muscels is firm...He can appreciate pinprick sensation at about the center of each malleolus laterally and medially and over the dorsum of the ankle. The only sensation that I could detect in the foot is vague appreciation of firm pinch of the pad of the big and second toes. The pinch would cause discomfort above the ankle. The skin of the foot is dry and shiny but entirely intact. He has no trouble with ulcers on the foot and reports that he has been playing sports, including softball and basketball."

SUMMARY

A patient with an extensive injury to the right sciatic nerve was treated successfully by a two-stage procedure in which the peroneal division was utilized as an autogenous nerve graft.

The surgical technic described in detail had been suggested in a previous communication.

REFERENCE


BRAIN TUMOUR WITH NORMAL AIR ENCEPHALOGRAPHY AND ARTERIOGRAPHY

REPORT OF AN UNUSUAL CASE

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That a cerebral tumour may produce gross hemiparesis, hemi-hypaesthesia, and dysphasia and yet produce no alteration of the ventricular system or of the carotid arteriogram seems rather surprising. As an unusual example of a problem which must be faced by all who endeavour to treat neurological disease, the following case is reported.

CASE REPORT

National Hosp. No. 19432. E. P., a married woman of 62 years, was admitted on Oct. 21, 1949 under the care of Dr. E. A. Carmichael. She had been well up to 1 year before admission when she seemed to be easily tired and suffered from a series of mild upper respiratory infections from which she never recovered to normal health. Five months before admission, slight weakness and clumsiness of the right arm were noticed, followed by weakness of the right leg 2 weeks later. This gradually progressed and for the last 14 days she had been completely paralysed in the right arm and barely able to walk on the right leg. Her walking was slow and she dragged the leg after her and tended to swing it outwards. The left arm and left leg were unaffected.

For the last 6 weeks she had had a disorder of speech. She described her disability as difficulty in finding the word that she wished to use. She maintained that she was well aware of what she wanted to say, that she understood perfectly what was said to her and what she read. She felt that if it were not for her paralysis she would be able to write. Although this speech disorder fluctuated considerably it gradually progressed. On admission her speech was understandable although the impairment was obvious.

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TUMOUR WITH NORMAL AIR STUDIES AND ARTERIOGRAM

Two months before the onset of weakness, she had suffered from headaches approximately once a week. These were generalised dull aching pains accompanied by injection of her eyes. They were not a prominent feature of her illness and disappeared after a few months. She had lost about 28 lbs. during the last year; she said that even though she often felt hungry, at the sight of food, she felt that she could not eat.

There was no family history of nervous or mental disease.

Examination. She was co-operative and there did not appear to be any gross intellectual deterioration. B.P. was 190/90. P. 72, T. 98.4°F. There was a dysphasia which was almost entirely expressive. On rare occasions she was able to produce an entire sentence without error, but for the most part she was hesitant, frequently using words which obviously did not express exactly what she wanted to say. No jargon words were heard. Her ability to understand the spoken word, to understand written commands, and to read out passages was good. She made a few errors in attempting to name objects, but ability to write could not be tested owing to her hemiplegia. No left-right disorientation could be demonstrated but there appeared to be a finger agnosia. There was no papilloedema or other abnormal sign in the cranial nerves.

The gait was hemiplegic; the right shoulder lower than the left, the right arm held immobile in a position of adduction and flexion, the right foot inverted with a slight foot drop. The leg was dragged stiffly behind her and circumducted, with the toes scraping the ground. She was unable to maintain a posture of the right upper or lower limbs. There was considerable reduction of power in the right arm and leg, worse in the arm than the leg and most marked in the flexors and extensors of the fingers. Owing to this weakness, co-ordinated movements were not possible on the right side. Slight spasticity was present in the flexors and pronators of the right arm but the right leg was not spastic. Deep reflexes were normal in the upper extremities and in the left lower extremity. The knee jerk on the right was hyperactive but the ankle jerk on that side could not be elicited. Abdominal reflexes were absent. The left plantar response was flexor; the right was absent. There was slight impairment of all forms of cutaneous sensibility and vibration on the right side of the body, with marked reduction in two-point discrimination and stereognosis. There was gross loss of sense of position in the right hand and right foot. There was a fairly severe degree of varicosity of the veins of both legs.

Laboratory Data (Dr. J. M. Cuming). Hb. 104 per cent; W.B.C. 6,800/c.mm.; 72 per cent polymorphonuclears; 26 per cent lymphocytes; 2 per cent eosinophils; platelets 250,000/c.mm. The lumbar CSF was clear and colourless with an initial pressure of 130 mm.; cell count normal, protein 40 mg. per cent. Pandy and Nonne Appelt tests negative, Lange normal.

EEG Report of October 11 (Dr. W. A. Cobb) indicated "a widespread lesion in the left hemisphere resulting in abolition of frontal fast activity and release of alpha activity, as well as the more direct effects causing a slow wave response. It is, I think, consistent with a cerebral neoplasm though rather atypical."

Roentgenographic Findings (Dr. D. Sutton). Films of skull and chest normal. Fluoroscopic examination of upper gastro-intestinal tract following ingestion of barium revealed no abnormality. On Oct. 25, 1949 left percutaneous cerebral arteriography was performed. "There was fair filling of the internal carotid artery, including the posterior cerebral artery. The anterior cerebral artery lay in the midline and the vessels generally appeared normal in position and distribution. Conclusions: no lesion detected." On Nov. 11, 1949 a lumbar air encephalogram was done. There was "good filling of the ventricular system. The septum pellucidum, 3rd and 4th ventriciles lay in the midline. The lateral ventriciles were on the large side of normal, but were symmetrical and showed no deformity" (Figs. 1 and 2).

Course. There was no change in her condition, and on November 15 she was transferred to a convalescent home. Two weeks later she suddenly lost consciousness and was re-admitted to the National Hospital. She was in status epilepticus with attacks involving the right face, arm and leg occurring at intervals of 5-6 min. and lasting 30 sec. There appeared to be no recovery of consciousness between the attacks. Intravenous pentothal sodium was administered and the attacks ceased. She recovered consciousness 30 min. later and was found to have gross right-sided hemiplegia and to be almost completely aphasic. Although there were no further seizures, the paralysis and dysphasia failed to improve. On the morning of Dec. 4,
1949 she could not be aroused; her lips were cyanotic and there was froth at the corners of her mouth. The right pupil was slightly larger than the left but both reacted briskly to light. Corneal reflexes were brisk but no other response could be obtained to painful stimuli. R. 25; B.P. 170/96. Both arms and legs were completely flaccid. No plantar response and no abdominal reflexes could be obtained; all deep reflexes were absent. There was no neck stiffness and no Kernig sign. Blood sugar, blood urea and urine analysis were normal. Lumbar puncture showed clear and colourless fluid under a pressure of 160 mm. The respiratory rate gradually increased and the temperature rose, and without regaining consciousness the patient died on Dec. 6, 1949, 2 days after the onset of this acute episode.

**Necropsy.** Skull, air sinuses, dura, venous sinuses, basal vessels and leptomeninges were healthy. The brain showed neither gross swelling nor atrophy. The gyri of the anterior oper-}

![Figs. 1 and 2. Anteroposterior and lateral views of the air encephalogram.](image)

culum of the insula on the right side were slightly broadened and firm in consistence. There was very slight temporal herniation, more marked on the right side.

On section there was a swelling and pallor of the anterior corpus striatum, lentiform nucleus and thalamus on the left side, with a cortical and subcortical replacement by firm pale tissue of most of the anterior part of the floor of the insula on the left side. The left cerebral pedunule was broader than the right but there was no obvious abnormality in the pons or medulla. The ventricles were not dilated. The body of the left lateral ventricle was very slightly narrowed and its lateral angle was 2 mm. further from the midline than the right (Fig. 3C).

**Microscopically** the tumour was found to be more extensive than was expected from the macroscopical appearances. The most active part of the tumour lay in the anterior part of the left insula. Here it replaced both grey and white matter and was composed of closely packed glial cells with rounded, ovoid, reniform or elongated nuclei, often in mitosis, and but little cytoplasm. Glial fibres were rare. Swollen-bodied astrocytes were occasionally included in the tumour. Capillaries showed endothelial hyperplasia and occasional foci of necrosis were bordered by radially arranged tumour cells. The appearances here were those of a glioblastoma multiforme (Kernohan's type IV astrocytoma).

From this region the tumour spread widely but without the nerve cell and myelin destruction seen in the most active part. It extended deeply to involve the left caudate and lenti-
Fig. 3. Coronal sections of the brain viewed from the front. (A) Anterior to the optic chiasm; (B) at the optic chiasm; (C) through the mammillary bodies; (D) through the red nuclei; (E) through the splenium of the corpus callosum; (F) through the mid-brain (left) and at the junction of the mid-brain and pons. The darkened areas show the extent of the tumour and “M” marks the site of its maximum activity.

form nuclei, the amygdaloid nucleus (and adjacent cortex), the left thalamus and posterior hypothalamus, the internal capsule, the lateral geniculate body. It had crossed the midline to involve the right posterior hypothalamus and the inferior part of the thalamus. Downward spread from the basal structures involved both red nuclei and subthalamic nuclei, the tegmentum of the upper midbrain and the superior corpora quadrigemina.

It extended superficially and widely in the cortex and immediate subcortex. All the floor and the frontal and parietal opercula of the insula were involved. Thence it extended supe-
riorly in a broad band, in the posterior frontal and anterior parietal regions, to the superior margin of the hemisphere and down the medial aspect to the inferior limit of the gyrus cinguli. From this major band of cortical involvement minor extensions passed backwards towards the middle part of the convexity of the occipital lobe in its anterior part. Another extension passed forwards to involve those portions of the supraorbital and medial frontal cortex that lay superficial to the anterior corpus striatum. Above the corpus callosum at the coronal level of the corpora mammillaria there was involvement of the indusium griseum.

In these deep basal regions and in the widespread cortical involvement the neoplastic change was different from that seen in the floor of the insula. In the deep basal regions it varied in activity from place to place, being more active in the thalamus than in the corpus striatum. In some regions nerve cells did appear to have been destroyed, in others not. Not only were there fairly numerous elongated abnormal glial cells but also there was a great increase in swollen-bodied and glial fibre-forming astrocytes. Within the internal capsule there were elongated tumour cells and swollen-bodied astrocytes in small numbers. The infiltration was less marked in the midbrain.

In the involved cortex, overlying the apparently intact deep white matter of the frontoparietal region, there was infiltration of all layers by abnormal glial nuclei with very little cytoplasm, elongated in shape and orientated radially and by fibre-forming astrocytes. Although nerve cell loss was only slight, several neurons showed satellitosis by glial or tumour cell nuclei.

COMMENT

It is well recognised that although the history of a patient’s disease may suggest that he has an intracranial tumour, air studies may show no abnormality. This has been stated by Poppen and Peacher in their report of 10 such cases, and also by Merritt and Brenner who recorded the findings in 3 similar patients. The study by Pennybacker and Meadows has the additional advantage of postmortem studies on the 4 patients whom they followed. Three of these had normal air studies 5½ months, 5 months and 5 days respectively before death; but at autopsy there was a shift of the septum pellucidum, though it was slight, in the third case. The ventriculogram of the fourth patient, performed 3 months before sudden death during a convulsion, was normal and although no mention of the ventricles is made in the necropsy report one assumes that there was no displacement since the tumour was a small one near the surface of the parietal lobe and not associated with oedema or haemorrhage.

None of the patients mentioned in the above three papers was investigated by arteriography. As a single ancillary investigation this method also has its limitations in helping to confirm the clinical diagnosis of cerebral tumour. Attention has recently been drawn to its fallibility by Culbreth, Walker and Curry who stated that: “In supratentorial tumours carotid angiography may fail to show tumours of the basal ganglia and midline structures, the inferior temporal region and the occipital region. For the demonstration of such tumours ventriculography is preferable to angiography.”

Air studies or arteriography alone may be normal in the early stages of cerebral gliomata owing mainly to the size and site of the tumour. Repetition of the test at a later date will give positive evidence of abnormality in many cases. In some cases, however, no such abnormal findings are revealed even in the late stages. We consider our case to be unique in having both normal findings up to 6 weeks before death, and at necropsy an undisplaced ventricular system in the presence of a glioma that had produced gross hemiparesis, hemi-hypaesthesia and dysphasia. Although in this patient there was involvement of the central grey matter, pronounced in-
volvement of the cortex distinguished it from the cases of thalamic tumour described by Nevin. The pure intracortical type of spread of this tumour is one of the modes of growth of a glioma named by Scherer a secondary structure. The extensive infiltration of the cortical grey matter produced widespread dysfunction with little distortion of the gross appearance of the cerebrum, and this is the reason why such profound disability could be present with normal air studies and arteriogram. This case emphasizes the fact that a glioma may kill its host without producing pathological changes demonstrable by the ancillary methods of investigation in use today. The most constant diagnostic feature in the natural history of such tumours remains their relentless progressive course.

SUMMARY

There is reported a case of brain tumour which produced hemiparesis, hemi-hypaesthesia and dysphasia in which carotid arteriography and air encephalography disclosed no abnormality and which at necropsy showed no displacement of the ventricular system.

The authors are indebted to Dr. E. Arnold Carmichael for permission to report the clinical aspects of this case.

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TORULA GRANULOMA OF THE CERVICAL SPINAL CORD*

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In their recent paper on the infection of the central nervous system by the Cryptococcus hominis, or Torula histolytica, Carton and Mount presented an exhaustive review of the literature. They found only 10 instances in which either the spinal cord or its membranes showed involvement by Torula. In most of these cases the infection was widespread, also affecting the brain or its meninges. In only 3 instances was the infection confined to the spinal cord, the cauda equina or their

* Read before the Neurological Section of the Academia de Ciencias Médicas de Cataluna, April 17, 1950.