

explanation of their low birth weight, who were heavily handicapped in all tests.

I wish to thank the chairman and members of the joint committee for their help and advice; the medical officers of health and the health visitors whose generous co-operation made the survey possible; and the mothers in all parts of the country who willingly answered numerous and detailed questions on their children's health.

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EPILEPSY OF LATE ONSET IN THE LIGHT OF MODERN DIAGNOSTIC PROCEDURES*

BY

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The number of diagnostic techniques available for the investigation of epilepsy of late onset raises the question how many are essential in the investigation of the individual patient. The accurate location of the causal lesion is obviously a different task from its detection, and is not dealt with here. This paper attempts only to assess which techniques are most likely to exclude the presence of a lesion and what are the clinical indications for their use.

The material consisted of 200 cases of epilepsy starting after the age of 20, excluding traumatic cases and those with papilloedema. Cerebrospinal fluid findings were not considered. The first group of 100 was investigated at the London Hospital Neurological Unit at the time of its evacuation in the second world war to Chase Farm E.M.S. Hospital, where facilities were limited. Pneumoencephalography by the lumbar route with partial replacement was the routine method of investigation. The second group consisted of 100 patients recently admitted to Maida Vale Hospital, where all the usual techniques were available. Little difference was apparent in the diagnoses when the two groups were compared (Table I). The term "idiopathic" was used, as formerly, to indicate that no significant macroscopic lesion was detected on radiological investigations, and was not restricted to central or genetic epilepsy.

The incidence of space-occupying lesions was about equal in the two groups, with a figure of 23 if angiomas are included.

Cerebral atrophy presenting as epilepsy provided just under a fifth of the cases. This condition was a definite clinical entity characterized by minor neurological signs, including some degree of mental deterioration in most patients. The radiological appearances of atrophy were

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present on pneumoencephalography. Occasionally the condition masks a tumour, but this combination was not observed in this series.

Epilepsy due to arteriopathy or arteriosclerosis, though not uncommonly seen in out-patient clinics, did not usually present a sufficiently difficult diagnostic problem for full neuroradiological investigation to be necessary. Epilepsy was attributed to this cause in 4% and 3% of cases respectively. Cerebral infarction from other forms of cardiovascular disease producing an epileptic focus was not represented in this series.

Clinical Features

Certain clinical features in both groups—that is, the age of onset of the attacks, the length of history, and the presence of abnormal physical signs—were associated with an increased likelihood that structural abnormalities would be found on investigation.

Age of Onset.—The cases are divided in Table I into those under 40 and those 40 and over, and show the age distribution of onset of attacks and diagnosis. Abnormalities were

TABLE I.—Age of Onset and Diagnosis

	Under 40		40 and Over		Total	
	Group 1	Group 2	Group 1	Group 2	Group 1	Group 2
Idiopathic epilepsy ..	36	43	16	15	52*	58†
Cerebral tumour ..	7	9	13	9	20	18
Angioma ..	3	4	1	1	3	5
Cerebral atrophy ..	11	6	10	9	21	15
Cerebral arteriopathy ..			4	3	4	3
Aneurysm ..				1	0	1
Lesion present	37%	33%	63%	61%	48%	42%

* Including 1 temporal lobe epilepsy.

† Including 15 temporal lobe epilepsy.

detected in 37% and 33% of patients under the age of 40, and 63% and 61% in the later age groups. Tumours and cerebral atrophy show a relatively increased incidence in those aged 40 and over. The angiomas differed, and in seven out of eight patients produced fits in the third and fourth decades.

Duration of Symptoms.—If the attacks had occurred for more than five years the probability of a tumour being present was much reduced. Of the 38 patients with tumours in the two groups, 35 had a history of attacks for five years or less, and in only three did the history exceed five years. Three-quarters of the cases with cerebral atrophy had a history of five years or less, but five out of the eight cases of angioma had a history exceeding five years (Table II).

TABLE II.—Duration of Symptoms

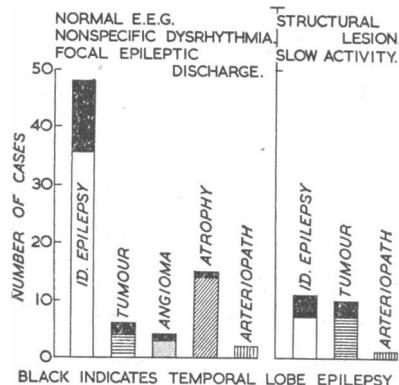
	5 Years or Less		Over 5 Years	
	Group 1	Group 2	Group 1	Group 2
Idiopathic epilepsy ..	40	46	12	12
Cerebral tumour ..	20	15		3
Angioma ..	3	3	3	2
Cerebral atrophy ..	18	9	3	6
arteriopathy ..	4	3		
Aneurysm ..		1		

Physical Signs.—A causal lesion was detected in four out of every five patients with abnormal physical signs, and in one out of five without abnormalities on clinical examination.

Focal Attacks.—The diagnostic significance of focal attacks was modified by the recent interest in temporal lobe epilepsy. The number of patients investigated for focal attacks increased from 30% in group 1 to 58% in group 2, but the incidence of macroscopic causal lesions fell from the high figure of 63% in group 1 to 45% in group 2, because radiological investigations were more commonly negative in temporal lobe epilepsy than in focal attacks originating in other sites.

Electroencephalograms

In group 2 E.E.G. investigations were made, and the accompanying Chart shows the preponderance of temporal lobe attacks in epilepsy without a macroscopic lesion and also the major E.E.G. findings. A non-specific generalized dysrhythmia, a focal epileptic abnormality, or rarely a normal E.E.G. was found in 83% of cases of idiopathic epilepsy and in all cases of cerebral atrophy.



Electroencephalographic findings.

Evidence of a structural lesion with slow activity was present in 63% of tumours, a figure approximating those obtained by Cobb (1950) and Millar (1955) for supratentorial tumours. Localizing features were present in the E.E.G. in two cases of proved tumour, with negative findings on contrast radiography.

The E.E.G. was lateralizing in one case of angioma showing a spike discharge, but the number of cases was inadequate for any conclusions to be drawn. Rosenberg (1952), in a series of 55 angiomas, found lateralizing or localizing E.E.G. abnormalities in 67%.

In a minority of patients with temporal lobe epilepsy sphenoidal electrodes were used for the purpose of accurate location of a temporal lobe focus with a view to lobectomy, or if the presence of a tumour was suspected.

Radiological Findings

Straight x-ray films of the skull showed significant abnormalities in half the cases with a space-occupying lesion in both groups.

Air Studies (Group 1)

Pneumoencephalography by the lumbar route in group 1 demonstrated the ventricles in 98 cases, and the results are recorded in Table III. Ventriculography carried out subsequently on two failures did not reveal any abnormality.

TABLE III.—Air Studies in Group 1

	No. of Cases	Pneumoencephalography 98 + 2 Failures	
		Normal	Abnormal
Idiopathic epilepsy ..	52	50	2
Cerebral tumour ..	20	III	17
Angioma ..	3	0	3
Cerebral atrophy ..	21	0	21
arteriopathy ..	4	2	2
Aneurysm ..	0	0	0

Roman numerals indicate misleading results

In idiopathic epilepsy ventricular abnormalities appeared to be related to hyperostosis frontalis interna in one case, and in another case a congenital abnormality was present.

Normal ventricles were present initially in 3 of the 20 cases of tumour, a similar proportion to group 2 (see below). One patient was explored on clinical grounds owing to the presence of calcification in the straight films, and the tumour was removed. In the two other patients the clinical diagnosis of tumour became evident after some months. In one of these a repeat pneumoencephalogram two months before death was again negative, although localizing signs had appeared on examination.

In the three cases of angioma, a deformity was produced in a lateral ventricle, readily detected on pneumoencephalography.

Cerebral atrophy was diagnosed by the usual criteria of pooling, widening of sulci, and dilatation of the ventricles.

Arteriopathy was associated with only a minor degree of generalized atrophy or dilatation of a ventricle following a vascular lesion. Perhaps the rigidity of the cerebral vascular tree prevents a greater degree of shrinkage.

Angiography and Air Studies (Group 2)

For group 2 percutaneous angiography was available, and was carried out in 68 cases, and pneumography in 88 (Table IV). Where the clinical examination or straight

TABLE IV.—Findings in Group 2

	No. of Cases	Angiography 68			Pneumography 88		
		No.	Normal	Ab-normal	No.	Normal	Ab-normal
Idiopathic epilepsy ..	58	40	38	II	51	49	II
Cerebral tumour ..	18	15	III	12	17	II later + 0	15
Angioma ..	5	5	0	5	1	0	1
Cerebral atrophy ..	15	4	2	1+I	15	0	14+I
Cerebral arteriopathy ..	3	3	2	1	3	1	2
Aneurysm ..	1	1	0	1	1	0	1

Roman numerals indicate misleading results.

x-ray findings suggested a lateralized tumour, angiography followed by ventriculography was the usual procedure.

Abnormal angiograms in idiopathic epilepsy consisted of a minor degree of shift of the anterior cerebral arteries, unassociated with a ventricular shift on pneumoencephalography. Failure to fill the ventricles adequately by the lumbar route occurred on two occasions, requiring ventriculography, which showed no abnormality.

The clinical diagnosis of tumour was in doubt in only 3 out of 18 cases, and in all three angiography failed to detect the lesion. Pneumoencephalography was positive in one, suggestive in another recorded as normal, and negative in the third. Both these latter findings became positive when air studies were repeated after an interval of two to three months.

Angiomas, as MacKenzie (1953) showed in a series of 50 cases, when presenting with epilepsy were usually large malformations, producing a bruit or prominent abnormal physical signs in every patient.

In this group angiomas were demonstrated by angiography, but with one exception the malformation had already been diagnosed on clinical grounds. This was the case of a man aged 49 with a history of uncinat attacks for 25 years. The E.E.G. revealed a right anterior temporal spike focus, but angiography showed a small angioma in the right posterior temporal region. It seemed doubtful whether this lesion was necessarily the cause of his uncinat attacks. The patient had a history of meningitis* before the onset of his attacks, which might have produced some scarring of the anterior region of the right temporal lobe.

Cerebral atrophy was again demonstrated by air studies, with one failure by the lumbar route. Angiography was misleading in one patient with bilateral atrophy owing to a shift of the anterior cerebral arteries away from the hemisphere producing abnormal signs.

In cerebral arteriopathy, angiography located calcification in the cerebral vascular tree, but otherwise was of no particular value. Minor atrophic changes were again observed on pneumoencephalography.

The single case of aneurysm presented as a space-occupying lesion with deformity of the frontal horn; its nature was identified by subsequent angiography.

Conclusions

Clinical features indicating that full neuroradiological investigations are advisable in epilepsy of late onset include

* I am grateful to Sir Geoffrey Jefferson for his suggestion in the discussion that a subarachnoid haemorrhage was the probable cause of this "meningitis."

onset over the age of 40, a history of under 5 years' duration, focal attacks (though in long-standing temporal lobe epilepsy the findings are more likely to be negative), abnormal physical signs, and a bruit.

Electroencephalography is of positive value, particularly if localizing delta activity is present.

Pneumoencephalography is the most useful initial neuro-radiological investigation for the detection of early cases of tumour, and cerebral atrophy is also revealed by its use.

Angiography is the best method of visualizing the angiomas, but these malformations are an uncommon cause of epilepsy of late onset and can usually be diagnosed on clinical grounds; it is, however, of no special value in atrophy and may be misleading. In arteriopathy, although no disaster occurred in this series, angiography carries a risk of hemiplegia if angiospasm occurs.

The results suggest that electroencephalography, combined with pneumoencephalography by the lumbar route, is the most suitable combination of investigations in epilepsy of late onset unless prominent lateralizing signs or bruit are present, when angiography is preferable. Repetition may be necessary in two to three months or at a longer interval. The patient's co-operation is more likely to be obtained if he has not been subjected to angiography in addition to pneumoencephalography on the first occasion.

Summary

A series of 200 cases of epilepsy starting over the age of 20, excluding traumatic cases and those with papilloedema, have been surveyed in an attempt to assess which investigations are indicated to detect the causal lesion.

The C.S.F. findings have not been considered.

A group of 100 cases, after clinical evaluation and straight x-ray examination, were investigated by pneumoencephalography.

The investigations employed in the second group of 100 cases were E.E.G. (100%), pneumography (88%), and angiography (68%).

The diagnoses showed a close similarity in the two groups.

Contrast radiography might be negative in the presence of a tumour which produced a focal abnormality in the E.E.G.

Pneumoencephalography was slightly more reliable than angiography in detecting tumours at an early stage, and demonstrated cerebral atrophy. Angiomatous malformations were an uncommon cause of epilepsy, and the presence of their characteristic clinical features indicated the need for angiography.

It is concluded that after clinical and straight x-ray examinations electroencephalography with pneumoencephalography is a suitable combination of techniques for the investigation of epilepsy of late onset, unless frank lateralizing signs or a bruit are present, when angiography is preferable.

I wish to express my thanks to Sir Russell Brain and Mr. D. W. C. Northfield for permission to use their clinical records at Chase Farm Hospital E.M.S. Neurological Unit, and to Dr. M. H. Jupe, for his reports on the radiographs. I am indebted to my colleagues at Maida Vale Hospital for allowing me access to their case records and investigations.

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VASCULAR DISEASES OF THE NERVOUS SYSTEM

A SERIES OF 315 CASES

BY

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Vascular diseases of the nervous system are the fourth most common cause of death in this country. While intracranial aneurysms and cerebral angiomas have received much attention lately because of new methods of diagnosis and treatment, the majority of deaths from cerebral vascular disease are due to cerebral thrombosis and haemorrhage. Wright and McDevitt (1954) estimated that in the United States there were 10 cases living in any one year for each death due to a cerebral vascular accident. The application of this factor here gives a figure of nearly 700,000 people suffering from some sort of stroke or its after-effects. As Wright and McDevitt point out, many of these people are still in the productive period of life and may be the main support of their family. Their frequent need for prolonged attention cannot always be met by a suitable relative, and the care of the paralysed is nowadays falling increasingly on hospitals and other institutions.

Between 1943 and 1953 the number of deaths a year due to vascular diseases of the nervous system rose from 49,000 to 68,000—that is, from 10% to 13% of the total annual deaths for England and Wales (Registrar-General, 1954). The rise in deaths from this cause, both absolutely and in relation to other causes of death, is largely due to the increasing age of the population and to the decreasing numbers of deaths from pneumonia and other infections since the advent of the sulphonamides and antibiotics. However, it is not clear why, even after allowing for the different numbers and ages of each, the rise is slightly greater in women than in men.

In view of the size and increasing importance of the problem, and of present developments in treatment, as by cervical sympathetic injection (Leriche, 1952) or by operation, it was felt that a study of the recent natural history of this group of diseases would be of value. All cases of vascular disease of the central nervous system admitted to St. Mary Abbots Hospital in the years 1951-4 inclusive have been reviewed. Selection on the basis of admission to hospital has been inevitable and is discussed below. It was thought that the cases excluded would be mainly the very mild and the immediately fatal, so that those admitted would be the ones most likely to benefit from recent therapeutic advances. Although there are limitations to the method of retrospective analysis, information has been obtained on age and sex incidence, mortality, degree and duration of recovery, and certain clinical features. In addition, a brief description is given of some special aspects of the population and districts from which most of the cases came.

Material, and Diagnostic Criteria

In the four years covered, 315 cases fell into the group of vascular diseases of the nervous system. This is 6% of all in-patients in that period, but since these cases stay longer than the average they take up more than 6% of hospital beds and time, particularly nursing-time.

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