreme visual defect.

Patient walks well and quite without ataxia; knee-jerks present but almost certainly defective. Superficial reflexes present except plantar. No evidence of disease in thoracic and abdominal viscera; urine free from albumen; some enlargement of cervical lymphatic glands.

Family History.—Father and mother alive and well; mother lost a sister from consumption. Patient is youngest of a family of eight; of these two died of "convulsions," one with "something in her brain" and one—the first-born—at seven months, cause unknown. The other children are said to be healthy (two examined show no evidence of hereditary syphilis).

Patient was kept under continuous observation for two months. At times he complained of pain in his head and displayed a very violent temper, but his appetite was good, he never vomited, and his general nutrition improved. Examined repeatedly during this period the knee-jerks were completely absent. The only other change was the advance of optic atrophy in the right eye; the details of the left fundus could never be seen. In the beginning of December, 1898, the boy was again seen; he seemed quite cheerful and fairly vigorous. Blindness was absolute. Knee-jerks absent.

Presuming that this is a case of intracranial tumour, the only indication of the site of the growth is afforded by the loss of the knee-jerks—a fact which points with some degree of directness to the cerebellum. The probabilities are that the tumour is tubercular in nature even though the boy is undoubtedly the subject of hereditary syphilis. Syphilitic tumours of the brain are not common in children whereas tubercular growths are frequent, and the cerebellum is their most usual site. The family history seems distinctly tubercular. The absence of evidence of tubercle in any other part of the body—unless possibly the cervical lymphatic glands—does not mean much in a child, though in adults, according to

Gowers, "the signs of phthisis are rarely absent when there is tubercular tumour of the brain." Further, the boy appeared to receive little or no benefit from antisyphilitic treatment, whilst he improved considerably under the use of iodide of iron and cod liver oil.

CASE 3.

Double optic neuritis followed by blindness and optic atrophy; history of vomiting, with slight headache every morning for six months preceding loss of sight, but no other cerebral symptoms and no "illness" confining patient to bed even for a single day; knee-jerks present, though right possibly defective.

Jas. C., æt. 13 years; examined on April 25, 1898. His complaint is loss of sight. According to the father, the boy had perfectly good sight until the early part of January, when he fell in the street and struck the back of his head against the pavement. No apparent injury followed the fall, but in the course of a fortnight the boy complained that he "could not see well," and soon he appeared to become quite blind. It seems, however, that for six or seven months before the fall the patient was troubled with vomiting, which occurred daily, but only in the morning before breakfast. After each act of vomiting he had some pain in his forehead for a time, but he was always able to go out and distribute newspapers and then to return and take breakfast, and afterwards to go to school. The vomiting still continues, but is entirely confined to the early morning. At no time has the boy been confined to bed or regarded as seriously ill, and as a proof of the comparatively recent failure of his eyesight it is mentioned that he won a prize for drawing at Christmas. It is solely the blindness which, in the view of the father, constitutes the lad's disability.

The boy has not even perception of light in either eye; the pupils are dilated, and show no light response; each fundus provides evidence of consecutive optic atrophy.

He is a fairly vigorous-looking boy, quite free from evidence of hereditary syphilis. There is some enlargement of the submaxillary lymphatic glands, but no evidence of disease in the thoracic or abdominal viscera.

Knee-jerk present on each side, the right not being easily provoked and less distinct than the left (confirmed at a later date); no ataxia in gait.

Family History.—Of three children born before patient two died from "suppressed measles," the third being alive and in good health. Since the birth of patient there have been eight pregnancies, all terminating at full time—three of the children survive; the other five all died when about eight months old from "wasting"—diarrhœa, vomiting, and swelling of belly. Father and mother in good health, and with reputed good family records.

There is nothing in this case to definitely indicate the site of the tumour. Perhaps all that can be said is that the family history is marked by a number of events almost certainly tubercular, and that the most frequent situation of tubercular tumours is the cerebellum. Some difference between the degree of the knee-jerks can hardly be claimed as of definite diagnostic value. The principal value of the case is the demonstration it affords of the great importance of giving the most careful attention to any symptom of possibly cerebral origin, and more especially if such a symptom—even though existing alone—is extended over a considerable interval of time. It is practically certain that grave intra-cranial disease in this case produced for some considerable time no evidence of its existence other than an attack of vomiting, with some frontal headache, every morning. Then followed optic neuritis, which did not seriously affect vision until it began to pass into post-neuritic atrophy. Whether the diagnosis of tumour be justifiable or not, the practical importance of such a sequence of events as is here presented is obvious.

CASE 4.

Serious illness at 15 *years, marked by frontal pain, vomiting, and failure of sight; four years later patient seen in good health, but the subject of post-neuritic optic atrophy.*

Rose M., æt. 19, complaining of defective sight; examined August 7, 1898.

The failure of sight is referred to an illness which confined her to bed for six weeks in the summer of 1894. The symptoms of this illness were obstinate vomiting, pain in the forehead, failure of sight, and general weakness. The medical man in attendance (now deceased) is reported to have said that she had a tumour of the brain. Gradually these symptoms subsided, but the sight, though it improved somewhat, soon reached the condition in which it is at present. No history of injury to the head. Menses have always been somewhat irregular; she cannot say there was anything unusual in this respect at the time of her illness four years ago.

Patient is a tall, healthy-looking young woman. R.V., counts fingers at 6 feet, and reads J. 16 at 8 inches. L.V., hand movements only.

Each pupil is dilated, and exhibits only a feeble light response. In each fundus the disc is white, with "fuzzy" edges, arteries small, and vessels at centre of disc concealed by a filmy haze. There are distinct "white lines" along many of the vessels; at the left macula there are fine retino-choroidal changes. The knee-jerks are distinct, and the gait free from evidence of ataxia. Cardiac sounds and pulmonary physical signs normal: some, but by no means marked, evidence of anæmia.

Family History.—Father and mother alive, and in good health. Patient is fourth in a family of ten—one brother was killed in an accident, the others are all strong and healthy. She has lived all her life in a small village in the west of England.

The history and existing post-neuritic atrophy show the

three symptoms, viz., headache, vomiting, and optic neuritis, which, to quote Hughlings Jackson, "make the diagnosis of an 'adventitious product' within the skull almost certain." That the facts justify such a diagnosis in the case of a patient apparently exceptionally favoured as regards hereditary and other influences seems most unaccountable. The pathetic spectacle of a seemingly healthy and vigorous girl reduced to hopeless blindness may well emphasize the urgency of every possible measure which may conceivably help to avoid so disastrous an issue.

CASE 5.

Acute evidence of intracranial disease of three months' duration in boy of 7 years, followed by recovery, with the exception of blindness (optic atrophy) and loss of knee-jerks; return of headache, vomiting, etc., with a fresh attack of papillitis after an interval of three years; repeated subsidence of all symptoms of active disease, and boy six months later known to be in good health.

Jno. D., æt. 10 years, seen in December, 1896. He is reported to be blind, but otherwise in good health. Some eighteen months ago he had a very serious illness, being confined to bed for nearly three months, and at times he was thought to be dying. The most troublesome symptom of this illness was vomiting; he also had pain in his forehead, his head was drawn backwards, and his sight failed. The diagnosis at the time was "tumour of the brain." He is now a plump cheerful-looking boy. He is quite blind, and the pupils have no light response; in each fundus there is decided evidence of a former papillitis, now in the atrophic stage. Knee-jerks absent, gait normal, memory and mental condition generally seem quite acute. Thoracic and abdominal viscera healthy.

The boy is said to have had a brick fall on his head many years ago.

July, 1898. The boy has been in good health until six weeks ago, when vomiting returned, accompanied by diarrhœa, and continued until within the last four days; there

has also been pain in the head (frontal); at times he has seemed to be unconscious, and on two occasions has had a "fit," in which he became "stiff," but not convulsed. He is now much better in every respect, and indeed he says he is quite well. The ophthalmoscope shows the addition of a recent papillitis to the atrophic condition formerly noted. Knee-jerks are absent.

In January, 1899, the boy is reported to be "stouter and more cheerful than ever." The family history shows a twin brother and three younger brothers and sisters alive and well. Father and mother also enjoy good health; no knowledge of family liability to chest or other disease.

This case, like Case 1, shows how liable the symptoms of intracranial disturbance are to return after even a considerable period of quiescence. Every care should therefore be taken to reduce the risk of this possibility by maintenance of the general health and by the use of such agents as cod liver oil, iodide of iron, etc.

There is one feature attending the recrudescence of the symptoms here which is very unusual, viz., a second attack of papillitis in discs atrophied as a result of the first attack. Gowers in his work on *Medical Ophthalmoscopy* writes, "If a disc has become completely atrophied it is very rarely again the seat of inflammation." Cases of second attacks of papillitis following complete optic atrophy have, however, been observed by Hughlings Jackson and by Schweinitz. "When atrophy is partial or absent, in rare cases two attacks of neuritis may occur" (Gowers).

CASE 6.

Blindness (post-neuritic optic atrophy) and occasional quasi-epileptic attacks, but otherwise good health, in a girl of 16 *years, who two years before was for several months seriously ill with symptoms of active intracranial disease.*

Ada L.,[1] æt. 16 years; examined on June 20, 1898. She complains of blindness. Patient is a delicate-looking girl.

[1] Seen in consultation with Dr. Jas. Smyth.

The pupils are dilated, and have only a slight response to light; they move freely in convergence. Each fundus shows evidence of a former neuro-retinitis with marked atrophy of the optic papilla. Blindness appears to be absolute. The cardiac sounds are pure; R.M. over lungs abundant and free from râle; nothing noteworthy in physical examination of abdomen.

The first note of the knee-jerks is that they are "present," subsequently the right one is "hardly obtained," and the left elicited only with difficulty. The gait is free from ataxia.

From May to November, 1897, she was very ill, suffering from attacks of giddiness, severe pains in the head (which was "drawn back"), and gradual failure of sight. In the course of this illness she had two "fits," attended by unconsciousness, and followed by "paralysis of one arm and leg." No history of injury to head or of discharge from either ear. With the exception of the defect of sight she has been improving since the beginning of the year, and is now able to walk about undisturbed except by an occasional pain in the head; there is no sign of hemiplegia. Her mother adds that patient at times has some kind of "fit" or "attack" which she cannot well describe. There is no loss of consciousness and no convulsion, but patient calls out "I am worse," and clutches hold of anyone who may happen to be at hand. Some of the attacks occur at night-time, and on several occasions there have been involuntary evacuations.

Family History.—Father and mother enjoy good health; the latter says a number of her relatives in the earlier generations died of consumption. There are three other children besides patient, and these are alive and well. No deaths; no miscarriages.

CASE 7.

Blindness and bilateral optic atrophy without evidence of previous neuritis or papillitis in girl of 21 ; *other evidences of* (*presumably*) *intracranial disease being nystagmus, quasi-epileptic seizures, explosive temper, and absence of knee-jerks. History quite free from evidence of any acute cerebral attack, the loss of sight occurring quite gradually when patient aged* 11 *years, and unaccompanied by any other symptom of intracranial or other disease.*

Edith H., æt. 21 years; examined on July 4, 1898. Her statement is that she is quite blind.

Until 11 years of age had good sight, and was in every respect strong and well. Then gradual loss of sight came on, and slowly progressed until in the course of twelve months or less she became quite blind. The defect was first noticed by the girl making mistakes when reading and tumbling over objects when walking. At no time did she complain of pain in her head or of vomiting. (Pressed on this point, she says that about a year ago she often used to vomit after breakfast, but with this exception she has never been troubled with vomiting.) She is sure that the failure in her sight was not accompanied by any general illness, and she was never confined to bed. No history of injury to head. For the last three years she has been subject to occasional fits in which she "gets stiff but does not fall, and is unconscious for a minute or so." She is liable to outbursts of temper. But her general health is and always has been very good; menses quite regular during last five years. Patient is a healthy-looking woman. She is quite blind, and the pupils which are widely dilated have no light response. The optic disc on each side is very white, the retinal arteries, and perhaps also the veins, diminished in calibre. Neither in the disc itself nor in the fundus generally is there evidence of a previous neuro-retinitis. There is marked horizontal nystagmus (present for five years). Knee-jerks absent ; no ataxia in gait. Examination of chest and abdomen entirely negative.

Father, mother, three other members of family—older than

patient—have quite good sight and satisfactory health. No knowledge of fits, nervous disease, or defective sight in any relatives.

The reasons for placing this case as one of intracranial tumour have been advanced in the body of the paper (p. 111). The failure of sight without any other suggestion of intracranial mischief is remarkable. It is another example of how grave intracranial disease may for a long time produce only slight signs of its existence.

CASE 8.

Paralysis of sudden onset of right external rectus, also double optic neuritis, but no other evidence of cerebral disease, and no considerable visual defect other than diplopia; patient, a girl of 17 *years, somewhat anæmic, but otherwise physical examination normal. Recovery complete and apparently permanent.*

Maud E.,[1] æt. 17, cook, complaining of double vision (June 7, 1898). The diplopia is of a fortnight's duration; it was of sudden onset, a group of children at whom patient was looking "suddenly becoming double." She has no other ground of complaint. Patient has always been healthy, though admits some recent loss of colour, breathlessness on exertion, and unduly long menstrual intervals. She was menstruating at the time the diplopia developed.

There is almost complete paralysis of the right external rectus muscle; tested with a flame and coloured glass there is homonymous diplopia increasing to the right; she also recognizes increasing separation of the images to the extreme left, but there is no appreciable defect in the action of the left external rectus. No ptosis or other defect of ocular movement, and pupils equal and with normal reactions.

R.V. 6/6 ·5 Hm. J. 1 at 5 inches.

L.V. 6/6 ·5 Hm. J. 1 at 5 inches.

There is marked optic neuritis in each eye—the disc is moderately swollen, with numerous hæmorrhages on the

[1] Seen in consultation with Dr. D. O. Macgregor.

surface of the swelling; the vessels as they leave the disc are concealed by exudation, and the veins generally are enlarged and tortuous.

The visual fields for white and colours are normal, and there is no central or other scotoma.

Physical examination reveals nothing beyond a venous hum at the root of the neck and a blowing murmur with each expansion of the subclavian arteries. Urine free from albumen and sugar.

She has but an incomplete knowledge of her family history, all she can say is that her mother died of "consumption and heart disease."

Patient ordered to take complete rest, and iron prescribed.

21st June. General condition improved; papillitis somewhat less marked; she does not read 6/6 fully with either eye; paralysis of right external rectus less complete.

25th July. Improvement still more manifest. There is now only a slight degree of paralysis of external rectus. With the right eye she makes mistakes in reading both 6/9 and 6/6, and with the left does not read 6/6 fully. There are now definite changes at each macula, a circular area of a deep red colour and about the size of the optic disc occupies the centre of each fundus, and on this, more especially in the right eye, are a number of irregular, whitish, lustrous spots. Papillitis subsiding.

2nd August. The patient is looking very well. There is now hardly appreciable defect in the outward movement of the right eye, but homonymous diplopia can still be detected to the extreme right when the flame and coloured glass are used. The greater part of the margin of each disc can now be seen; fundi otherwise unchanged. Visual acuity as before; recognizes all colours in 1 mm. square in central vision.

30th August. Improvement continues. The discs are nearly normal and the congested areas in the macular regions less prominent. There is no diplopia even to the extreme right.

20th September. Patient regards herself as quite well, and appears to be so. The fundus on each side remains

as before, and there is slight defect in the visual acuity of each eye.

She is allowed to return to work; to continue to take iron for another three months.

30th March, 1899. In good health. Evidences of former neuritis readily appreciable in each fundus, but vision of normal acuteness.

The difficulties of diagnosis in this case have already been discussed (p. 118). As the patient was undoubtedly anæmic, it seemed right to order complete rest and the administration of iron. Had there not been decided improvement under this treatment it would have been wise to have tried mercurial inunction (see Case 9).

CASE 9.

Rapid failure of vision in girl of 14 *years, with marked double papillitis not accompanied by headache, vomiting, or any other suggestion of cerebral disease; prompt improvement under mercurial inunction, the visual acuity reaching almost to the normal level within three months. Patient after an interval of nine months found to have nearly full vision, normal visual fields, and good general health.*

Ellen D.,[1] æt. 14, seen on June 15, 1898, complaining of failure of sight in each eye. She could see quite well until a week ago; at that time she had a shooting pain in the right eye, and the sight of the eye rapidly failed. A similar experience soon occurred in the left eye. No pain in the head and no vomiting, and general health quite unaffected.

R.V., denies even perception of light. L.V., hand movements only. Pupils dilated: right no response to direct light, but contracts in consensual stimulation; left, considerable light response, but contraction not well sustained. No ophthalmoplegia externa. Right fundus shows decided papillitis (fully +3 D), with a number of hæmorrhages on the surface of the exudation; similar but less marked condition in left eye.

Movement of the eyes in any direction does not cause pain, and palpation of the globes scarcely causes complaint.

[1] Reported by permission of Mr. Poulett Wells, M.B.

Patient is well nourished; no evidence of anæmia; physical examination of chest and abdomen normal; urine normal. Knee-jerks normal, no ataxia in gait, superficial reflexes easily obtained, cutaneous sensibility and motor functions undisturbed. At no point is the skull tender to percussion. She has never been very strong, had rickets in infancy, jaundice four, and measles two years ago. No history of injury to head or of discharge from ears. She has never menstruated.

Family History.—Father alive and well; his mother and a sister and brother died of consumption; mother lost a brother from the same disease. Patient is the eldest of a family of four—one died of consumption, the other two are "delicate."

6th July. Mercurial inunction has been used every night. She can now count fingers in the peripheral part of the field of each eye, but not with central vision, and can move about the ward without stumbling against objects as she did on admission. The papillitis has distinctly subsided.

6th August. Has been away at seaside. Vision still improving. R. counts fingers at three feet in central vision; L. = 6/24. Contraction of pupils under light much more marked, but that of right is not well sustained. Discs white and filled in.

13th August. R.V. = 6/18; L.V. = 6/12 (less one letter).

3rd September. R.V. = 6/9 (two letters); L.V. = 6/9 nearly. Pupils not definitely abnormal.

17th September. R.V. 6/6 part; L.V. 6/6 part. Fields full, no central scotoma, recognizes all colours through an opening 1 mm. sq. Whiteness of discs very decided, and arteries look small.

June, 1899. Patient is in good health, and the visual acuity is almost, though not quite, up to the normal level. Objective examination of the eye negative, except for evidences of former papillitis.

It is suggested that this is a case of retro-bulbar neuritis affecting both sides, and with a greater degree of papillitis than is usual, and the reasons for holding this diagnosis are given in the body of the paper (p. 122).