

## Epilepsy and brain abscess

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Among 22 children who had recovered from brain abscess, 9 later developed epilepsy. Epilepsy developing as a consequence of brain abscess depends on the length of the catamnestic period and the localization of the abscess. The appearance of epilepsy is more frequent after frontal and temporal abscesses and in cases presenting symptoms in the acute phase of the abscess.

Since epilepsy may develop years or even decades after recovery from the brain abscess, it is recommended to keep the patient under control for years.

Epilepsy subsequent to brain abscess is common in children but literary data on this type of epilepsy are scarce and often contradictory.

Weber [4] believes that every second patient recovered from brain abscess may develop epilepsy. Douglas et al [2], further Matthes and Kruse [3] report on a similar frequency, whereas among the patients of Beller et al [1] suffering from supratentorial abscess only 15% developed epilepsy. The differences may be due to the length of the history; the longer it is, the more the number of epileptics [2].

In the period 1954 to 1978 we have observed 31 children and 7 infants with brain abscess. Of the 22 recovered patients, 9 developed epilepsy (41%).

In 5 children, the first seizure was observed within 1 year of recovery from the abscess, in 1 patient each in the 2nd, 4th, 11th and 16th year.

The question seems justified whether epilepsy after 11 or 16 years was actually a consequence of the brain abscess. Since in between neither of the two latter patients had any disease that could have provoked epilepsy and the attacks were characteristic, and the paroxysmal activity in the EEG corresponded to the site of the former abscess, it seemed justified to assume that despite the long interval the epilepsy was a consequence of the abscess.

What factors predispose to epilepsy subsequent to brain abscess? Literary data indicate that none of the following factors have a decisive role in the development of epilepsy: the aetiology of the abscess (otogenic, sinusitis, trauma, congenital heart defect accompanied by cyanosis, cryptogenic), the causative agent (staphylococcus, streptococcus, mixed bacterial flora, etc.), the manner of treatment (puncture, drainage or sur-

gical removal). The frequency of epilepsy is not significantly higher when the abscess has produced irreversible nervous system defects as e.g. hemiparesis. The only important factor appears to be the localization of the abscess. One may reckon more often with epilepsy after abscesses occurring in the frontal or the temporal lobe than after those in the parietal or occipital lobes, whereas the possibility of epilepsy can be practically excluded in cases with the abscess in the cerebellum.

Among our 9 epileptics, 2 had frontal, 4 temporal, and 1 each a fronto-parieto-occipital, centro-parietal or parieto-occipital abscess. Since the majority of frontal and temporal

abscesses is due to sinusitis, chronic otitis or trauma and only rarely to haematogenous dissemination (cryptogenic abscess), epilepsy more often follows these diseases. Besides, abscesses in the frontal or temporal lobes seem to provoke seizures in the acute stage more often than do abscesses located elsewhere in the brain.

Among our 38 patients, 10 had seizures prior to the diagnosis of the abscess. Of these children, 6 had a frontal, 2 a temporal, 1 a centro-parietal and 1 a cerebellar abscess. In the last case it was probably the increased intracranial pressure that provoked a generalized epileptic attack.

Thus, among our 9 patients developing epilepsy, in 5 children seizures

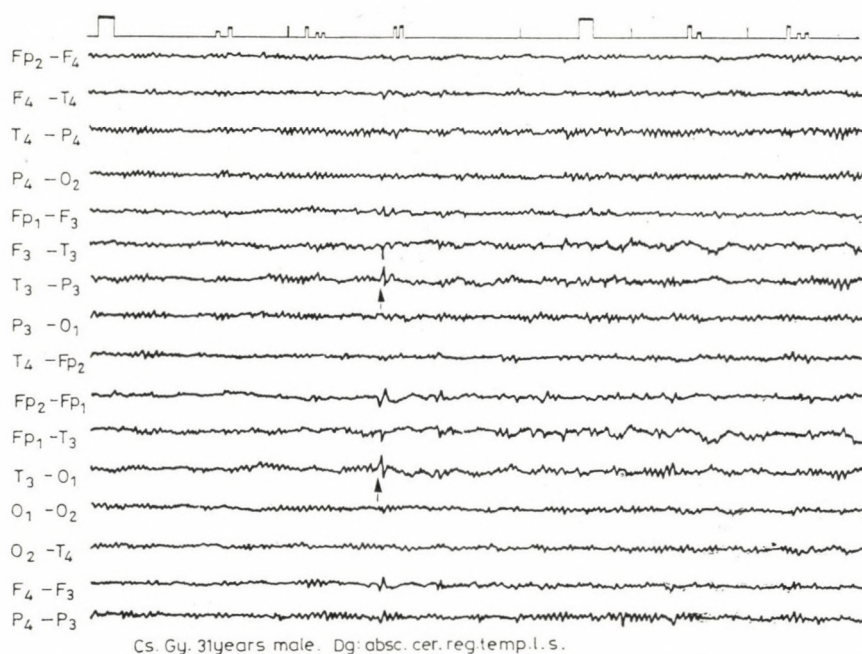


FIG. 1. C. C., a male epileptic 31 years of age. With 12 years he had had an otogenic left temporal brain abscess. On the EEG, left temporal central spikes (arrow) followed by irregular slow background activity



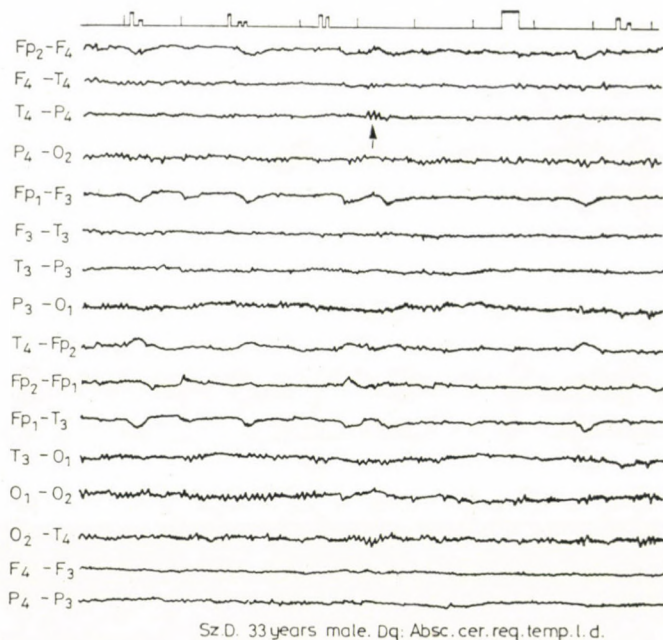


FIG. 2. S. D., a male patient 33 years of age. When 14 years old he had had an otogenic right temporal brain abscess. The EEG shows a rapid disturbance of irritative character in the right temporo-central area (arrow)

already appeared in the acute phase of the basic disease. Of the other 5 patients with seizures in the acute phase, 4 died. Among the patients who had had attacks in the acute phase and among those who had recovered from the brain abscess, only the patient with a cerebellar abscess did not develop epilepsy. Localization of the brain abscess in the 4 epileptics who did not have attacks in the acute phase was left temporal lobe (otogenic), right temporal lobe (traumatic), left fronto-parieto-occipital region (cryptogenic), left parieto-occipital region (cryptogenic).

Our observations support the finding that after frontal and temporal abscesses and especially in cases where

epileptic attacks occur early, in the acute phase of the abscess, epilepsy is more likely to develop following recovery from the abscess (Fig. 1).

The EEG records of the 7 patients who recovered from the brain abscess without any organic neurologic or behavioural aftereffect and now live a normal life, now show an adequate basic activity corresponding to the patient's age and an anomaly localized to the site of the former abscess. The anomaly is characterized by an irritative alteration composed of the periodic appearance of rapid spikes with a transient slower activity non-lesional in nature (Figs. 2 and 3).

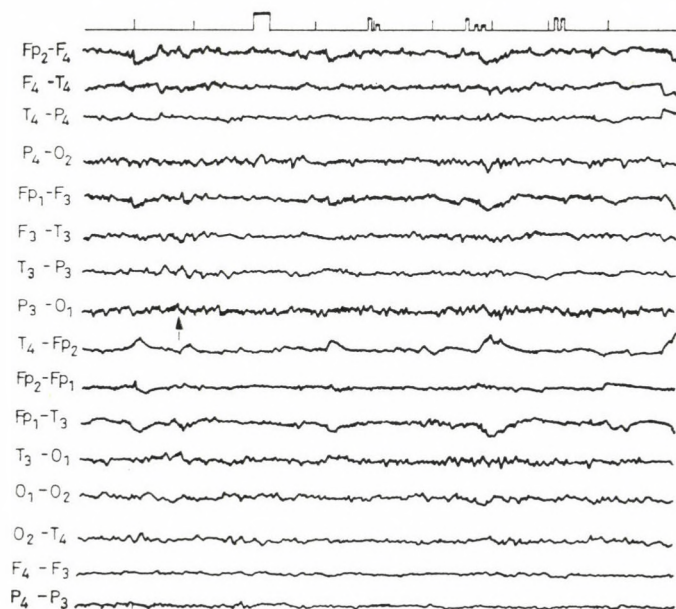
The EEG findings of a patient deserve special attention. The child had

a congenital heart defect with cyanosis. At the age of 2 years he suddenly developed a total hemiparesis on the right side. Left carotid arterial angiography showed neither vasospasm nor a tumour, so the hemiparesis was ascribed to a temporary vasospasm. Recovery was prolonged but almost complete. The child was again admitted at 6 years of age because of a right temporal brain abscess. This was cured by drainage. The subsequent EEG records revealed more signs pointing to the left vasospasm than to the healed right temporal abscess. This observation seems to support the view that a vasospasm and associated hypoxia damages mainly the neural cells

whereas in brain abscess intracellular oedema and oedema around the abscess are responsible for the irreversible EEG changes. Therefore, if no epilepsy develops after the brain abscess has healed, we do not have to reckon with cell lesions more serious than the above mentioned irritative anomaly.

Since epilepsy may develop many years after the brain abscess, patients who had had an abscess must closely be followed-up until the end of puberty. If they develop epilepsy, the following treatment is applied.

In the case of a supratentorial abscess, anticonvulsive treatment is indicated already in the acute phase. The same is valid if the abscess is lo-



P.A. 14 years female. Dq: Absc. cer. req. parieto-occip. l.s.

FIG. 3. P. A., female, 14 years of age. At 10 months of age she had a cryptogenic left parieto-occipital brain abscess. Now the EEG shows a left parietal slowing (arrow)



cated in the frontal or temporal lobe, or if signs pointing to epilepsy are observed.

After healing of the abscess, if the child is free from symptoms and complaints, no preventive anticonvulsant treatment is necessary.

Anticonvulsants are not prescribed when follow-up EEG records show paroxysmal signs but no behavioural disorders have been observed and no seizures have occurred. In such cases, however, the application of anticonvulsants should be considered individually, especially if in the acute phase of the abscess the child had had seizures, or if the abscess was of frontal or temporal localization.

In cases of epilepsy subsequent to brain abscess, anticonvulsive therapy must be instituted.

Although anticonvulsive therapy resulted in the improvement of our 9 patients, no lasting and permanent symptom- and complaint-free cure could be achieved in any of them.

#### REFERENCES

1. BELLER, A. J., SAHAR, A., PRAISE, I.: Brain abscess. *J. Neurol. Neurosurg. Psychiat.* **36**, 757 (1973).
2. MATTHES, A., KRUSE, R.: *Neuropädiatrie*. G. Thieme, Stuttgart 1973.
3. MCGREAL, D. A.: Brain abscess in children. *Canad. med. Ass. J.* **86**, 261 (1962).
4. WEBER, G.: *Der Hirnabszess*. G. Thieme, Stuttgart 1957.

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