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EEG features associated with the occurrence of epilepsy after surgery for intracranial aneurysm and acoustic neuroma[1]

R. J. CABRAL[2] AND D. F. SCOTT

From the EEG Department, Section of Neurological Sciences, London Hospital, London

SUMMARY An investigation was carried out on 199 postoperative EEGs from 83 patients who underwent surgery for ruptured intracranial aneurysm or acoustic neuroma removal. The tracings were quantified without knowledge of the diagnostic group and whether or not epilepsy had supervened. The number of spikes and sharp components and slow waves at the site of the operative brain lesion were substantially and often significantly greater in the tracings from patients who developed epilepsy than from those who did not.

There is a dearth of information regarding the value of the EEG in the determination of the likelihood of postoperative epilepsy occurring in neurosurgical patients. However, two recent reports (Legg *et al.*, 1973; Prior *et al.*, 1973) did indicate that the postoperative EEGs could be of use. Because of these results it was decided to examine in more detail the tracings from two sets of patients who had undergone neurosurgical treatment. The first set were recordings of patients who had an acoustic neuroma removed by the combined transtentorial and translabyrinthine approach (Morrison and King, 1973) and the second set was taken from patients treated neurosurgically for ruptured intracranial aneurysm. Although the main aim was to establish the numbers of spikes, sharp, and slow waves associated with the occurrence of postoperative seizures, we also investigated, in some of these patients, the effects on the EEG of anticonvulsant medication.

Method

The present study was based on 199 postoperative EEGs taken from 83 patients who underwent surgery for aneurysm or who had an acoustic neuroma removed. The clinical aspects of the postoperative epilepsy in these groups have already been reported (Cabral *et al.*, 1976; Cabral *et al.*, in press).

[1] This study formed part of a PhD thesis of R.J.C. accepted by London University.

[2] Present address: Rua Capuraque, 52 (Floresta), Belo-Horizonte (MG) 30.000 Brasil.

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The distribution of the patients by diagnosis and the number of EEGs is shown in Table 1. The aneurysm cases were divided into three groups depending on the location of the lesion. They were assigned to the 'anterior group' when the aneurysm was on the anterior cerebral or anterior communicating artery, while those patients with aneurysms on the posterior cerebral or posterior communicating or internal carotid artery were designated the 'posterior group'. The remainder, where the lesion was on the middle cerebral artery, formed the 'middle group'.

Table 1 *Numbers of EEGs in the various groups*

	Epilepsy	*No epilepsy*
Aneurysm		
Anterior group	27 (7)	30 (11)
Middle group	41 (14)	18 (10)
Posterior group	15 (5)	27 (13)
Acoustic neuroma	15 (6)	26 (17)
Totals	98 (32)	101 (51)

Number of patients in parentheses.

In all there were 32 patients with 98 EEGs who developed epilepsy and 101 tracings from 51 patients who did not. Forty-four patients who provided 131 EEGs were followed up retrospectively over a period of 20 years. They were given anticonvulsants only if seizures occurred. The remaining 29 were studied prospectively from 1973 to 1975. Some were given routine anticonvulsants postoperatively (usually phenytoin 300 mg and phenobarbitone 60 mg a day). Others did not receive this treatment.

The EEGs were masked and coded. The assessments were carried out by R.J.C. after a training period in which over 90% of agreement in the quantification of the phenomena under study was obtained between the two authors using a similar series of EEGs. The ratings were made without reference to the diagnostic group as well as to whether the patients had or had not developed seizures. Further, for the individual patient, the chronological order of the tracing was not known to the rater. Many features were quantified but for the present report only slow activity and sharp waves and spikes over the area of maximum abnormality will be considered. These features were assessed, always on one channel, for any particular montage, using only those parts when the patient was resting and alert, not overbreathing or during photic stimulation. The numbers of sharp waves and spikes were summed and will be referred to subsequently as 'sharp components'. In order that comparisons could be made between one EEG and another the features were calculated on the basis of 100 seconds of recording. Occasionally, shorter segments were rated, but never less than 50 seconds and in these instances the findings were extrapolated to 100 seconds of recording. In the Results section all figures refer to the average amount of abnormality in 100 second epochs of EEG.

After rating, the EEGs were unmasked and an attempt was made to establish the criteria which separated those recordings made on patients who developed epilepsy from those who did not have fits. Differences were assessed using the chi-square one sample test.

Results

'Sharp components' were seen in almost all the EEGs. However the amounts of these depended on whether or not epilepsy supervened (Table 2). The recordings taken from patients treated for an aneurysm of the anterior group of arteries and who developed epilepsy showed at the time of onset of seizures, 80 sharp components (range 50 to 110). Most of the EEGs from the patients in whom fits did not occur showed fewer such components with a value of 40 (range 25 to 85). This was a statistically significant difference ($P < 0.01$), for in only one patient who did not develop seizures were there as many as 85 sharp forms.

Table 2 *Number of 'sharp components' at time of onset of seizures for aneurysms in various sites*

	Epilepsy	*No epilepsy*
Anterior group	80 (50–110 SD 13.9)	40 (25–85 SD 10.4)
Middle group	60 (50–80 SD 10.3)	30 (20–70 SD 15.5)
Posterior group	220 (150–320 SD 26.4)	120 (80–230 SD 40.1)

Range and standard deviation (SD) in parentheses.

The tracings of patients who developed seizures after surgery of middle cerebral aneurysm or acoustic neuroma were similar. They showed on average 60 sharp components (range 50 to 80). The EEGs from patients without fits exhibited substantially fewer sharp components with a figure of 30 (range 20 to 70).

Recordings from patients with posteriorly located aneurysms by the time fits had occurred showed on average 220 sharp components (range 150–320). Most of the EEGs from patients who did not develop epilepsy presented substantially fewer of these paroxysms with a value of 120 (range 80 to 230) ($P < 0.01$). However, in two patients this figure was reached.

When localised slow activity was studied, this was found to be significantly more persistent ($P < 0.05$) in those patients with seizures than those without. For example, it was observed for periods varying from six months for the middle group to 10–15 months for the anterior group.

Patients who were prescribed anticonvulsant medication from the time of the surgery exhibited fewer sharp components than those who developed epilepsy. Indeed, for the middle group no definite sharp forms appeared at any stage. When patients were given anticonvulsants after the onset of seizures these reduced the number of sharp paroxysms to below previously recorded levels. For example, in the acoustic neuroma series, two patients who had their seizures controlled with anticonvulsants showed a decrease of about a third in number of sharp components in the EEGs subsequently carried out.

Discussion

Information on the value of the EEG in predicting whether epilepsy would occur after neurosurgical procedures is scanty. However, Legg *et al.* (1973) and Prior *et al.* (1973) indicated that it could be of use in patients with cerebral abscess and ruptured intracranial aneurysm. The present EEG investigation studied patients with the latter condition and a group who had the combined translabyrinthine, and transtentorial approach to acoustic neuromata (Morrison and King, 1973). This operation, in contrast with the posterior fossa approach, carries a risk of the development of postoperative epilepsy (Cabral *et al.*, 1976).

Serial changes in the EEG were examined in detail (Cabral, 1975) but here we have reported only on the

numbers of spikes and sharp waves, and the amount of slow-wave activity at the time of the occurrence of epilepsy. For those patients without fits, the corresponding amounts of these EEG abnormalities described here were those appearing in their tracings taken at the equivalent time.

The significance of slow-wave components in post-traumatic epilepsy has been commented on by Jasper *et al.* (1940) and Cobb (1950). Their presence is suggestive of an active destructive process around a cortical lesion. In the EEGs of patients investigated in the present study there was a distinct association between the persistence of slow-wave activity and the occurrence of fits, the suggestion being that their appearance could be contingent upon the occurrence of a certain amount of cortical 'destruction'.

The appearance of spikes and sharp waves in themselves is not a predictor that epilepsy will occur. However, as in the study of Legg *et al.* (1973), larger amounts of these components were seen in patients with seizures. Ward (1975) has put forward the view that a 'critical mass' of firing neurones must be attained to allow the occurrence of clinical seizures. The present results are in keeping with his opinion in that critical amounts of spikes and sharp waves were noted in those patients who subsequently developed epilepsy. Also of interest is the fact that this critical number of sharp components was not the same for all areas of the brain. Thus, there was a marked difference between the middle cerebral and the posterior cerebral groups. The former had far fewer sharp components than the latter. In addition the time between operation and the occurrence of seizures was shortest for the middle cerebral and longest for the posterior group (Cabral *et al.*, in press). These findings are in keeping with the observations on the higher epileptogenicity of areas supplied by the middle cerebral artery.

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