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## CLINICAL NOTE

# CRITERIA FOR THE EARLY DIAGNOSIS OF EXPANDING SPACE-OCCUPYING INTRACRANIAL LESIONS*

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WHEN A PATIENT SUFFERS FROM intractable and prolonged headache most practitioners are anxious lest they overlook a case of intracranial space-occupying lesion (I.S.O.L.). This anxiety arises from the indefinite clinical picture which such cases may initially present.

The purpose of this article is to review the development of four of these cases with features in common, which shed light on this problem and point the way to an observational research project which may yield information of practical use.

## Case histories

**Case 1.** W.W. was a 22-year old mother who had a history of neurotic breakdown under stress. She first complained of occipital headaches in August 1958, after her baby had been admitted to hospital with pneumonia. The initial diagnosis was reactive depression, and she was given both analgesics and psychotherapy without relief. After three weeks her nervous system was examined but no abnormality was detected. It was felt that this excluded organic disease of the nervous system, but her symptoms might have arisen from sinusitis. She was referred to the ear, nose and throat department where she was found to have a

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deviated nasal septum, but no toher abnormality. Following this investigation her headache went and she felt very well for a week.

She then had a relapse and complained of severe headache. No sign referrable to the nervous system was found, but it was felt that the emotional reaction to her complaint was exaggerated. She was given analgesics and reassurance, but in October she requested an emergency visit as she said the pain was increasing. There was still no sign referrable to the nervous system so the dose of analgesic was increased. The following day she complained of vomiting. Since no physical sign was obtained she was admitted to a medical ward as a case of severe neurosis which had not responded to treatment.

In the ward she was found to have temporal headache, a history of vomiting on three occasions, weak legs, paraesthesiae, and diplopia with impaired position sense of the right great toe. She also had increase in the right patellar deep tendon reflex, ataxia, past pointing to the right, and Rombergism. Papilloedema, at first equivocal, became definite overnight. She also developed right external rectus palsy.

She was transferred to a neurosurgical unit where the presence of a left parieto-occipital space-occupying lesion was demonstrated radiologically. The localizing signs had been produced by pressure on the right cerebellum. A glioblastoma multiforme was removed and she was given radiotherapy, but she died a year later from multiple metastases.

**Case 2.** D.W. was a 24-year-old mother of one child when she first complained of headache in 1950. There was no physical sign, but she was referred to the ear, nose and throat department as a possible case of sinusitis. This diagnosis was not substantiated.

For two years after this she complained of intermittent headache. At the end of this period she was referred to the medical outpatient department and admitted to a medical ward for observation. The initial findings in the ward were amblyopia, convergent squint, enlarged blind spots and possible papilloedema, but these were not confirmed at a later examination. She was discharged with the diagnosis, " no organic lesion ".

In 1953 she was admitted again after a dental operation. The anaesthetist had noted loss of the right corneal reflex, right trigeminal anaesthesia and doubtful suffusion of the discs.

She was referred to a neurosurgical department and suffered an attack of headache during the examination. The observing clinician noted tenderness over the left parietal region, decreased left trigeminal sensation, most marked in the mandibular division, and absence of the left corneal reflex. The protein level of the C.S.F. was 100 mg. per cent. She was admitted to the ward one month later and these findings were confirmed. The diagnosis was altered to " virus infection of the root of the left trigeminal nerve or demyelinating disease with psychological overlay ".

After this she remained well until 1955 when she sustained a head injury in a motor accident. This was not serious but she was admitted to a surgical ward where ptosis and transient diplopia were observed.

She came into my care in 1959. Her complaint was headache and she had left trigeminal anaesthesia. She was again sent to the medical outpatient department as a possible case of I.S.O.L. The trigeminal anaesthesia was confirmed and weakness of the right leg with increased tone in the left leg were also noted. Angiography and ventriculography showed enlarged ventricles. The protein level of the C.S.F. was 60 mg. per cent. Despite these findings the diagnosis was altered to ' hysteria '.

After 18 months of intermittent headache which was unrelieved by analgesic

she was examined again. At this examination she had nystagmus, unequal pupils, left trigeminal anaesthesia, right facial weakness, weakness of the right leg and ataxia. She was referred once again to the outpatient department and thence admitted to the ward. On this occasion she had ataxia, aphasia and impaired left trigeminal sensation. She had also Rombergism and fell to the left when walking. All deep tendon reflexes were increased and sensation in all modalities was diminished on the left side. The protein level in the C.S.F. had risen to 220 mg. per cent. While she was in the ward her signs settled and the comment on discharge was " Not sufficient evidence for expanding I.S.O.L. Diagnosis remains obscure, to be kept under observation." One month later at follow up, no sign was demonstrated, but the C.S.F. protein level remained raised at 250 mg. per cent.

After six months of almost continuous headache and intermittent staggering she was re-examined. Two new signs had developed, dysphagia and mandibular clonus. In view of the chronicity of her illness she was sent back to outpatient department for consideration as a case of demyelinating disease. At her appointment only nystagmus remained. She was admitted for further investigation and she was found to have ataxia, right nystagmus with difficulty in looking up and in convergence. The C.S.F. protein level was 100 mg. per cent. and the pressure was 213 mm. water. She was discharged without a definite diagnosis.

Her condition deteriorated over the next three months. The headache and ataxia increased and she developed left supratrochlear palsy, scanning speech loss of mandibular power and left facial weakness. She was readmitted for consideration as a case of disseminated sclerosis. In the ward she was unable to focus her eyes. She had left trigeminal anaesthesia with loss of left corneal reflex, nystagmus, left superior oblique weakness. There was general increase of the deep tendon reflexes and a possible left extensor plantar response. After three days papilloedema developed and she was transferred to a neurosurgical ward. All these signs were confirmed and in addition she now had right abducens peresis, right facial weakness, neck stiffness, inco-ordination of the left upper lid, loss of power in the left side and reduced muscle tone in all limbs. Angiography was performed and she was shown to have a right cerebellar tumour. At operation this was found to be a choroid plexus tumour but unfortunately the basilar artery ruptured. The bleeding site was packed, but some days later she died following a secondary haemorrhage.

**Case 3.** W.H. was 58 when he first took ill. His wife came to the surgery and asked for treatment for his headache. After questioning she said that he was drowsy, lethargic and had become bad tempered. He had formerly been even tempered. He was visited the following day and examined. He admitted to severe frontal headache with increasing lethargy and loss of memory. There was no sign of disease of the nervous system, but his disc margins were slightly blurred. He was referred to the medical outpatient department as a case of brain tumour.

The following day, before an appointment could be arranged, he was seen as an emergency because the headache had become intolerable. There was only one physical sign; a right extensor plantar response. This history suggested an expanding I.S.O.L. in a silent area which was now interfering with the function of the left pyramidal outflow. The logical site for this type of lesion seemed to be the left temporal lobe with involvement of the left basis pedunculi. He was admitted to the ward with this diagnosis.

On admission he was found to have nominal dysphasia and receptive aphasia. After an observation period of one week there was almost complete remission, although there was slight residual aphasia. On the tenth day he had a relapse

with complete aphasia and a right extensor plantar response. He was transferred to a neurosurgical department.

On arrival, in addition to the signs above, he was confused and had papilloedema, right facial weakness, increased tone and weakness in the right arm and leg, and increase of the deep tendon reflexes on the right side. The right plantar response remained extensor. A left carotid angiogram showed a left temporal I.S.O.L. and a glioblastoma multiforme was removed. Following radiotherapy he was transferred to a hospital for chronic diseases where he remained until he died.

**Case 4.** J.M. was 69 when he first complained of headache in early December 1962. He was given an analgesic and asked to report back. The headache became worse and the treatment gave no relief. Two weeks later, at follow up, he staggered coming through the surgery door and behaved as though he were drunk. His speech was slurred. There was no smell of alcohol in his breath and he denied drinking. On further questioning he admitted that he experienced increasing drowsiness. Although he was warned that he might have a serious illness he remained euphoric. His nervous system was examined in detail. He had ataxia, nystagmus, cogwheel rigidity of the right arm and loss of vibration sense in both lower limbs. All deep reflexes were exaggerated. He was referred to the outpatient department. Because of the scattered signs the referral note suggested that he may have had demyelinating disease, but that the headache biased the provisional diagnosis to I.S.O.L.

The examining physician in the outpatient department elicited a history of head injury, but could not confirm the signs. The opinion given was that while the patient may have had cerebro-arterial disease neither tumour nor subdural haematoma had been excluded. It was suggested that the wisest course was to continue observation and admit the patient to the ward whenever the practitioner felt this to be necessary.

The headache increased in severity and the drowsiness became worse. Finally he needed an emergency visit and was seen by the partner on call for the practice. He was admitted to the receiving ward as a case of brain tumour. On admission his speech was slurred, he had ptosis, increased deep tendon reflexes and a bilateral extensor plantar response.

He was seen by a neurosurgeon who diagnosed subdural haematoma, since there was a history of trauma, and the intracranial space was explored. This diagnosis was confirmed and the haematoma was removed. The patient made a good recovery although this was complicated by an attack of cardiac failure.

## Discussion

It is generally recognized that craniotomy should be performed as rapidly as possible if a patient is known to have an operable expanding I.S.O.L. (Cube, 1958). This means that a general practitioner must be in a position to diagnose these cases in an early stage of development. This should be possible, since diagnosis in most cases is considered to rely on an accurate evaluation of the clinical picture (Roberts, 1951).

The accepted criteria for the diagnosis are headache, vomiting and papilloedema with localizing signs in the central nervous system.

It has been recognized for some time that the first three are not

constant (Worster-Drought, 1948), and that localizing signs are not always reliable (Hartson, 1951; Arias *et al.*, 1962). In cases of doubt ancillary aids, lumbar puncture, electroencephalography, ventriculography, and angiography often help.

Large neoplasms above the posterior fossa may also be localized by radioactive iodinated human serum albumin (Dunbar and Ray, 1954).

The salient points of the four cases reported in detail above are summarized in the table. It will be seen that apart from headache none of the standard criteria for the diagnosis of I.S.O.L. was demonstrable in the early stages of the disease. On the other hand, personality disorders and remission were features common to all four. Furthermore, in the second and fourth cases there were distinct but scattered signs in the nervous system, but these signs were insufficient to localize the lesion beyond doubt. This means that it may be necessary to adopt new criteria to diagnose I.S.O.L. in its early stages. To do so would require a new conception for the progress of these conditions. At present we assume that the progress of an expanding I.S.O.L. follows one of the curves shown in figure 1, and can first be diagnosed when the intensity of the clinical picture reaches a certain level.

TABLE

SUMMARY OF SALIENT FEATURES OF THE FOUR CASES DESCRIBED IN THE TEXT

| *Case 1* | *Case 2* | *Case 3* | *Case 4* |
|---|---|---|---|
| Headache | Headache | Headache | Headache |
| Neurosis+ | Neurosis+ | Personality change | Personality change |
| Early absence of signs | Scattered varying signs | Early absence of signs | Scattered varying signs |
| Remission | Remission | Remission | Remission |

In these four cases the progress followed a curve similar to figure 2. This would explain why identical signs were not obtained by the different clinicians examining the cases and also why it was difficult to establish the diagnosis until the disease was in an advanced stage.

From these findings it is clear that there is a gap in our knowledge of the early stages of I.S.O.L. Organized and detailed observations should be made on such cases in general practice and the results of these observations should be collected so that more detailed information about the early signs of I.S.O.L. can be studied and evaluted.

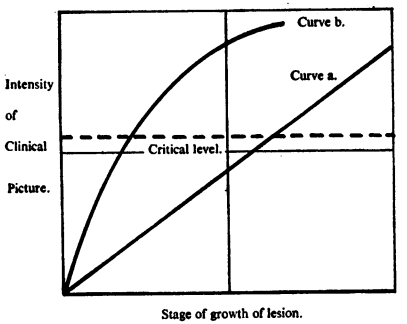


Figure 1
**Graphical representation of hypothetical course of expanding intracranial space-occupying lesion.**

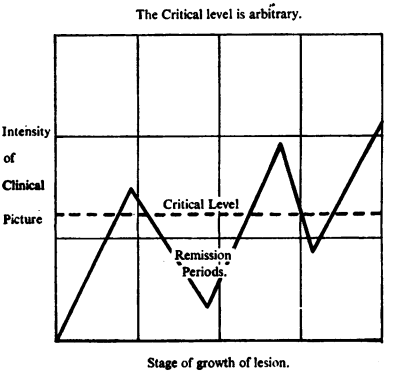


Figure 2
**Graphical representation of observed course of four cases described in text.**

Curve a. Course assuming that growth of the lesion and intensity of the clinical picture bear a simple arithmetical relation to each other.

Curve b. Course assuming that the intensity of the clinical picture bears a logarithmic relation to the growth of the lesion.

Critical level: The level at which the intensity of the clinical picture produces sufficient symptoms and signs to allow a diagnosis to be established. Its position in this illustration is arbitrary.

## Summary

The clinical course of four cases of intracranial space-occupying lesion is described in detail.

The symptoms and signs common to the cases are headache, personality disorder, vague or absent signs referrable to the nervous system in the early stages and partial or complete remission.

These features differ from the accepted criteria for the diagnosis of intracranial space-occupying lesions.

It is suggested that this is a field which requires further observational research.

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