PERSONAL EXPERIENCE IN THE SURGICAL TREATMENT OF EPILEPSY*

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One of the most impressive and dramatic symptoms of disease of the nervous system is an epileptic attack. The neurosurgeon sees a good number of patients afflicted with various types of epilepsy. Sometimes this symptom is only part of a clinical picture obviously produced by an intracranial lesion, like tumour, scar or atrophy. But in other instances the evidence of a cerebral lesion is not so clear, in spite of the great help afforded by radiographic and electroencephalographic auxiliary methods. Although all neurosurgeons are agreed upon the necessity of operation in cases of epilepsy secondary to definite cerebral lesions, there are some differences of opinion concerning those cases in which the criteria under consideration are mainly functional.

Our personal tendency in the surgical management of epilepsy is towards a careful selection of patients, and radical excision of the affected epileptogenic regions of the brain, whenever this is possible, while taking care to avoid causing great neurological disability. Few epileptic patients should be operated on, but with those selected for this type of treatment, the neurosurgeon, with a knowledge of the pathophysiology of the disturbance, must be radical in his efforts to remove the affected regions.

In reviewing our material of the last 5 years it was found that in a series of 755 major intracranial operations, there were 130 patients (17 per cent) with epilepsy. These cases may be divided into the following groups.

I. Expansive intracranial lesions (90 cases)
II. Focal cerebral lesions
   A. Localized cerebral scars (11 cases)
   B. Cerebral atrophies (19 cases)
III. Psychomotor and other functional disturbances (10 cases)

I. EXPANSIVE INTRACRANIAL LESIONS

Epileptic seizures were an important symptom in about a fourth of our patients with intracranial expansive lesions. The incidence was of course much higher among the patients with supratentorial lesions, nearly half of these having a history of attacks.

The distribution of the cases according to the pathological type of the lesion was as follows:

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In the group of supratentorial gliomas, epileptic manifestations were present in 35 per cent of the patients. As is well known, with some of the slower growing gliomas (oligodendrogiomas, astrocytomas) epileptiform symptoms may be present for many years. There is a wide variation in the type of the attacks, depending on the localization of the tumour.

With supratentorial meningiomas the incidence of epilepsy was similar (35 per cent). It was the predominant symptom in cases of meningioma of the convexity and parasagittal region near the Rolandic areas. Jacksonian motor attacks were the most frequently recorded. The duration of the epileptiform symptoms varied from 1 to 15 years. Another important practical point is that definite signs of increased intracranial pressure were absent in many of these patients.

Tuberculomas represent about 7 per cent of our total series of 370 intracranial tumours. Although more frequently localized in the posterior fossa, those of the cerebral hemispheres often give rise to Jacksonian focal attacks because of their situation near the Rolandic areas.

No particular comment need be made on the rest of these lesions. However, an interesting feature of our material is the number of parasitic cysts. Cysticercosis cysts represent 3 per cent of our series, but produced epileptic symptoms in only a few cases; signs of increased intracranial pressure were more frequent. Hydatid cysts represent 2 per cent, and were usually localized in the cerebral hemispheres (7 of 8 cases). They sometimes caused epileptiform manifestations.

In these cases of epilepsy caused by expanding intracranial lesions, the neurosurgeon makes an effort to extirpate the abnormal mass and to leave a cerebral scar as clean and as small as possible, removing all the damaged cortex. The results of such operations are usually evaluated on the basis of the late survival period and not by the presence or absence of postoperative attacks. Definite figures cannot be given on the results in our material, which is presented mainly to show the variety of pathological lesions. However, from different sources it appears that at least half or more of the survivors have postoperative epilepsy. As shown by Penfield and Erickson, the incidence of postoperative convulsions is higher with gliomas (astrocytomas) than with meningiomas.

II. FOCAL CEREBRAL LESIONS

A. Localized Cerebral Scars. In our series there are 11 cases of localized cerebral scars caused by trauma, bullets or circumscribed infections: 5
were in the frontal poles, 3 in the Rolandic region, 2 in the parietal lobe and 1 was in the occipital pole. Most of the patients were young; only 3 were over 30 years of age. In 6 cases the cerebral lesion was produced by a head injury and in 5 the causes were gunshot wounds. A history of infection of the primary wound with local abscess formation was obtained in 3 instances.

An interesting feature of post-traumatic epilepsy is the interval between the time of injury and occurrence of attacks. Many patients, in spite of a very severe post-traumatic scar in the brain, do not have epileptiform attacks until years after the injury. The electroencephalographic technique has been of help in some of these cases by showing abnormal discharges in the absence of clinical symptoms, and in such instances the cerebral scar may be removed at the time of the usual bone repair.

In our series of epileptics the intervals between the time of the injury and the appearance of the first attack varied from a few months to 11 years. In only 3 cases was the interval short (3, 9 and 15 months). In the rest it was long: in 5 cases between 5 and 6 years after injury, in 1 case after 8 years, and in 2 cases after 10 years. These figures confirm the view that the development of epilepsy after injury to the brain is slow and progressive. Sometimes early fits may occur within the first weeks after the injury, disappear completely for several years and then reappear to persist in the usual recurrent manner. This happened in one of our cases. These examples show the distinction between the immediate and late forms of post-traumatic epilepsy. They seem to have a different pathogenesis.

In the majority of our post-traumatic cases the attacks were referred to in the history as generalized seizures although sometimes a focal component or adersive beginning was noticed. With scars in the Rolandic areas there were typical Jacksonian fits which often progressed into a generalized discharge. Psychomotor automatic states were recorded in some cases of frontal scars.

An electroencephalographic study was performed in 8 cases and, with one exception, abnormal discharges were recorded in the injured area, such as the typical and sporadic spikes and sharp waves together with slow waves (4 cases) or only slow high voltage waves (3 cases). In one of the latter the slow waves of the occipital region spread to the other occipital lobe.

In all cases we performed an excision of the meningoencephral scar. The excisions in the frontal or occipital poles were more extensive than in the Rolandic areas. Sometimes we tried the effects of electrical stimulation of the atrophic convolutions at the border of the cerebral scar in an effort to determine the firing epileptogenic points. In the latter cases electrocorticography was also carried out and found useful in showing the region where spontaneous or provoked fits were initiated. When there was enough evidence the abnormal epileptogenic parts of the cortex were included in the surgical resection, but often the results of the electrical stimulation were disappointing and either no responses could be obtained or a generalized fit spoiled the examination.
SURGICAL TREATMENT OF EPILEPSY

In resecting the cerebral scars the white matter often appeared very hard and in those cases the excision was extended to the ventricular wall, especially when the scars were in the frontal and occipital regions. In two patients a bullet was still lodged in the brain, and after resecting the menin-gocerebral scar at the point of entrance, the bullet was also removed. In one case it lay against the falk and over the corpus callosum, and in the other it was on the floor of the anterior fossa. After resections of scars the
defect in the dura was repaired with temporal fascia or with polyethylene film, and in some cases acrylic plates were used for bone defects.

It is extremely difficult to evaluate the results of operations performed for post-traumatic epilepsy. It is obvious from the histories of these patients that convulsions may develop many years after the injury. Therefore the postoperative course must be followed for a very long period before a real evaluation may be made. All that can be done for these patients is to remove a very sclerotic menin-gocerebral scar and leave another clean one with better preserved and vascularized edges. In some cases the electroencephalo-
gram may indicate a favorable prognosis by showing normalization of the

Fig. 1. Electroencephalograms of an epileptic who had had an injury of the right occipital lobe.  
Note improvement of the records after removal of the cortical scar.
electrical record after surgical removal of the scar and surrounding convolutions (Figs. 1 and 2). Another factor that complicates the assessment of the surgical results is the effect of anticonvulsive drugs which most patients take for some time after the operation.

![Electroencephalogram](image)

Fig. 2. Pre- and postoperative electroencephalograms in a case of post-traumatic epilepsy produced by a meningocerebral scar of the right frontal lobe. More than 2 years after frontal lobectomy, the attacks and also the abnormal waves have disappeared.

A summary of the results in our small series, based on reports obtained from 1 to 4 years after operation, is given in Table 1.

It appears from Table 1 that better results are obtained in those cases in which the scars are localized in the frontal or occipital poles, where one can make a wider excision of the surrounding tissue in the way of a partial

<table>
<thead>
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<th>TABLE 1</th>
<th>Results</th>
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<tr>
<td>UnKnown</td>
<td>Bad (Fits Continue)</td>
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<tr>
<td>5 frontal scars and 1 occipital scar</td>
<td>1</td>
</tr>
<tr>
<td>3 scars of Rolandic region (all patients had increased motor deficit after operation)</td>
<td>1</td>
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<tr>
<td>2 scars caused by bullets, with a long intracerebral lesion. Removal of scar of entrance and bullet</td>
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or total lobectomy. Our small figures agree with those of Penfield and his coworkers\textsuperscript{13,14} and Walker.\textsuperscript{17}

B. Cerebral Atrophies. There were 19 patients with atrophic lesions of the brain producing epileptic seizures who were treated surgically. The lesions may be subdivided into the following groups: (a) cerebral hemiatrophy, (b) porencephalic cysts and malformations and (c) cortical atrophy of unknown origin.

(a) The group of cerebral hemiatrophies is the largest and most interesting one. There were 12 patients; most of these were children or young persons between 5 and 18 years of age; 2 were men, 27 and 30 years old respectively. The cause of the disease was apparently known in only half of the cases and was recorded as anoxia and trauma at birth (3 cases), meningitis (2 cases) and an infectious fever disease (1 case).

The epileptic manifestations started usually in early childhood and always before the age of 10 years. Sometimes the first attack was followed by a hemiplegia and in all cases examination revealed a typical picture of infantile hemiplegia with a definite mental defect and deterioration. The epileptic fits were usually focal Jacksonian in character; after local initiation a generalized spread of the seizure often developed. In some patients there were also associated generalized convulsions, petit mal attacks and psychomotor states. The epileptic symptoms recurred frequently in all cases.

Radiographic studies of these patients with infantile hemiplegia conform to the well-known picture of cranial and cerebral hemiatrophy, with different degrees of enlargement of the homolateral ventricle. The electroencephalographic disturbance was also very marked and abnormal discharges were always recorded from the affected hemisphere. Foci of spikes and slow waves were quite frequent over the side of the lesion, but in most cases the electrical disturbance was more diffuse and bilateral, although the abnormality dominated in the diseased hemisphere. A picture of fast spiky activity on the side of the lesion together with slow waves on the other side was also obtained. Besides the focal and propagated disturbances we observed in some records the presence of synchronous and bilateral slow discharges.

The operative procedure was completely different in the first and second half of these 12 cases of infantile hemiplegia with epilepsy. In the first 6 cases only a limited removal of the cystic atrophic areas or partial resection of the most affected cortical regions was performed. Electrical stimulation for detecting the most irritable points of the exposed cortex was used in some of the cases. The results as a whole were disappointing and the seizures usually persisted, although sometimes an improvement was noticed. One of the patients has been free of attacks for more than 1\(\frac{1}{2}\) years since a partial excision, although an electroencephalographic focus persists. In another of these patients there was a striking diminution in frequency of the fits together with an impressive change in the electroencephalogram 2 years after a limited cortical removal of a very hard and extensive lesion of the left cerebral hemisphere (Fig. 3).
These generally unsatisfactory results after limited surgical resections led to the trial of hemispherectomy or hemidecortication by removing all the cortex of the affected hemisphere and sparing the basal ganglia (Fig. 4). The first time we performed this operation was in 1949.11 Up to the present we have done six hemispherectomies for this condition, some of which have been briefly reported.19 Two patients died in the immediate postoperative period. The other 4 are doing well 3 years, 1½ years, 1 year, and 6 months after operation.

*Fig. 3. A case of left cerebral hemiatrophy producing epilepsy. The electroencephalograms show improvement 2 years after a limited cortical excision of the affected hemisphere.*

Our personal impression at the moment is that much better results are obtained by hemispherectomy than by partial resections, but hemispherectomy is also more dangerous, and may not solve the whole problem of epilepsy in these cases. Our first patient is free of attacks, with a complete absence of abnormal discharges in the electroencephalogram 3 years after the hemispherectomy. But in the other 3 cases there are still some abnormalities in the electrical recording from the remaining hemisphere (Figs. 5 and 6). Although all these 3 patients have improved tremendously after the operation and the fits have decreased markedly in frequency, some slight epileptiform manifestations still appear in 2 of them and even more rarely in the third (4 slight attacks in 1 year).

There is no doubt that hemispherectomy is a good operation for the treatment of severe epileptic disturbances of the infantile hemiplegias
(Krynauw, Cairns and Davidson) but it must be emphasized that besides its definite surgical risk there is the possibility that some electrical abnormalities and infrequent clinical discharges may persist after the operation. The subcortical origin of the seizure discharges has been substantiated by recent studies of deep electrical recording in epileptics (Hayne, Belinson and Gibbs, Meyers et al., and Spiegel and Wycis). There is also the possibility that there are abnormal areas in the opposite hemisphere or that the long-lasting abnormal discharges of the patients with cortical hemiatrophy have induced functional changes in the other hemisphere and perhaps these may subside in the course of time after the removal of the affected hemisphere.

(b) There were 3 patients with porencephalic cysts and malformations. One was a child with absence of the corpus callosum associated with cerebral
malformation. Two were young men (31 and 36 years old) who began to have fits at the age of 18, caused by large cerebral cysts. In one an absent temporal lobe was replaced by an enormous cyst isolated from the ventricle by a fine membrane. In the other there was also a large paraventricular cyst, in the parietal region. All that could be done with these 2 patients was to open the cysts widely and form a communication with the ventricular system, but the attacks persisted. Drew and Grant\textsuperscript{3} reported a series of porencephalic cysts with postoperative improvement in the seizures in a large proportion of the cases. They presumed that the favourable results

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**Fig. 5.** Electroencephalograms before and 3 weeks after hemispherectomy in a case of right cerebral hemiatrophy.

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**Fig. 6.** A typical epileptiform discharge recorded from the right side of the brain 1 year after a complete left hemispherectomy in a patient with left cerebral hemiatrophy.
might be attributable to relief of tension by removal or opening of the cyst.

(c) Finally there were 4 patients (aged 18, 30, 46 and 52 years) with focal epileptiform attacks. Neurological examination disclosed some positive findings (monoparesis, some aphasia, sensory disturbances) in 2 of these cases; in the other 2 the findings were negative. The electroencephalograms showed some focal disturbances. Although the operations were performed with strong suspicion of a cerebral neoplasm, only some cortical atrophy of unknown origin was found. In 3 patients nothing more than a surgical exploration was done and the epileptic manifestations have continued. In the last case a cortical resection of the most atrophic parietal region was carried out and the fits have disappeared in the 3 years since the operation was performed.

III. PSYCHOMOTOR AND OTHER FUNCTIONAL DISTURBANCES

This last group of epileptic patients is even more complex because the localization and treatment of the epileptiform area are based on functional evidence only. In fact one can affirm that this section of neurosurgical treatment has emerged from the recent development of electroencephalographic techniques. There are 10 examples in our series: 8 patients with psychomotor epilepsy treated by anterior temporal lobectomy and 2 patients on whom we tried the effect of stereotaxic lesions of the thalamus. The histories of the first 7 patients with psychomotor epilepsy have already been reported and the results are in agreement with the larger experience of Bailey and Gibbs and Green and coworkers.

The patients with psychomotor epilepsy were young, from 22 to 38 years old. While 4 presented a history of pure psychomotor seizures, the other 4 had in addition some generalized convulsive attacks. In the purer psychomotor forms the electroencephalographic studies demonstrated the typical abnormal discharges in the temporal regions with spikes and sharp waves and sometimes also some slow waves, as described by Gibbs, Gibbs and Fuster. In the other 4 patients, with a combination of psychomotor and generalized seizures, besides the focal temporal discharges other more diffuse dysrhythmic abnormalities were found.

The operation performed in all these cases was removal of the more electrically abnormal temporal lobe. The excision corresponded to the tip of the temporal lobe, and was about 3 to 5 cm. in length. All this anterior temporal tissue was removed by suction. Before lobectomy, electrical stimulation under local anaesthesia and electrocorticography were carried out. There were no marked neurological or psychical changes following the anterior temporal lobectomy, which was performed on the left dominant side of the brain in 3 cases and on the other side in 5.

The results have been followed for only 1½ to nearly 3 years after operation. They have been generally good in the group of patients who had psychomotor seizures only; the attacks have not recurred in the period of observation (1½ to 2 years) and only 2 of these patients have experienced occasional slight visceral sensations without any spread or form of seizure.
There has also been a great improvement in the electroencephalograms, and the spikes and abnormal temporal discharges have disappeared or greatly diminished (Fig. 7). An interesting fact that was noted in the serial electroencephalographic studies in our cases is the progressive normalization of the record in the months and years after temporal lobectomy, and this change had no relation with any medical anticonvulsive therapy.

**Fig. 7.** An example of psychomotor epilepsy treated by right temporal lobectomy, showing the great improvement in the electroencephalogram nearly 2 years after operation. This was accompanied by a good clinical result.

In contrast with this favourable response in the group of pure psychomotor seizures, anterior temporal lobectomy was not very useful in those other 4 patients with a combination of psychomotor and generalized convulsive attacks. Although there was some occasional postoperative improvement in the electroencephalographic records, the seizures have persisted. It seems, therefore, that the surgical treatment for psychomotor epilepsy should be limited to the purer clinical forms, with a well-localized electrical focus in the anterior temporal region.

Finally in 2 epileptic patients we tried the therapeutic effects of making small stereotaxic lesions of the thalamus. One of them, with a curious localized myoclonic form of attacks, has already been reported, and some improvement was obtained. In the experience of Spiegel, Wycis and Reyes it is not possible to abolish completely the subcortical petit mal attacks by diencephalic stereotaxic lesions. They believe that there is a relatively wide area where the abnormal discharges may originate and therefore small lesions produce only partially successful therapeutic results. Further neurophysiological and neurosurgical studies are necessary in this field before assessing the possibilities of localized subcortical lesions.

**SUMMARY**

A brief review is presented of personal experiences with 130 patients
operated upon for epilepsy during the last 5 years. In 90 cases the epilepsy was secondary to expansive intracranial lesions. Because of their rarity, tuberculomas, cysticercosis and hydatid cysts are specially mentioned.

In the group of localized cerebral scars the best results were obtained in those cases in which the scars were in the frontal or occipital poles, where wide resections can be performed.

In 12 cases of cerebral hemiatrophy with infantile hemiplegia the epilepsy was treated by partial cortical resection or complete hemidecortication. Six examples of hemispherectomy are reported and in the 4 survivors good results were obtained although 3 still have occasional seizures and contralateral electroencephalographic disturbances.

Anterior temporal lobectomy seems to be a useful treatment for the pure forms of psychomotor epilepsy with an electrical focus in the temporal region (4 cases), but it fails in other epilepsies who have generalized convulsive seizures associated with the psychomotor attacks (4 cases).

Stereotaxic lesions of the thalamus were tried in only 2 epilepsies. One of these patients, who had a rare form of partial myoclonic seizures, obtained some relief.

REFERENCES