

THE FALSE LOCALISING SIGNS OF INTRACRANIAL TUMOUR.

BY JAMES COLLIER, M.D., B.Sc. F.R.C.P.

*Assistant Physician to St. George's Hospital and to the National Hospital,
Queen Square.*

THIS communication is based upon the results of 161 consecutive cases of intracranial tumour which have been examined in the Pathological Laboratory of the National Hospital during the years 1894-1904. The examinations were made by Dr. Colman, Dr. Risien Russell, Dr. Batten, Dr. Farquhar Buzzard, and by the writer of this paper.

The majority of these cases came under the writer's personal clinical observation. His best thanks are here expressed to his colleagues upon the staff of the National Hospital, to Dr. Colman, and to Dr. Farquhar Buzzard, Pathologist to the Hospital, for their kind permission to make use of the clinical and pathological notes upon these cases.

The object of this paper is to show :—

(1) That local signs appearing late in the course of intracranial tumour, where general signs alone have pre-existed, are often of false portent.

(2) The relative frequency with which local signs have been due in this series of cases to the presence of vascular lesions, meningitis, hydrocephalus, local spreading œdema of the brain, secondary deposits of new growth, and posterior degeneration.

(3) That the absence of usually accepted local signs during the early days of illness in intracranial tumour is in itself a most important localising indication, confining the disease to the supratentorial region.

(4) That true localising signs at one time present may later become concealed or undemonstrable owing to the

development of other signs, and that in cases which come under observation for the first time late in the disease, diagnosis may be difficult, erroneous, or impossible.

The relative frequency with which the different parts of the brain were the seat of new growth in this series of cases was as follows:—

Frontal	24
Central	20
Parietal	7
Occipital	3
Temporo-sphenoidal	12
Centrum ovale	17
Basal ganglia	14
Brain stem and pons	22
Cerebellum	26
Other situations	16
Total ...					161

THE FALSE PORTENT OF LATE LOCALISING SIGNS.

The meaning which the writer wishes to convey by this heading will be at once apparent in the following examples:—

A man, aged 20 years, suffered with headache, vomiting, and optic neuritis. Seven months after these symptoms appeared he was totally blind from post-neuritic atrophy. In the fifteenth month of his illness localising signs began to appear. In the seventeenth month his condition was as follows:—Paralysis of left external rectus. Marked nystagmus with the slow movement to left. Complete nerve deafness of left ear. Left peripheral facial paralysis. Left cerebellar position of head (head inclined to left shoulder, and face rotated to right). Marked head retraction during paroxysms of pain. Bilateral ataxy left > right, with lurching to left on the attempt to walk. Knee-jerks: left, normal; right, diminished. Double extensor response. His condition remained unchanged until death in the twentieth month.

The localising symptoms were apparently conclusive of a growth in the left posterior fossa involving the cerebellum and the sixth, seventh and eighth nerves. The autopsy

revealed a glioma of the left prefrontal region. The cranial nerves seemed normal to the naked eye, but the intramedullary portion of the left eighth nerve showed degeneration. The cerebellum was much indented by the edge of the foramen magnum, the left lateral lobe forming more of the pressure cone¹ than the right.

In this case the entire absence of localising symptoms during the first fifteen months of illness, when several general symptoms were present, not compatible with the diagnosis of tumour in any but a "silent" region of the brain. The late development of signs indicative of a lesion in the posterior fossa was the indirect and secondary result of a tumour situated at a distance.

A child, aged 8 years, suffered with headache and vomiting, and developed, within a few weeks, well-marked optic neuritis, and signs of a left-sided cerebellar lesion. These signs persisted with exacerbations and remissions for twelve months, when convulsive seizures, commencing in the left hand and being confined to the left arm, occurred, without loss of consciousness. The Jacksonian attacks were repeated at intervals, and in some of the later attacks the convulsion became general and consciousness was lost. The child died after an illness of eighteen months. A subtentorial tumour was found, which had probably arisen in the left cerebellar hemisphere, and had extended into the right cerebellar hemisphere. The fourth ventricle was much occupied by growth, and the aqueduct was obstructed. The ventricles were considerably distended, and the dilatation was more marked in the central region of the right lateral ventricle. There was no local lesion in the right central region that would account for the Jacksonian attacks other than the thinning of the ventricular wall.

This case shows that local convulsions are not always of localising value, and that they may be symptomatic of ventricular distension.

¹In many cases of intracranial tumour of long duration, it is found *post mortem*, that the posterior and inferior parts of the cerebellum have become pushed down and backwards into the foramen magnum, and the medulla itself being somewhat caudally displaced, the two structures together forming a cone-shaped plug tightly filling up the foramen magnum.

False localising signs were met with in twenty cases out of 161 cases in this series = 12·5 per cent. In two cases the false localising signs were due to vascular lesions (hæmorrhage, one case and thrombosis one case), situated at a distance from the growth.

In the rest of the cases the false signs were attributable to the indirect intracranial results of intracranial new growth. They were met with in 13 per cent. of the supratentorial tumours, and only twice among fifty-four cases of subtentorial tumours.

The following localising signs occurred either singly or in combination as false signs :—

Paralyses of cranial nerves.

Hemianopia.

Jacksonian epilepsy.

Bilateral spastic paresis.

Cerebellar signs.

PARALYSIS OF THE CRANIAL NERVES.

The Olfactory Nerves.—Anosmia was only found in this series, as the direct result of local pressure from the growth, upon the olfactory tract and bulb. There was not the least evidence of the occurrence of olfactory neuritis comparable with optic neuritis, as some authors have argued. The writer has, moreover, examined a large number of cases of long-standing intracranial tumour, in which optic neuritis has caused blindness, and has not found anosmia to be more frequent among them than among patients suffering with diseases other than of the nervous system.

Hemianopia was met with in two cases.

In one case the tumour was situated in the left upper central region and was of large size. The absence of the hemianopic pupillary reaction suggested that the right hemianopia present was due to interference with the left optic tract. The hemianopia made its appearance, together with paralysis of the left fifth and right sixth nerve, four and a-half months after paralysis of the right leg had become complete. There was no *post-mortem* evidence of abnor-

malinity of any part of the optic path or of the fifth and sixth nerves.

In the second case right tract hemianopia and left trigeminal paralysis and anæsthesia appeared four and a-half years after the onset of the earliest symptoms in a case of left prefrontal tumour.

The association of right tract hemianopia with left trigeminal palsy strongly suggested that preponderant indirect pressure upon the left middle fossa by a tumour in the left cerebrum had determined the combination.

Paralysis of the third nerve was met with in two cases. It was associated with paralysis of the sixth nerve, and appeared later than the sixth nerve paralysis.

One of the cases was that of a large growth in the sub-cortex of the right temporo-sphenoidal lobe. Seven months after the development of optic neuritis, paralysis of the right sixth nerve appeared, followed in a few days by paralysis of the left sixth nerve. Three weeks later paralysis of the right third nerve appeared, followed in a few days by paralysis of the left third nerve.

The second case was that of a large prefrontal growth upon the left side. Nine months after the onset of symptoms the left sixth nerve became paralysed, then the right sixth, and, some time later, the left third nerve became paralysed.

The important facts which these two cases illustrate are: (1) That the third nerve palsy occurred subsequently to paralysis of the sixth nerve, and (2) that the nerves were affected earlier upon the side of the lesion.

If the paralysis of these nerves is the result of shifting backwards of the brain stem from supratentorial pressure, the effect being traction upon the nerves attached to the brain stem, in proportion as their direction is more nearly a frontal-caudal direction, then the shifting backwards of the brain stem would cause paralysis of the sixth nerve first, then of the third nerves, and lastly of the seventh and eighth, while the nerves which are transversely directed or with sinuous course would be little affected. Such traction would obviously be first manifested upon the side

where supratentorial pressure is greatest, that is upon the side of the lesion.

Paralysis of the fourth nerve was not met with as an indirect effect of intracranial tumour. The course of this nerve from its origin to its point of passage through the dura mater is so sinuous that traction could hardly be exerted upon this nerve by backward shifting of the brain stem.

If it be argued that fourth nerve paralysis is difficult to determine and may have been overlooked, it can be stated with certainty that fourth nerve paralysis never occurred as an isolated ocular palsy in this series of cases. Every case in which diplopia occurred was carefully investigated, and the diplopia of superior oblique palsy is so distressing as to render impossible its being overlooked in a patient who is fully conscious and who is not blind.

Paralysis of the sixth nerve was met with in twelve cases of supratentorial tumour. It was unilateral in six cases, and bilateral in six cases. The paralysis when unilateral was right-sided in four cases and in two cases left-sided. It is therefore, more common as an indirect result of intracranial tumour than paralysis of any other cranial nerve. This fact has been recorded by the majority of observers, and the explanation has been given that the sixth pair of cranial nerves are more liable to be affected as a result of general increase of intracranial pressure on account of their long intracranial course and exposed position. I am unable to accept this explanation since the intradural position of the nerve alone is exposed to the intracranial pressure, the portion between this and the sphenoidal fissure being protected by the dura mater and by the blood of the cavernous sinus.

In many cases of supratentorial tumours the tentorium is pressed downwards, and the brain stem and cerebellum are also pressed downwards. The medulla and posterior part of the cerebellum come to lie partly within the foramen magnum, the cerebellum being deeply indented by the edge of the foramen magnum so that these structures together form a conical plug which fills up the foramen magnum. This alteration in the position of the brain stem may be also

demonstrated *post mortem* by the deep indentation of the crura cerebri by the free edge of the tentorium.

All the cranial nerves attached to the brain stem, with the exception of the sixth pair, are directed transversely or obliquely from their superficial origins to their exits from the subdural space. The sixth pair of nerves, however, are directly straight forwards. The shifting backwards of the brain stem can have little traction effects upon the roots of those nerves, which are directed transversely, but considerable traction effect must occur from such shifting in the case of the sixth nerves.

I wish to submit it is from this cause that sixth nerve paralysis comes to be the most frequent cranial nerve palsy, resulting indirectly from the presence of intracranial tumour.

The fifth nerve was affected in two cases, and in both of these tract-hemianopia was also present. The tumours were large and were situated in the left prefrontal region and left frontal centrum respectively. In both cases the motor paralysis appeared before the sensory paralysis.

The fifth nerve paralysis and the hemianopia probably resulted from the indirect pressure of the growth upon the middle fossa of the same side.

The sensory loss appeared in the area of distribution of the three divisions of the trigeminus in the order 3, 2, 1.

The Seventh Nerve.—Peripheral facial palsy occurred in two cases. It was preceded by paralysis of the sixth nerves. In one case a large tumour occupied the posterior horn of the left lateral ventricle; the facial paralysis was upon the left side and was not associated with paralysis of the left eighth nerve. In the second case a large tumour was situated in the left frontal region, and complete paralysis of the left seventh and eighth nerves occurred.

It may be here remarked that slight weakness of the lower face is of very common occurrence in supratentorial tumours of any situation. It is not associated with any alteration of electrical excitability. The writer has no explanation for its occurrence to put forward.

The Eighth Nerve.—Bone deafness was present in two cases. It occurred once in connection with paralysis of the

seventh nerve in the above-mentioned case of frontal tumour and once without facial paralysis in a case of temporo-sphenoidal growth.

The writer's experience directly negatives the theory that auditory neuritis in any way comparable with optic neuritis occurs in intracranial tumours. Excluding these two cases, and those cases where the auditory path was directly involved by growth, he has found that defects of hearing are not more common in long standing cases of intracranial tumour than in patients suffering with diseases other than of the nervous system.

The ninth, tenth, eleventh, and twelfth nerves were not indirectly affected in any of the cases.

Speaking collectively therefore, palsy of the cranial nerves, as an indirect result of intracranial tumour, is not of very common occurrence. It was present in sixteen cases out of 161 cases, or 10 per cent. The possible fallacy of statistics was strikingly shown in this series. Of the cases occurring between 1894 and 1899 it occurred in fourteen cases out of ninety-two, equal to 15 per cent., while of the cases between 1900-1904, it occurred in two cases out of sixty-nine, under 3 per cent. One possible reason for its rarity of late years is that cases of intracranial tumour are operated upon earlier and more frequently than was formerly the case. In all the cases the cranial nerve paralysis has been a relatively late symptom.

The determination as to what period is "early" or "late" depends upon the severity of the symptoms and the rapidity of growth of the tumour. A local sign appearing three months after the onset of symptoms would be a late sign in a case where the general signs were of great and persistently increasing severity; and on the other hand, it would be an early sign in a case where the general signs were not obtrusive and increased slowly. Paralysis of the external rectus was observed as early as the sixth week in a case with severe general signs. This patient died on the tenth week, and a tumour the size of a large orange was found, the pressure signs being very well marked.

The cranial nerve palsies once appearing are generally permanent, but this is not always so. Paralysis of the sixth nerve has disappeared in two cases, and in one case paralysis of the fifth nerve, at one time complete, recovered to a great extent. In such cases it is possible either that there is some alteration in the mechanical interference with the nerves in the course of time, or that they become accustomed as it were to the changed conditions.

THE OCCURRENCE OF JACKSONIAN EPILEPSY, HEMI-EPILEPSY AND GENERAL CONVULSION. IN CONNECTION WITH TUMOURS OF THE BRAIN STEM AND CEREBELLUM.

In two cases of tumours of the cerebellum local convulsion of slow spread, and confined to the arm and face, were repeatedly observed. Both were cases of long standing, and in both slight signs of bilateral spasticity were present. In the early stages of the illness the signs of cerebellar involvement were conclusive. The autopsies showed that considerable ventricular distension was present, but there was no other lesion present to account for the local convulsion.

Hemi-epilepsy was observed in one case of glioma of the pons and in one case of cerebellar tumour, while general convulsion occurred in several cases of tumour of the brain stem and of the cerebellum.

In all these cases the symptoms were of long standing when the convulsions first occurred, and in all of them the autopsies revealed considerable ventricular distension.

These remarks have reference to local convulsion of the cortical type and these must not be confused with certain local convulsions of a peculiar nature and order, which undoubtedly occur in lesions of the pons and cerebellum, and which have their origin in these structures. Such "lowest level fits" have been long since described by Dr. Hughlings Jackson. Most interesting and important observations upon local convulsion of cerebellar origin are published in this number of *BRAIN* by Dr. Grainger Stewart and Dr. Gordon Holmes, who have given me the privilege of acquaintance with their observations while in progress.

That hydrocephalus alone is capable of causing local convulsion is proved by the not rare occurrence of the phenomenon in cases of primary hydrocephalus.

Further, if the weighty arguments of Dr. Hughlings Jackson be followed, that every epileptic seizure has of necessity a local commencement, the distinction between local and general convulsions as local signs of organic disease of the brain becomes invalid.

It is, I think, a very commonly accepted fallacy that local convulsion is a sure sign of local gross lesion of the cerebral cortex. The commonest cause of Jacksonian convulsion is probably idiopathic epilepsy. Of the patients who have attended the out-patient department at Queen Square during the past five years, and who have suffered with local convulsion, the majority have been cases of idiopathic epilepsy, provedly so by the history and course of the malady; while a minority have been cases of local gross disease of the cerebrum.

On the other hand, patients who have attended with symptoms apparently conclusive of idiopathic epilepsy and who have later on developed symptoms of intracranial growth have been numerous.

It is submitted, therefore, that the occurrence of local convulsion of hemi-epilepsy and of general convulsion, when presenting for the first time, long after the general signs of intracranial growth have appeared, is to be disregarded as a localising sign. It is the result of secondary hydrocephalus, and the latter may result no matter where the growth may be situated within the skull. Slight bilateral spasticity is not infrequently an indication of the existence of such ventricular distension.

Bilateral spastic paresis occurred late in several cases where there was not involvement of the pyramidal tracts neither directly nor by pressure. It amounted to clumsiness of movement in the limbs, slight rigidity, increase of the deep reflexes, foot clonus, and the extensor response in the plantar reflex. In all the cases there was considerable distension of the lateral ventricles. Bilateral spastic paresis thus occurring in the late stages of intracranial tumour is

to be ascribed to the cortical wasting which results from secondary distension of the ventricles and perhaps also to degeneration of the cortical elements, which the chronic partial evascularisation consequent upon slowly increasing intracranial pressure must entail.

The occurrence of bilateral spastic paresis in cases of intracranial tumour usually indicates that the growth is situated in some region where the pyramidal tracts are close together; in other words, that the growth implicates the brain stem. It is especially in connection with infiltrating gliomata of the brain stem that this sign is met with.

The series of cases here dealt with show that bilateral spastic paresis may occur as a late sign in cases of intracranial tumour wherever the growth is situated, provided that distension of the ventricles occurs. Now secondary hydrocephalus may occur with a long standing growth in any situation.

It has been already pointed out that palsy of certain cranial nerves is of frequent occurrence as an indirect result of long standing intracranial growth.

The combination of bilateral spastic paresis with cranial nerve palsies, usually a sure sign of implication of the brain stem may therefore be produced in the late stages of a growth in any situation within the skull. The determination as to whether this combination is a true or a false localising sign is sometimes difficult. The reasons for the difficulty, and the points which may serve in the discrimination, will be apparent from the following table:—

	Glioma of Brain Stem.	Late Pressure Effect.
Bilateral spastic paresis	Occurs early or late ...	Occurs late.
Degree	Slight or severe, often unilateral for a time	Bilateral.
Cranial nerve palsy ...	Almost always of nuclear type at first	Always of peripheral type
	Any nerves may be involved	Never the 4th, 9th, 10th, 11th and 12th.
	Optic neuritis often absent	Optic neuritis always severe.

The question as to whether signs significant of cerebellar lesion result from the distortion of the posterior parts of the lateral lobe of the cerebellum, in the formation of the pressure cone, in cases of long standing intracranial tumour, is one of great difficulty.

The writer's opinion is that such signs are of not infrequent occurrence. Such signs must of necessity be bilateral since both lateral lobes are distorted. Further, the signs of cerebellar disease are for the most dynamic signs, and for their elicitation it is necessary that the patient shall be in possession of his mental and physical powers. For an instance, it is impossible to detect the presence of bilateral ataxy with certainty if bilateral paresis be present, or if the patient is mentally unable to attend and obey commands. Similarly the gait and tendency to fall in a particular direction cannot be tested in a patient who cannot stand and walk, or who is blind. In the late stages, therefore, of long standing intracranial tumour the tests for cerebellar involvement are often inapplicable.

In many cases, however, of long standing intracranial growth, nystagmus is met with, unilateral or bilateral ataxy may occur, the patient's gait and attitude may suggest cerebellar disease and he may constantly fall in a particular direction and in such cases no other abnormality of the cerebellum has been found.

The writer has had the opportunity of examining a very large number of cases of cerebellar tumour and is of opinion that tumours of the cerebellum rarely fail to show the signs of cerebellar involvement, as described by Risien Russell, early in the course of the case. The same holds good for all cases of subtentorial growth that they give early localising signs, with the distinct exception of some cases of glioma confined to the pons.

If this opinion be correct, the appearance of signs indicative of cerebellar involvement, late in the course of a case where no signs of subtentorial growth had previously existed, should not be allowed much weight in local diagnosis.

The absence of the usually accepted localising signs for many weeks or months after the general symptoms have

become severe is in itself of very important localising significance.

Since the supratentorial division of the intracranial cavity is roughly seven times as large as the infratentorial division, other things being equal, a non-localisable tumour has a probable supratentorial situation in seven cases out of eight. In this series the proportion of supratentorial tumours to infratentorial tumours has been rather more than three to one.

While "silent areas" form a large proportion of the cerebral hemispheres, and the possibilities of compensation are considerable in this region, below the tentorium the tracts connecting the brain with the periphery are densely grouped together and further, it appears that lesions of the cerebellum are almost always early productive of characteristic symptoms. During the last five years, of a very large number of cases of cerebellar tumour in the wards of the National Hospital, there was no case verified by operation or by necropsy that had not been correctly diagnosed during life.

It appears, therefore, that a tumour which, while giving rise to marked general symptoms, affords no localising symptoms for many weeks, may be located alone the tentorium.

THE PRODUCTION OF SYMPTOMS FROM OTHER LESIONS CO-EXISTING WITH A NEOPLASM.

Meningitis.

Sir William Gowers draws attention to the frequency with which some degree of meningitis co-exists with intracranial tumour, more frequently in the immediate neighbourhood of a growth which implicates the meninges, but sometimes also existing in isolated patches at a distance from the growth. With the exception of one case of tuberculous tumour of the cerebellum, in which tuberculous meningitis supervened, inflammation of the meninges was not found in this series of cases.

Vascular Lesions.

The occurrence of vascular lesions in regions remote from the situation of the tumour may give rise to prominent localising signs which are likely to lead to a false diagnosis, as in the following case: A man, aged 39 years, was admitted into the National Hospital on July 16, 1900, under the care of Dr. Hughlings Jackson. He had suffered with headache and vomiting for two months, and he presented well marked optic neuritis. Two days before admission he had a fit, commencing upon the right side, in which he lost consciousness. On recovering consciousness he presented aphasia and weakness of the right arm and face. After admission the paralysis of the right arm and face became progressively worse. An exploratory operation in the left fronto-central region revealed an extensive softening. Subsequently the necropsy showed that a large tumour was situated on the right optic thalamus, and that the anterior branches of the left Sylvian artery were extensively thrombosed.

While it seems impossible to avoid diagnostic error in cases of this nature, yet it is advisable to bear in mind the possibility of vascular lesions in cases of intracranial tumour above middle age, and who have hard arteries.

In one case of frontal tumour a hæmorrhage the size of a walnut was found *post mortem* on the right lateral lobe of the cerebellum. It was a very recent hæmorrhage and had probably occurred just before death, for no corresponding symptoms were observed. A similar hæmorrhage into the cerebellum was found in a case of rapidly fatal hydrocephalus in an adult; again the hæmorrhage was very recent and no symptoms were referable to its occurrence.

It has been stated by many authorities that the presence of a tumour upon one of the larger cerebral arteries may cause extensive thrombosis in the region of its distribution. In this series tumours have often existed in close proximity with the larger arteries but thrombosis has not been met with. It may be pointed out that the pressure which a tumour can assert is governed by, and cannot exceed, the maximum of the cerebral arterial pressure.

Local Spreading Œdema.

The occurrence of cerebral œdema in the immediate neighbourhood of cerebral neoplasms is well known. It is referred to here as the probable cause of acute symptoms in two cases among this series; the patients, while in apparent good health, were seized with urgent and rapidly fatal symptoms.

A girl, aged 23 years, came as an out-patient to the National Hospital in May, 1897. She had been perfectly healthy till May, 1895, when she had three sudden attacks of loss of consciousness at intervals of a few days and in the third of these attacks she was slightly convulsed. For the following year she suffered with occasional attacks of giddiness associated with pallor, and it was for such attacks that she sought advice. She presented no sign of organic disease. She was treated with bromides and her attacks of giddiness became less frequent. She came regularly to the out-patient department for the next eighteen months, during which time her health was good and she continued her work. On November 12, 1898, she was apparently in excellent health. The following morning she was found to be very somnolent. The somnolence deepened and she was admitted as an in-patient into the National Hospital, under the care of Dr. Bastian, on November 14. When admitted she was evidently very ill physically and she lay in bed in a half conscious state and never moved a muscle unless roused. Early optic neuritis was the only other abnormal physical sign that could be detected. Two days later paralysis of both sixth nerves made its appearance. The optic neuritis became severe, the coma deepened, and she died on the fourteenth day of her illness. The autopsy was performed by Dr. Risien Russell. A small, hard, thickly encapsuled tumour, evidently of long standing, was found, involving the second and third left frontal convolutions and the posterior part of the orbital lobule. The measurements of the growth were $5 \times 3 \times 3$ cms. For a considerable distance round the growth the white matter of the centrum semi-ovale was much swollen. It was much

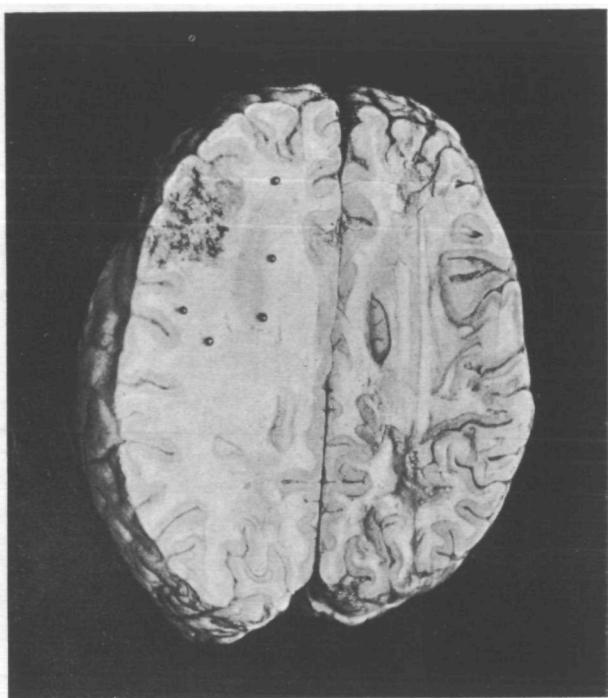


FIG. 1.

Acute oedema in the region surrounding a hard, slowly-growing tumour.

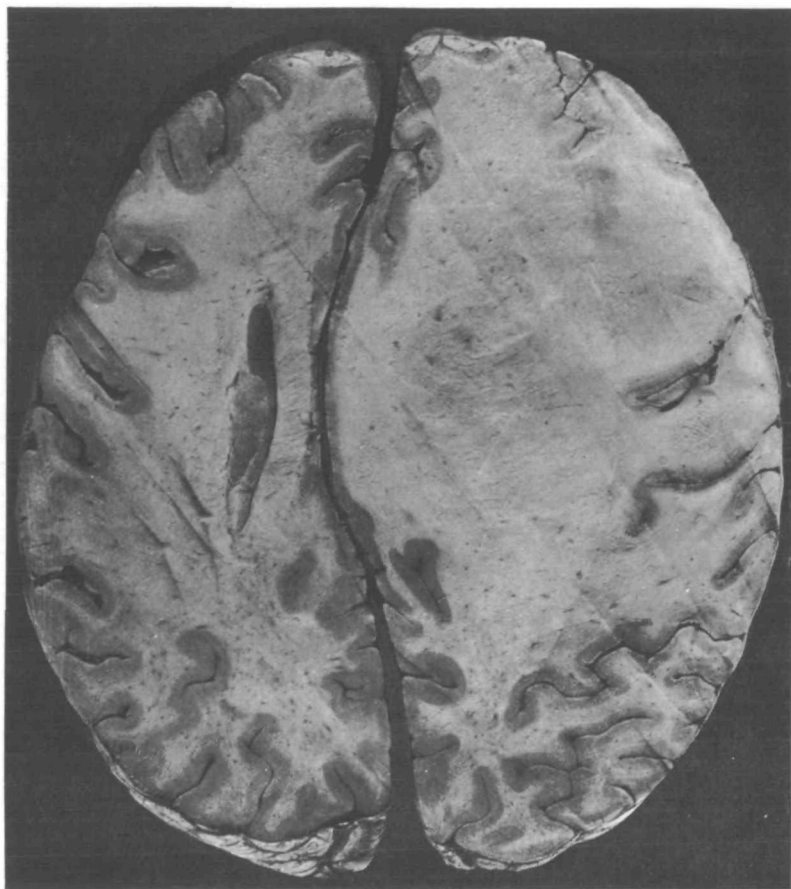


FIG. 2.

Edema of the centrum semiovale, from a small nodule in the right prefrontal cortex, secondary to a renal carcinoma.

softer than normal and was almost translucent. Examined microscopically this swollen white matter presented marked vacuolation and lymphatic distension as the only peculiarities (fig. 1).

In this case one cannot but conclude that the epileptic symptoms were referable to the presence of the growth, and that the urgent and rapidly fatal symptoms were the result of the spreading oedema.

The second case in which symptoms were referable to spreading oedema, was one in which, as a result of primary renal carcinoma, a secondary tumour about the size of a walnut occurred in the cortex of the right prefrontal lobe. The cerebral symptoms were very urgent and death occurred in fourteen days after their appearance. There were no localising signs during the first seven days. Afterwards, some degree of hemiplegia appeared.

The right hemisphere was found to be greatly increased in size and its white matter enormously swollen even as far back as the occipital region and softer than normal and somewhat jelly-like in appearance. The tumour was sharply defined (fig. 2).

It is submitted that in this case the rapidly fatal cerebral symptoms were directly referable to the condition of oedema of the right hemisphere, and not to the presence of the growth.

SYMPTOMS WHICH MAY RESULT FROM THE OCCURRENCE OF DEPOSITS SECONDARY TO A PRIMARY CEREBRAL GROWTH.

Secondary growths are very rarely met with in cerebral neoplasms. They occurred in two cases among this series. In both cases the primary tumour was situated in the neighbourhood of the fourth ventricle and in both the growth had burst through the ependyma or the pia and was in contact with the cerebro-spinal fluid. In one case, a secondary growth was found at the junction of the anterior and posterior roots of each of the spinal nerves, from the third cervical region to the lowest sacral region, a naked eye

appearance resulting of great enlargement of each posterior root ganglion. The filum terminale was the seat of many secondary growths, and its appearance resembled that of a string of beads. The symptoms referable to those secondary growth were severe shooting pains in all the limbs. Later, the knee-, wrist- and elbow-jerks were lost, and complete motor and sensory paralysis of the fifth and sixth cervical nerves on the right side occurred (fig. 3).

In the second case, a solitary secondary deposit was found upon the left first dorsal root. Right hemiplegia was present, and the patient lay in a half-unconscious state many weeks prior to death and no symptoms referable to the growth were detected.

In these two cases it was quite obvious that the secondary deposits had been planted by the cerebro-spinal fluid, and they afford important confirmatory evidence to the opinion that the main path of exit of the cerebro-spinal fluid from the intradural space is along the spinal nerve roots.

The nature of the neoplasms was that of fibro-sarcoma.

SYMPTOMS WHICH MAY POSSIBLY RESULT FROM POSTERIOR DEGENERATION.

The frequency with which degeneration of the posterior columns of the spinal cord occurs in association with intracranial tumours, no matter where these may be situated, has been pointed out by Dr. Batten and the writer, in this journal, vol. xxii., p. 473.

In some cases this degeneration is very marked and in three such cases among this series, shooting pains in the limbs, loss of the deep reflexes in the lower extremities, and ataxy of the legs, occurred, and were presumably referable to the posterior degeneration.

THE CONCEALMENT OF LOCALISING SIGNS.

Many cases of intracranial tumour come under observation for the first time long after the onset of symptoms, and, in some of these cases, localising signs, which judging from

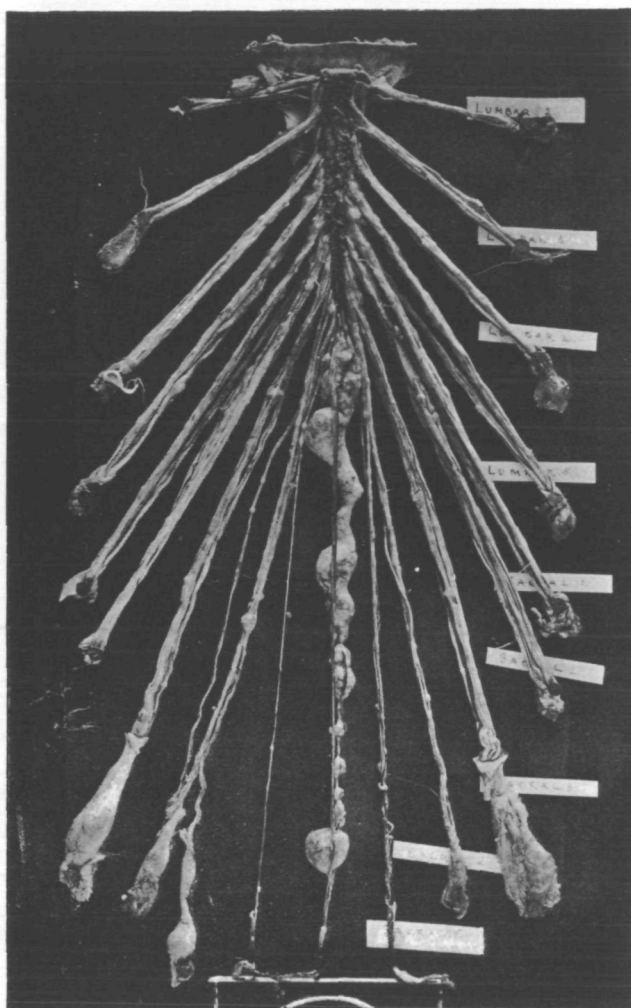


FIG. 3.

Secondary deposits in the filum terminale and root sheaths from a case of fibro-sarcoma of the cerebellum. Dr. F. E. Batten.

the situation of the lesion, must have been present in the early days, are no longer recognisable.

HEMIANOPIA.

Two cases among this series came to the National Hospital presenting no localising signs and in which optic neuritis had produced complete blindness. Careful questioning, both of the patients and their relatives, revealed no indication that hemianopia had existed while sight remained.

In both cases, the tumour directly involved and apparently originated in the region of the cuneus and hemianopia must have been present early. But this, the only localising sign of a lesion in the posterior pole of the hemisphere, had been obliterated by the optic neuritis.

CEREBELLAR SIGNS.

While diagnosis should not be difficult in the early stages of cerebellar tumour, it may be very difficult in cases seen for the first time when the disease is of long standing. The signs of cerebellar disease may be called dynamic signs. It is necessary for their proper elicitation, that the patient be in the possession of his mental faculties, and of his power of voluntary movement. He must be able to sit up, to move his limbs, to stand, and to walk, and to attend intelligently to command.

In case of long standing, as a general result of intracranial pressure or of ventricular distension, mental deterioration, physical weakness, blindness from optic neuritis, and bilateral spastic paresis may conceal the cerebellar signs more or less completely, while the occurrence of hemiplegia, or of double hemiplegia from extension of the growth into the brain stem, or from pressure upon the brain stem, effectually conceals the cerebellar signs in the region of the paralysis.

THE DISAPPEARANCE OF LOCAL SIGNS.

The local signs which result from pressure and altered anatomical relations are sometimes temporary. It has been

already pointed out that indirect cranial nerve palsies may disappear in the course of time. Hemiparesis from pressure upon the pyramidal path may be similarly transient. As a tumour grows, the structural relations and directions of pressure are constantly changing and a region at one time pressed upon may subsequently be relieved. Possibly a structure may become used to the pressure as it were, and may regain its functional capacity. In two cases of glioma of the pons, however, the disappearance of a pathognomonic local sign was remarkable and difficult to explain.

Both cases, when admitted into Hospital, presented optic neuritis, bilateral spastic paresis of the limbs, much more marked in the legs and loss of lateral conjugate deviation. The last sign was indicative of interference with the sixth nucleus. Both patients lived many months and secondary hydrocephalus became manifest. Notwithstanding that the other signs became progressively more marked, the paralysis of lateral conjugate deviation became progressively less marked until some three months after admission it was no longer demonstrable. No other signs indicative of involvement of the cranial nerve nuclei appeared.