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RESULTS OF SURGICAL THERAPY FOR FOCAL EPILEPTIC SEIZURES*

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A. Introduction

FROM TIME TO TIME a surgeon should scrutinize his own results and make that scrutiny available to others. The purpose of this study is to determine the results of cortical excision in the treatment of epileptics over a six-year period from 1945 through 1950 inclusive; to summarize briefly two earlier reports of previous surgical therapy (Penfield and Erickson, 1941; Penfield and Steelman, 1947), and so to provide adequate information upon which the success of the procedure in our hands can be judged. The follow-up analysis of this series was carried out by the junior author, as in the previous studies.

In these three successive series which extend over a period of 20 years there is, of course, a gradual evolution of method which has served to enlarge the field of operation. An attempt has been made, in this present paper, to analyse the reasons for the success or failure of operation, and to indicate those features which have prognostic value. The physical and, particularly, the mental effects of cortical removal will be referred to superficially here and given more exhaustive study in later publications.

The senior author turned his attention quite early to the problem of focal epilepsy by studying the cytology of the brain and the healing of brain scars. This was followed by a visit to Breslau, to the Clinic of Otfried Foerster who had, at that time, carried out radical excision of atrophic brain lesions in a small series of patients who suffered from focal epilepsy. The excised tissue was studied by all available methods (Foerster and Penfield, 1930) but the nature of the irritant which makes areas of gray matter become epileptogenic was not discovered. Unfortunately, the nature of this irritant is still the unsolved riddle, the secret of a sphinx that seems to smile at our everchanging therapy.

Much has happened in this field in 25 years (Penfield and Jasper, 1954). We have electroencephalography now and better anticonvulsive drugs. We have learned the localizing value of epileptic patterns and can detect the site of initial discharge in brain stem epilepsy as well as cortical seizures. We can recognize temporal lobe epilepsy and understand the mechanism of its production when birth compression is its cause.

And yet, since we are ignorant of the essential cause, therapy must continue to be judged empirically.

B. Rationale

Radical operation depends, for its rationale, upon the conception, elaborated repeatedly by Hughlings Jackson, that every epileptic seizure begins with ganglionic discharge in some area of gray matter. The aim of surgical excision is to remove the focal area in which epileptogenic discharges originate and thus put an end to the seizures.

This would be as simple as it is logical if the focal area were invariably discrete and if the surgeon could be sure to leave behind him normal cortex in a normal postoperative state. But completely discrete lesions are rare and the brain from which a part has been removed cannot be called normal, however great the effort to

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avoid scarring and to prevent alteration in the circulation of remaining gyri.

Sir Victor Horsley (1886) introduced the technique of subpial removal of cortical convolutions, and the technique is still in use. When convolutions are removed thus by suction, leaving a bed of white matter, and the circulation of the pial banks of the surrounding convolutions is spared successfully, there is little or no scarring. Electroencephalography at three weeks after operation may give little or no evidence of cortical abnormality. The difficulty, however, is to remove all of the epileptogenic cortex and that only.

An epileptogenic focus is not a point. It is a zone of cortex. This may be as small as a single shrivelled gyrus or it may consist in a group of somewhat atrophic gyri. When the obviously abnormal area has been removed there may remain hidden areas of abnormal cortex capable of carrying on the spontaneous discharge.

Habitual seizures rarely, if ever, begin at the time of brain injury. There is a ripening period of months or years that follows the initial insult. On the other hand, in many cases seizures never develop after brain injury.

Many patients have traumatic injuries of the brain without developing subsequent cerebral seizures. Many infants suffer acute anoxæmia of some area of cortex and consequently go through life with areas of focal cortical atrophy, large or small, without having to face the "curse" of epilepsy. On the other hand, many other such individuals do suffer from attacks because such abnormal areas of brain do become epileptogenic. Furthermore, a patient who is epileptic may have a large lesion of one hemisphere and yet seizures may seem to arise in only one area of injured cortex and not other areas of injured cortex.

C. Localization of the Epileptogenic Focus

The focus is that area in which the periodic outbreak of discharge seems to originate, the outbreak that gives rise to the initial phenomenon of the habitual attack. It is the area in which electrical potentials are highest between attacks but the extent of epileptogenic cortex may be considerably greater than these findings would suggest.

The position of the focus and of the abnormal epileptogenic cortex may be determined as follows:

1. Sometimes the nature of the initial phenomenon (sensory, motor or psychical) betrays the position of initial discharge. Thus the *ictal pattern* provides initial localizing guidance.

2. *Electroencephalography* may detect abnormal electrical potentials at the site of the pacemaker between attacks, and an enormous augmentation of such discharges at the time of attacks. When the recording electrode is placed directly upon such an area during operation, or when the scalp electrode is over a superficial cortical focus before operation, discharges of high potential and short duration ("spikes") are usually recorded. If the electrode is at a distance the disturbance may appear in the form of sharp waves or abnormal alterations in brain wave rhythms. At the onset of a seizure these interictal spike discharges become multiple.*

3. Electrical *stimulation* of the exposed brain may reproduce the initial phenomenon of the patient's habitual minor seizures and this may be taken as confirmatory evidence of the general position of the focus.

4. At the time of operation, *inspection* of the exposed brain often leads the surgeon to a presumptive identification of the epileptogenic focus. It can never be more than presumptive, for identification depends on the information just described. On the other hand, when the cortex is completely normal its removal gives little or no hope of a successful therapeutic result unless that removal serves to give access to initially hidden areas of objectively abnormal gray matter.

D. The Epileptogenic Lesion†

If the neurosurgeon finds that he is dealing with a "space-occupying" lesion, such as an encapsulated tumour, the focus is usually discovered in atrophic gyri nearby, gyri which have been narrowed by the progressive pressure of the neoplasm.

If a brain cyst is found, or an area of complete destruction such as that produced by intra-

*In certain areas of the cortex these epileptogenic discharges are sometimes associated with a general suppression of electrographic activity. (See Penfield and Jasper, 1954.)

†The epileptogenic lesion may be defined as that area of gray matter in which spontaneous epileptogenic discharges arise. Tumours, cysts, vessel abnormalities, areas of destruction, adhesions—these may be associated findings, but the epileptogenic lesion is the gray matter that is still alive but has taken on this abnormal activity because of some pathological influence.

cerebral hæmorrhage or thrombosis, the focus may be found in the narrow gyri of tough consistency that border the cyst or the area of destruction. In such cases numerous gyri of this type may be discovered forming the boundary of the lesion, and we have learned by experience that such atrophic gyri should all be considered real or potential areas of origin and therefore should be removed when practicable.

If the abnormality of the cortex has been produced by birth compression the focus may be found in an area of small gyri (microgyria) or in shrivelled buried gyri.

Occasionally the surgeon may not encounter abnormal gray matter of the type that he considers could be epileptogenic until after he has made a partial removal guided by other evidence. Then he comes upon a zone of cortex that is sclerosed, yellowish, atrophic, tough. This may be on the undersurface of a lobe, as in incisural sclerosis of the undersurface of the temporal lobe. There may be atrophic gyri deep in the interhemispheral sagittal fissure.

The electrocorticograph is of increasing importance at the operating table before excision of convolutions and also as a check-up immediately afterward, and a help in deciding whether further removal is indicated and where. Spike discharges and sharp waves may appear on the border of an initially removed area where they were not observed before. This is not an evidence of traumatic effect but indicates that some area of epileptogenic cortex has been overlooked.

The "diviner's rod" is said to be used to find water. The electrograph detects epileptogenic abnormality, whether on the surface or hidden, and the surgeon must find it with a minimum of damage. Removal of normal convolutions will not bring therapeutic success, and therefore electrographic evidence is not sufficient to justify excision of normal brain unless it leads to exposure of hidden abnormality.

After years of experience with the gross appearance and the microscopic structure of the gray matter of epileptogenic areas, it is possible to point out certain characteristic features.

In the total series of patients, 78 were found to have clearly atrophic lesions of the cerebral cortex. Ten more had cerebral cysts with atrophy of surrounding gyri. There were 45 examples of meningocerebral cicatrix. Such a scar results from cerebral laceration or local abscess formation and drainage which produces a central core of connective tissue, densely adherent to the dura. This central mixed scar is surrounded by atrophy and gliosis of cerebral tissue.

In 25 cases there were cortical areas of microgyri, such as are produced by birth compression, obvious to inspection on the surface.* In the ten examples of cerebral cyst mentioned above, there were microgyri around the margins of each cyst.

Incisural sclerosis of the hippocampal and uncal area is the most frequently encountered pathological entity. The manner of production of this lesion by herniation of temporal lobe through the incisura of the tentorium with arterial compression is described below under the heading of Birth Lesions.

In the convolutions which form the epileptogenic focus a varying degree of atrophy is often evident on inspection. The consistency is increased so that the tissue enters the aspirator (sucker) used for cortical removal less readily. Sometimes it is so tough and rubbery that it must be removed by sharp dissection.

In this series of cortical excisions the operator was prepared to say that there was gross abnormality in the tissue removed in all but eight cases. Presumably the removal in those eight cases was made entirely on the evidence provided by electrocorticography and electrical stimulation. It may well be significant, in passing, that only two of the eight patients were eventually placed in the success groups of the follow-up analysis.

Histologically, the toughness of epileptogenic cortex seems to be due to the change of protoplasmic astrocytes into fibrous astrocytes in the gray matter and the over-all increase in these cells. Careful microscopical study also often reveals the presence of minute foci of cortical destruction, either acute or long standing, as though at scattered points the circulation had become progressively inadequate even many years after the initial injury to the brain.

In general, it might be concluded that one feature is common to all of these cortical epileptogenic lesions, i.e., a slight persistent inadequacy of circulation. This could cause the patches of acute swelling of oligodendroglia which are found, the fibrous gliosis of astro-

*When such gyri were encountered deep to the surface, it was often difficult to see the size of the gyri although the toughness of the tissue removed was obvious. In such a position the operator did not call them microgyri in his operative note.

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cytes, the occasional dropping out of ganglion cells, and the minute islands of phagocytosis. These common features are found in convolutions compressed by an expanding lesion, but they are also found in areas which had been partly destroyed by acute ischæmia years before the onset of the seizures.

It remains to be shown by further histological study, or by other methods of analysis, wherein the non-epileptogenic atrophic gyrus differs from the epileptogenic atrophic gyrus. It remains to be determined whether this hypothetical inadequacy of circulation is really present between attacks and whether it may thus be related to the cause of continuing interictal discharge, or whether it is a purely passive expression of the excessive increase in the metabolic demands made upon the tissue during a seizure.

It remains to be shown, also, just what the nature of the local biochemical peculiarity may be in the epileptogenic cortical focus. The work on this subject which has been carried out in the Neurochemistry Laboratory of our associate, Dr. K. A. C. Elliott, has been summarized by him in the following personal communication:

Biochemical studies have shown an anomaly in the metabolism of acetylcholine in human focal epileptogenic tissue (Tower and Elliott, 1952, also 1953). Normal brain tissue, when incubated *in vitro* under appropriate conditions, produces free acetylcholine and also increases its content of the bound form of acetylcholine. Epileptogenic tissue fails to produce this bound acetylcholine. The same failure is found in cortex from animals suffering repeated seizures following the administration of methionine sulfoximine. This anomaly may be the basis for the tendency of the tissue to initiate seizure activity *in vivo*. Epileptogenic tissue *in vivo* may tend to produce free, physiologically active acetylcholine at times when normal brain would store the acetylcholine in the inactive bound form.

The nature of the anomaly is not yet clear from the biochemical standpoint. The same defect appears in normal tissue *in vitro* under conditions of partial anoxia. The production of bound acetylcholine can be restored to normal by the presence *in vitro* of glutamine or asparagine. The production of bound acetylcholine is found to be greatly increased in tissue from animals treated with certain narcotics and anticonvulsants. Evidently the changes in production of bound acetylcholine depend in a complex manner on the condition of metabolic systems. Earlier work (Elliott and Penfield, 1948) had shown that there is no simple relation between respiratory or glycolytic capacity and epileptogenic activity.

E. THE NEUROSURGEON'S TASK

This field is a difficult one. There are as yet few rules of routine procedure. Success must depend upon balanced clinical judgment.

The neurosurgeon who would enter the field must learn to identify the abnormal cortex in which epileptic discharges are apt to originate by means of clinical, physiological, and electrical analysis. He must carry out his excisions with a minimum of disturbance of the circulation of the remaining gyri. He must balance the chance of freeing his patient from seizures against the risks and functional losses that may be associated with ablation.

Thus it is evident that conclusion as to location of an epileptogenic focus must depend upon all of the available information: (1) seizure pattern; (2) electroencephalography before and electrocorticography during operation; (3) electrical stimulation of cortex under local anæsthesia; (4) the appearance and consistency of the cortex.

There are other important aids—the help of a neurologist who understands all forms of modern medical treatment and also repeated consultation with a wise electroencephalographer who understands the total problem. When operation is being considered it is almost always possible to identify, by means of the E.E.G., those cases in which the originating focus of epileptic discharge is subcortical. Thus, with rare exceptions, it is now possible to avoid negative exploratory craniotomies.

F. MATERIAL

During a six-year period (1945-1950 inclusive) 234 patients were treated by craniotomy for the relief of epileptic seizures. This series will be compared in the final discussion with the 76 similar cases analyzed for the preceding six years (1939-1944 inclusive). None of the patients included had an intracranial tumour. During the same period approximately 80 additional patients who actually had such tumours came to operation with the primary complaint of seizures rather than pressure. They were operated upon also—but they will not be considered in this study.*

All of these patients reported here were under the care of one of the authors (W.P.) and were operated upon by him or by one of his neurosurgical assistants.†

*Still other patients whose seizures were obviously secondary to intracranial tumour were admitted to the Montreal Neurological Institute during this period but will not be included in this analysis. Most of them were obviously suffering from increase of intracranial pressure and many of them were operated upon by our associates, Dr. William Cone and Dr. Arthur Elvidge.

†Drs. Theodore Rasmussen, Keasley Welch, Frank O'Brien, John Hanbery and Maitland Baldwin contributed each in turn to the study and the care of these patients and we owe them an obvious debt of gratitude.

1. *Follow-up Method*

Our follow-up method was as follows: An attempt was made to discover the fate of every patient operated upon in the six-year period 1945-1950 inclusive. The follow-up was continued at least to the end of 1951, so that there is a minimum follow-up of one year from the date of operation and a maximum of seven years. In most instances, the files consisting of inpatient and outpatient records and correspondence did not contain all the information required, and such patients received a questionnaire form to be completed and returned.

Approximately 190 patients filled out the form and returned it. This questionnaire was made as simple as possible with the intention that a letter would be sent later asking for answers to specific questions if further information should be required. The patient's relatives were encouraged by the questionnaire to write a letter to add their opinions to that of the patient, and in about half of the cases they did so. In some instances it was necessary to write a supplementary letter to the patient's own doctor, for elaboration of ambiguous replies. A relatively small number of patients lived in Montreal or were able to return for examination during 1952. Fuller details are thus available for these cases.

In spite of the inevitable difficulty of securing exact information by correspondence, the summaries in the following pages seem to provide material adequate for assessment of the value of cortical excision as used for the treatment of focal epileptic seizures during this six-year period.

2. *Sex Incidence*

Table I shows the sex distribution in the group of 234 patients with epilepsy due to non-neoplastic lesions of the brain. It shows, also, the relationship of this distribution to probable cause of seizures.

TABLE I.

SEX INCIDENCE AND RELATIONSHIP TO CAUSE

Etiology	*Male*	*Female*
Birth lesions	57	27
Post-traumatic	61	19
Miscellaneous	35	35
Total cases (234)	153 — 65%	81 — 35%

Males outnumber females as a whole, 65% to 35%. In the post-traumatic epilepsy group they outnumber the females 3 to 1. This might be expected, as the incidence of head injury is generally greater in males, and especially so in our series which includes servicemen who have had war wounds of the head. However, it was unexpected that males should have exceeded females 2 to 1 when the cause was birth injury or birth anoxia. Holt (1940) gives the average circumference of the head of the newborn infant as 13.9 inches for the male against 13.6 inches for the female, a rather small difference in size to account for the difference in incidence of brain injury.

The cause in each case was frequently a matter of difficult decision. But, after operation and microscopical study of excised tissue, a presumptive conclusion has been made that must be correct in the great majority of instances. This will be discussed further.

3. *Family History*

In all but 10 of the 234 cases in this series our hospital records gave specific information regarding the occurrence of seizures in other members of the family. Eleven near relatives of 10 patients have been, or are, afflicted with epilepsy. Thus, 4.4% of the patients have a family history of some convulsive disorder. More distant relatives, of an additional seven patients, had had one or more convulsions. As the average number of near relatives, calculated from the Canadian Census figures, is about five for each patient, the incidence of epilepsy in the total number of near relatives is thus estimated to be below 1%.

This approximates the incidence of 0.9% for all forms of epilepsy among Americans drafted for service in the U.S. Army during the Second World War. Alström (1950), referring to epilepsy of known etiology only, in Sweden, gives the incidence of epileptic relatives as 1.1%.

Therefore, it seems fair to conclude that hereditary tendency may be ruled out as a factor in the development of focal cortical seizures among the patients in this series.

A misleading example of apparent hereditary tendency might be cited here.

Two sisters (B.S. and M.N.) suffered from seizures due, in each case, to a temporal lobe focus. Each was operated upon and, in each case, sclerosis of the mesial zone of the temporal lobe was found and the area removed. The sclerosis was clearly due to temporal herniation through the incisura of the tentorium at the time of birth. The significant factor in the production of "incisural sclerosis" was the pelvis of the mother through which each child came into the world, rather than a common inheritance of a tendency to epilepsy.

There was a history of mental or personality disorder among relatives of 10 patients. It is interesting that seven of these were in families in which epilepsy was also reported among the relatives.

G. ETIOLOGY

It was stated above that there are common features which may be recognized by gross and microscopical examination in the excised epileptogenic tissue. But it is obvious that these

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common pathological changes are produced in a great variety of ways by many pathological agents. These agents are summarized in Table II.

TABLE II.

CAUSE OF FOCAL CORTICAL SEIZURES

	Known	*Probable*	*Total*	
1. Birth lesions	67	17	84 —	36%
2. Postnatal traumatic lesions.	70	10	80 —	34%
3. Local suppuration	13	0	13 —	5%
4. Cortical thrombophlebitis	6	4	10 —	4%
5. Angiomas	7	0	7 —	3%
6. Hæmangioma calcificans	4	0	4 —	2%
7. Miscellaneous			18 —	8%
8. Lesions of uncertain etiology			18 —	8%
		Total cases	234 —	100%

This table shows the nature of the cerebral lesion or the mechanism of its production, so far as it could be ascertained from the history of the patient's illness, the findings of special examinations, and the gross features of the lesion discovered at operation or its later investigation by histological methods.

The table illustrates the fact that about one-third of all cases fall into a group in which birth injury or anoxæmia was assigned as the cause. In a second group of about one-third of the cases the cause was postnatal trauma. These and the smaller groups are considered below separately for purposes of analysis and comparison. The cases in which there was good presumptive but not certain evidence as to cause are placed under the heading "Probable" in Table II.

1. *Birth Lesions*

An abnormality that was produced in the brain during birth we have called a birth lesion. There may have been actual trauma by instruments, or unequal cerebral anoxia, intracranial hæmorrhage or herniation of temporal cortex through the tentorial opening. One of these, or a combination of them, may be the actual cause of the abnormality, but it seems likely that cortical ischæmia or anoxia most often plays the important role.

It is, to some extent, the area of brain injured at birth which determines whether or not the child's history will give evidence of it. A lesion of the central region produces a defect in the use of arm and leg on one side that parents usually detect within the first six months. Failure of the infant to suck well during the first few weeks probably points to diffuse injury or perhaps compression of the brain stem.

But a lesion of the temporal lobe may be produced by head compression during birth without resulting in any defect in the child's behaviour which even the most expert pædiatrician can detect.

Earle, Baldwin and Penfield (1953) made a pathological study of 157 consecutive cases in which seizures originated in the temporal lobe. Many of these cases are included in this follow-up study. In 100 of these cases (63%) it was concluded that anoxæmia or compression at the time of birth had been the actual cause of lesion and seizures. Smallness of the middle cranial chamber, as shown by x-ray, is sometimes found and may be taken as strong confirmatory evidence that the lesion dates from birth or early infancy.

The pathological lesion thus produced consisted in sclerosis of the infero-mesial zone of the temporal lobe. This they showed could be produced by herniation of temporal convolutions through the incisura of the tentorium into the posterior fossa, thus probably producing temporary compression of the arteries which cross the free edge of the tentorium to supply the uncus, hippocampal gyrus and adjacent cortex.

This pathological change they designated as *incisural sclerosis.* At the time of operation this lesion can be recognized easily by the colour and consistency of the tissue (Penfield and Baldwin, 1952). Consequently, when it is encountered and when no other obvious cause is discovered, and when the seizures are arising in the area, the conclusion may be drawn that birth compression is the real cause, even though no abnormality was reported by obstetrician or parents at the time of birth.

Febrile convulsions of infancy and childhood: The fact that the first convulsion may have occurred at the time of a febrile illness early in life does not necessarily throw any light on the nature of the cerebral lesion. We believe that the fever in such cases is most often a precipitating factor, rather than evidence of encephalitis. Thus, the child with an unsuspected atrophic lesion of the cortex may have his first attack due to the precipitating influence of a febrile illness, or even a mild head injury. The objective findings at operation bear out this

belief.* In Table II there are 84 examples of birth lesion, and in 28 of these cases there was history of an early convulsion during a febrile illness early in life.

TABLE III.

TIME OF ONSET OF RECURRING SEIZURES DUE TO A BIRTH LESION — 67 CASES

Age at onset	*Lesion in temporal lobe*	*Lesion elsewhere*
0 to 5 years	1 — 4%	20 — 46%
5 to 10 years	8 — 34%	9 — 21%
10 to 20 years	10 — 42%	13 — 31%
20 to 55 years	5 — 20%	1 — 2%
Totals.....	24	43

Onset.—Table III shows the age of onset of the habitual seizures following a cerebral birth lesion. Only the 67 cases in which the cause was certain, rather than presumptive, are chosen for this analysis. It is seen that temporal lobe seizures commence most often between the ages of 5 and 20 years, 78% in all. On the other hand, those affecting other parts of the brain become manifest in the first five years of life in almost half the patients, 48%. Another 40% appear between ages 5 and 20. The mean age of onset of convulsions is 13 years in the temporal lobe cases and six years in the others.

It has been pointed out above that the cause of the temporal lobe damage in birth injuries is evidently temporary vascular occlusion and ischæmia of infero-mesial temporal cortex. On the other hand, the lesions elsewhere are usually produced by direct trauma or hæmorrhage. Superficially placed temporal lobe foci may have originated from direct cerebral trauma, but these lesions are rare when compared with incisural sclerosis of the temporal lobe.

First-born.—The patient's position in the family has been noted in those cases where a birth injury is suspected as the cause of later convulsions. In the 53 case records where this evidence is available, it is found that there are 33 first children, 9 second, 6 third, 2 fourth, and 3 fifth or later. The Canadian Census figures for 1948 give the following proportions in the total population: 31% for first-born, 25% for second, 15% for third, 9% for fourth, and 18% for the remainder. If one can assume that the deaths in each group are in proportion to the total numbers in each, then first-born children are more commonly met with in the patients with focal epilepsy due to birth injury in our series than in the general population.*

2. *Traumatic Lesions (Postnatal)*

In many instances, it was obvious that a local lesion of the brain had been produced at the time of trauma. This was especially so in the cases of penetrating injury and depressed fracture of the skull. On the other hand, when the patient had sustained a closed head injury without perforation of the dura by depressed bone fragments, the evidence was often not so clear. But the presence of a hemiparesis or sensory loss, or of an aphasia dating from the time of the injury, may be taken as an indication of a localized lesion of the brain. Unconsciousness, following trauma, indicates only the degree of severity of the trauma.

In this series we have not accepted trauma as the cause unless there was a localized epileptogenic lesion that seemed to be beneath the point of impact, or in a few cases in a position favoured as a site of contrecoup injury, such as the tips of the temporal lobes or tip and undersurface of the frontal lobes. In the case of direct or of contrecoup injury, it is expected that adhesions between dura and brain will be present if that trauma is to be accepted as cause.

In one case, for example, the epileptogenic focus proved to be an area of atrophic cortex with fine adhesions between it and the overlying dura. It lay directly beneath a scar in the scalp that marked the point of impact of a blow. The patient had no recollection of the event! Nevertheless, this was taken as adequate evidence of the traumatic nature of the original lesion.

In another case of so-called closed injury of the skull, a basal fracture in the middle fossa was found and there was a tear in the dura at the site of fracture. This had produced erosion of the bone at the site of the dural defect and there were dense, but localized, adhesions between dura and brain in the neighbourhood. The patient's epileptogenic lesion was on the

*It is of course also true that in groups 3 and 4 (Table II, cortical thrombophlebitis and localized suppuration) there were initial seizures at the time the lesion was acquired in the majority of cases. After such infections there followed a lapse of time (the ripening period) before the eventual onset of habitual cerebral seizures.

*No allowance has been made for the changing birth rates, and the population figures apply to all individuals. Furthermore, the case histories are more likely to contain reference to single children, i.e., first-born, than to the position of other patients in a fraternity. Both of these factors would tend to make the incidence in the patients under discussion appear proportionately larger. No correction is made for American and foreign origins.

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undersurface of the temporal lobe, in the region of this abnormality.

Onset of post-traumatic seizures.—Table IV shows the interval between the postnatal trauma and the onset of habitual seizures, indicating a mean interval of about a year after penetrating head injury, and of slightly less than a year after head injury.*

TABLE IV.

TIME OF ONSET OF RECURRING SEIZURES DUE TO POSTNATAL TRAUMA

Interval after injury	*Dura penetrated*		*Dura not penetrated*	
Less than 1 year	21	50%	25	58%
1 to 5 years	11	26%	6	14%
5 to 10 years	8	19%	7	16%
Over 10 years	2	5%	2	5%
Interval uncertain	0		3	7%
	42		43	

The table is made up with regard to the time of onset of habitual recurring seizures. Five of these patients also had one or more seizures immediately following injury. In three of them the habitual attacks began at four, five and six months after the injury. In the case of one, the onset was delayed for one year, and in the remaining case for five years. Three other patients had seizures during the acute phase of injury, which could also be said to be the time of onset of their habitual seizures. That is to say, no attack-free interval could be recognized after the injury.

3. *Localized Suppuration*

Table II shows that there were 13 patients who suffered from some form of intracranial infection before the onset of their seizures, and in whom it appeared that the infection had a causal relationship to the attacks.

Seven of these patients had had a brain abscess previously treated by drainage. Four had a primary focus in the ear, and in one, although the intracranial abscess followed a tonsillectomy, an unrecognized focus of infection may have existed in the middle ear as well. One patient had an osteomyelitis of the frontal bone following scarlet fever, with later intracranial extension of the infection. The other patient, with a parieto-temporal abscess, had no detectable primary source of infection.

Onset.—The interval between the acute stages of the infection and the onset of seizures was 2 years in three cases, 3 years in two, and 5 and 10 years respectively in the other two. One additional patient had had a subdural abscess secondary to mastoiditis and developed habitual seizures 13 years later.

The five remaining patients had infected penetrating wounds of the brain and are considered with the traumatic cases with regard to the onset of their seizures in relation to the causal agent.

4. *Cortical Thrombophlebitis*

Ten patients were believed to have suffered a cortical venous thrombosis in infancy which had led to changes in the cortex, later to produce an epileptogenic focus. In most patients with such a history, as mentioned above, it appears that the febrile illness is only a precipitating factor when the brain has already been disturbed by a birth lesion, a very early trauma, or a vascular abnormality.

Before accepting cortical venous thrombosis as the probable cause of seizures, we have demanded evidence of brain damage at the time of the pyrexial illness. This evidence is usually hemiparesis, sensory loss, visual field defect, or aphasia which persisted for some days or even weeks, thus differing from the usual post-ictal neurological defect which ordinarily clears up in a shorter period of time. The distinction, however, must often remain in doubt.

5. *Angiomas*

Table II shows that there were seven angiomas in the series. These included three capillary hæmangiomas, one racemose arteriovenous communication, one cortical arteriovenous malformation and one capillary hæmangioma, previously excised in another clinic without cessation of seizures. In one of these cases calcification was present. In all of these cases the vascular abnormality was excised, together with the surrounding convolutions which formed the epileptogenic abnormality. In the seventh case a large racemose angioma of the right central region was investigated at operation but not excised for fear of producing a severe hemiplegia.

*Patients in this latter group are more likely to have been placed there if the interval between the trauma and the onset of seizures was short, and some truly post-traumatic epileptics may have been classed in the uncertain group because of the doubt as to causal relationship of trauma because of a long interval between trauma and seizures.

There were four more cases of a different type, described in the next section.

6. *Hæmangioma Calcificans*

There were four of these epileptogenic lesions. They constitute an interesting pathological group previously described by Penfield and Ward (1949). Degeneration is marked and vascularity is much reduced. In all of the four cases of this present series, the lesions were in the temporal lobe of one side or the other. This was true also of the previously reported cases.

7. *Miscellaneous*

Brief reference may be made to the 18 lesions included under this heading:

Postnatal ischæmia.—Two cases. In one case there had been arrest of respiration during general anæsthesia and in the other there had been a "narrow escape" from drowning.

Lead encephalopathy.—One. Ingestion of lead-containing paint was followed by coma of two days' duration with generalized convulsions. Habitual seizures began a few months later.

Cerebral malaria.—One. A little girl, aged 2, had malaria of the cerebral type. At 6 years habitual seizures appeared. At operation there was abnormality within the right Sylvian fissure suggesting an earlier inflammatory process, particularly over the island of Reil.

Tuberous sclerosis.—Two. In one case the condition was recognized only on postoperative microscopical study. Attacks were not arrested by excision.

In the other case, the nature of the pathological condition was clearly diagnosed before operation, but as the attacks were invariably of the same pattern and the E.E.G. focus was well localized, it was finally decided to yield to the parents' insistence and excise the epileptogenic area. It consisted of tough, rubbery gyri. Abnormality was obviously present elsewhere, but the result of operation is nevertheless excellent for the time being. The parents are delighted. During the three years since operation, attacks have stopped and the boy has improved greatly in intelligence and behaviour. He has improved in school work, and at home. He has learned his "catechism", a thing he had repeatedly failed to accomplish before operation! Since he took phenobarbital before and after operation, this is a good example of the effect of excision of "nociferous cortex" on the patient's intelligence and behaviour (Penfield, 1952).

Uræmia and acute nephritis.—One. At the age of 10 years, this condition, associated with hypertension, was thought to have produced the epileptogenic lesion of the brain. Focal motor attacks had been described at the time of his acute illness and recurred as habitual attacks nine years later.

Encephalitis.—Four. The diagnosis of encephalitis (probably due to virus infection) is always difficult to make with certainty. But examination of the case histories has led us to this conclusion in four cases. There was pleocytosis in the cerebrospinal fluid of two of the patients at the time of their initial illness, according to records from other hospitals. No bacteria were found, and the description of the illness was compatible with the diagnosis of encephalitis. The third patient, who was unconscious for a prolonged period during measles, was thought to have had a morbilliform encephalitis.

The fourth patient had had convulsions during a febrile illness associated with slight meningismus, but without any recorded changes in the cerebrospinal fluid. She was comatose for 12 days and developed a left hemiplegia at that time. We found evidence of moderate bilateral enlargement of the lateral ventricles in the pneumoencephalogram and, on operative exposure, extensive atrophy of the cortex was encountered.

Hæmorrhagic disease of the newborn.—One. During the first few days of life, this patient was noted to have petechial spots on her skin and mucous membranes. She also had melæna. On the third day, evidence of a cerebral lesion developed suddenly and physicians considered that the body had suffered a cerebral hæmorrhage. Years later, when craniotomy was carried out for relief of seizures, she was found to have a localized area of brain destruction, now filled in by an outpouching of the lateral ventricle. Hæmorrhagic diathesis of the newborn was taken as the cause of intracerebral hæmorrhage.

Brain tumour suspect.—Three. Included in the series are three patients who were suspected of having an intracranial tumour, but pathological examination of the biopsy specimens did not confirm this impression. These patients are still alive and show no evidence of spread of tumour inside the skull. For this reason they are retained in the non-neoplastic group but none had a cortical excision at the time of operation.

Meningitis.—Two. One patient, who had had a meningitis at the age of 7 years, developed seizures five years later. At craniotomy, 21 years after the onset of his seizures, the main abnormality found was thickening of the arachnoid over the exposed cortex. The second was a girl, whose attacks began six years after an illness diagnosed as meningitis, and who was found at craniotomy to have widespread but fine subdural adhesions. In addition, she had calcified granulomatous masses in both frontal lobes, one of which was removed at operation, the other only visualized by radiography. She was known to have had pulmonary tuberculosis in early life, and the granuloma was assumed to be tuberculous.

Arteriosclerosis.—One. Only one patient was over 55 years of age at the time of onset of his seizures. The exposed cortex in this case showed diffuse atrophic changes with slight yellowing of the fluid in the subarachnoid spaces. Our tentative conclusion was that the cortical abnormality responsible for his seizures was caused by sclerotic disease of the cerebral vessels. He had generalized arteriosclerosis without hypertension.

8. *Lesions of Uncertain Etiology*

In the remaining 18 patients, either the initial lesion is unknown, or two or more etiological factors could equally well be incriminated, so that the diagnosis is classed as uncertain.

9. *Etiological Significance of Age of Onset*

The time of onset* of the recurring seizures, in those cases in which there was clearly a lesion dating from birth, is collected in Table III. It is seen that the onset is considerably later in the temporal group. This may be due to the fact that direct trauma or laceration is apt to occur in other parts of the brain while incisural sclerosis is an ischæmic lesion. But the latency is, on the average, greater during infancy and childhood than later in life, regardless of location.

*In this section, time of onset of seizures is taken to be that time when the habitual attacks seem to have begun. A convulsion associated with a febrile illness of childhood or seizures occurring at the time of brain abscess or brain trauma are not considered as the signal of onset of recurring seizures.

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This conclusion is borne out when Table IV is compared with Table III. In adult life half of the post-traumatic epileptics experience the onset of recurring attacks in less than one year. In contrast to this, half of the temporal cases due to an accident of birth begin around the age of puberty. Among adults there was little difference in the time required for "ripening" of the epileptogenic process when the penetrating injuries were compared with the non-penetrating (Table IV).

When the series of patients is re-examined from the point of view of age of onset of habitual seizures rather than the time after brain insult, Table V is produced. This shows that, of the 73 patients whose seizures began between ages 10 and 20, for example, the cause was about equally distributed among birth, postnatal trauma and other causes.

TABLE V.

AGE OF ONSET

ONSET AT AGE	0-2 yrs.	2-10	10-20	20-35	35-55
No. of cases	31	66	73	50	13
Due to birth lesion	58%	46%	34%	12%	31%
To postnatal trauma	13%	26%	37%	60%	54%
To miscellaneous	29%	28%	29%	28%	16%

TABLE VI.

CRANIOTOMIES FOR FOCAL EPILEPSY

Cases during 6-year period 1945-1950		234
Cortical excision, patients	219	
Negative explorations, patients	15	
Number of operations		263
Deaths		4
Case mortality		1.7%
Operation mortality		1.5%
Adequate follow-up		217

H. RESULTS OF OPERATION

Table VI enumerates the patients, the operations and the follow-up records to be included in the analysis of therapeutic results.

Among the 234 patients operated upon during the period 1945 to 1950 inclusive, 203 had one operation only in this time for the purpose of relieving epileptic seizures, 29 patients had two operations, one had three and one had four. Thus a total of 268 craniotomies were carried out with the intention of excising an epileptogenic focus. This does not include any operations for postoperative hæmorrhage, infection or repair of cranial defects. One operation performed in January 1951 is included, as this was the second on a patient during one admission, the first having been performed in December 1950.

Four of the patients had had a previous operation for epilepsy in the Montreal Neurological Institute and returned here during the period under study for a further procedure. One of these patients died postoperatively and the other three benefited only slightly by the second operation. All are included in the analysis.

Each of six patients had had one operation to relieve his epileptic seizures at another hospital before being seen at this Institute. Seizures had recurred, and the patients were seeking advice again (forever hopeful!). Only one of these was successfully relieved of the attacks, three were improved and the other two were unchanged. These also are included in the analysis.

Eleven patients who were operated upon for the first time between 1945 and 1950 have since returned, after 1950, for a second operation. The follow-up results in regard to these patients are accepted, as they were assessed before and not after the second operation.

Multiple operations during the six-year period under study, irrespective of the time interval between the craniotomies, have been analyzed as two-stage procedures and are thus only represented once for each patient.

There were four deaths in hospital after operation, so that the case mortality was 1.7% and the operative mortality 1.5%.

Table VI shows 219 patients who had excision of cerebral cortex that was considered to be epileptogenic and 15 craniotomies without excision. In the first group, 203 patients could be followed up satisfactorily and in the second, 14. In 11 cases there was inadequate follow-up, some have not been heard from since discharge from hospital, and the remainder were known to have had no seizures after operation during the short time that information was available, but since they could not be reached at the time of this final analysis they are not included here. Two patients died after discharge, one in a few weeks, the other in four months.

Assessment of results is possible, therefore, in 217 cases, 203 in which craniotomy was carried out and 14 in which exploration was negative.

TABLE VII.

POSTOPERATIVE SEIZURE RESULTS

Group				
4.	Perfect result	57	(28%)	Success — 45%
3.	Nearly perfect result	34	(17%)	
2.	Satisfactory result	40	(20%)	Good — 20%
1.	Slight improvement	37	(18%)	Failure — 35%
0.	No improvement	35	(17%)	
Total cases followed up		203		

1. *Cortical Excisions*

The results of these excisions are outlined in Table VII. It should be borne in mind that these patients had all received what was considered adequate trial of the best medical therapy without reasonable success. The estimates of results, made after complete study by one of us (K.P.), are expressed in a form comparable to that used in the earlier case follow-up studies from this clinic.

For the purposes of this study, we have defined a seizure as an episode with one or more of the following features: ictal movement, lapse of consciousness or lapse of memory. Minor ictal sensations alone are not counted and neither are

alterations in consciousness or memory so brief as to pass unobserved by persons about the patient. These exclusions, which apply to a very few patients, seem reasonable since the surgical objective is to reduce the patients' social and economic handicap. The exact incidence of such subjective episodes, not included as seizures, will be described later.

Each of the five follow-up groups shown in Table VII may be defined as follows.

Group 4: No seizures since discharge from hospital.

Group 3: Not more than three seizures since discharge from hospital. Patients followed up for less than three years after operation must have no more than one attack a year if they are to be included in this group. Many of the patients in this group had one or two attacks shortly after discharge and subsequently none for the three, four, or five years up to the time of analysis. In such cases the result is gratifying and the patient probably considers himself cured, but we have not scored his result as perfect.

Group 2: A patient who is having six (or less) major seizures a year or six (or less) attacks of automatism, or less than 12 minor motor seizures a year, is classified in this group, provided this constitutes a significant improvement over the preoperative level of frequency.

Group 1: Seizures definitely less frequent or less severe than before operation.

Group 0: Little or no improvement in frequency or severity of seizures; in some cases worse than before operation.

In the discussion of results, group 4 will be called perfect, group 3 almost perfect and the two together will be rated as successful results. In group 2 the results may be considered satisfactory, but for groups 1 and 0 the operation will be considered to have failed from the point of view of control of seizures.

Medication.—It has been our invariable practice to forbid the use of all anticonvulsive drugs following operation, except phenobarbital. This drug is continued at 2 to 3 grains a day for adults. The tendency to postoperative attacks seems to decrease with the passage of time, and the patients in groups 3 and 4 usually stop all medication within a year or two.

Patients in lower success groups may find that the effectiveness of dilantin has been greatly increased by operation. They may consider this a most welcome change. Nevertheless any patient who feels that he must take dilantin after operation is recorded as a surgical failure.

In general, no patient has been accepted for operation in the first place until he has had extended preoperative trial of medication including adequate doses of phenobarbital and dilantin.

In conclusion, therefore, there are 57 patients who have had no attacks and 34 who had not over three attacks before apparent cessation of seizures.* If groups 4 and 3 are combined, operative therapy may be called a "success" in 45% of the total. In addition to this, in 20% of the cases there was what may be called a satisfactory result.

2. *Negative Explorations*

In 15 cases craniotomy was carried out but no cortical excision was deemed advisable. Nothing was done in addition to exploration except perhaps separation of adhesions or subtemporal decompression.

The records of these patients may serve as a control. None had a successful result. Of the 14 who could be followed up, none was in group 4 or 3, two were in group 2, two in group 1 and 10 in group 0.

The length of follow-up in this negative series was 6 years in one, 5 years in four, 4 years in three, 3 years in one, 2 years in four and 1 year in one.

In the series of cases previously published by Penfield and Erickson (1941) one patient was placed in group 4 as the result of negative exploration.† A second patient was placed in group 3, seven patients were put in group 2 and 26 in group 0.

In the series published by Penfield and Steelman (1947), no case of negative exploration was placed in group 4 or 3, but five were placed in group 2, two in group 1 and nine in group 0.

Thus it is seen that as the result of 65 negative explorations in the three series (470 patients in all), only two patients could be placed in the success groups (4 and 3). The cure of these two patients in a consecutive series of 65 negative craniotomies can hardly be considered as more than a chance variation.

3. *Partial Temporal Lobectomy*

It may be of interest to separate from the total 234 patients the 68 who were suffering from

*There were four patients who, because of excellent performance in subsequent years, were finally raised to group 3 from group 2. They did have more than three early seizures.

†This was a young woman with temporal lobe epilepsy (M.G.). She was thought at the time of operation to have a deep temporal neoplasm and a subtemporal decompression was carried out. She has had no attacks in the 17 years since operation and shown no evidence of tumour growth.

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temporal lobe epilepsy and who were treated by partial temporal lobectomy in this period.*

For the sake of brevity we will divide them into two groups: (1) Successful (patients who have had no more than three attacks in the first three years after operation and most of them no attacks). (2) The rest we may call unsuccessful, although many in this group were much benefited.

Using this definition, 32 patients or 47% had successful results and 35 unsuccessful. The follow-up period varies from one to more than seven years; in 32 of the patients it is more than three years.

The results show that recurrence of seizures is most likely in the first six months after operation, and that for this group none recurred after three years. Of course, we realize that recurrence is possible at any time. Considering only the patients followed up for over three years, 16 or exactly one-half had a successful result.

Eighteen patients had an upper quadrantic field defect after operation, but in none was it disabling. Temporary aphasia occurred in 11 patients but persisted in only one. Twenty patients complained of memory defect, but this was severe and disabling only in one.

The pathological lesions discovered at operation were: atrophic lesions with or without cysts in 38 cases, microgyri in six, meningocerebral cicatrices in four, angioma in one, hæmangioma calcificans in four. In 13 cases the only abnormality noted was increased consistency of the gray matter deep in the temporal lobe.

I. Comparison with Earlier Series and Factors that Influence Results

We may now return to a reconsideration of Table VII which shows the results of cortical excision in the 203 patients of this series. It will be seen that there are 57 patients with perfect results (28% of the total), and this should be compared with the earlier figures of 24 (21%) of 115 patients (Penfield and Erickson) and 15 (25%) of 59 patients (Penfield and Steelman). There appears to be a slight improvement in the number of perfect results with each successive series. In regard to the other follow-up groups a more severe standard of selection is set in the present series as compared with the earlier ones.

In this present series, 34 patients achieved a nearly perfect result (group 3), only 17% of the total as compared with 25 patients (22%) of the first report and 18 (30.5%) of the second. At first glance this would appear to be less satisfactory in the latest series, but the classifications of group 3 in the three series are not strictly comparable. In the first study, a few patients with one or two seizures a year were included in group 3 and in the second a 75% improvement was classed as group 3. This might include patients who were having one or two attacks a year and who would in this series be classed in group 2, and also include those who had been free of attacks for three, four or five years up to the time of follow-up, but who might have had a sufficient number of seizures in the first year or two after operation to exclude them from group 3 by the standards adopted for the present analysis.

In this series, 37 patients (18%) derived slight benefit from operation, and this can be contrasted with 11 (9.6%) of the first and 7 (12%) of the second series. Those patients who were not benefited by operation or were made worse (very few of the latter) numbered 35 (17%), whereas in earlier operations 25 (22%) and 11 (18.6%) had this unsatisfactory outcome.

Group 2 patients in this present series are even less comparable with those of the earlier reports. Forty patients have had a satisfactory result after operation, placing them in group 2—that is, 20%. If one could guarantee a group 2 result, as graded in this study, to every patient, cortical excision would be considered a useful procedure.

The higher incidence of perfect results in the last series suggests that we are deriving benefit from longer experience, although the incidence of failures remains about the same. The chief changes are the ever-greater reliance placed upon electrocorticography during operation and the great increase in temporal lobe cases.

The percentage of patients with successful or satisfactory results in this series has remained just below 70% over the years, despite the wide extension of operation year by year to cases previously considered unsuitable for surgery. Complete freedom from attacks (see Table X below) applies to almost half of the patients when considered year by year.

Thus there are patients, in groups 3 and 2, who have had no attacks for a certain period of years up to the time of their follow-up. Fifteen patients in group 3 have had no seizures in the last one to three years and three patients included in group 2 have had no attacks in that period. Phenobarbital was the only medication used by 11 of these patients, three are taking anticonvulsants other than phenobarbital and

*Some of these patients were reported by Penfield and Flanigin (1950) but others are added and the follow-up period is extended. There were 19 other patients who had temporal removals in addition to excision elsewhere. They are excluded. Since 1950 the proportion of temporal lobe cases has steadily increased so that they now compose well over 50% of the total patients who come to operation.

three are taking no drugs. In one case, the medication is unknown.

In addition to the above, seven patients in group 3 and one patient in group 2 have had no seizures for more than three years up to the time of follow-up. Five of these are taking no medication, one phenobarbital only and the other two anticonvulsants other than phenobarbital.

In females the rate of success was higher than in males, and this was especially so in those patients with lesions due to birth or postnatal trauma.

At follow-up, 48% of patients with epilepsy following a birth lesion were classed in group 3 or 4, but only 39% of those with post-traumatic seizures. The remaining etiological subdivisions are too small for comparisons.

Those patients whose seizures had been present for less than two years before operation had a better success rate—54%—than the remainder—only 44%. However, it is interesting that of the patients (24 in number) who had their seizures for over 20 years, 46% could be said to have had a successful result.

Well-localized electroencephalographic foci, with or without transmission of the abnormal wave forms to the opposite side, and indicative of a localized epileptogenic lesion which could be excised, promised a better prognosis—47% in groups 3 and 4—than were bilateral abnormal discharges with uncertain localization—only 33% success.

Excision of the frontal area seemed to be more favourable—54% success—than other excisions, the worst being perisylvian and parietal with only 27% of patients placed in successful groups after excisions in these regions.

Early postoperative seizures are of bad prognostic import, whether the seizures be similar to the preoperative attacks or not. The electroencephalographic recording three or more weeks after operation is a comparatively good guide to the ultimate result.

J. The Patient's Assessment

The patient's own opinion of the operation is worth recording briefly. All patients in group 4 were satisfied, but three in group 3 believed themselves worse off because of some physical defect following operation. Even in group 0, there were six grateful patients. Altogether there were 40 patients who thought themselves worse after operation, out of a total of 150 who expressed their opinion in this regard.

There were 125 patients, each of whom expressed his assessment of the result of operation in percentages at the time of this survey. This personal assessment is shown in Table VIII. Many elements are, of course, included in this voluntary expression of opinion—disabilities of various types, emotional sequelæ and, most important, freedom from attacks.

TABLE VIII.

Patient's Assessment of Result		
"100% cured"	39	31%
"Over 75% improved"	25	20%
"Over 50% improved"	37	30%
"Over 25% improved"	1	1%
Not improved	23	18%
Total who gave opinion	125	

K. Multiple Operations

When the result of cortical excision is not satisfactory a second procedure may be carried out in selected cases to extend the removal in one direction or another. Occasionally, a failure may thus be converted into a brilliant success.

During the years of our analysis, 27 of the patients adequately followed up were operated upon twice.* Nine of them were converted from failures to group 4 successes and two became group 3 successes. Thus 41% of the 27 patients reached the successful groups and another 19% were in the satisfactory group (2). Within this series a second operation proved to be worthwhile in 60% of the patients thus treated.

The epileptic patient should be interested in the control of attacks throughout his life span. In our experience there is apt to be little change in the result of operation after a three to five-year postoperative period, although there may be steady improvement far beyond that period, as we have learned from our early cases.

In Table IX the results for the 51 patients in this series, whose follow-up is longer than five years, are set out according to our method of group tabulation. It is seen that 41% of the patients are in successful groups five to seven years after operation. But this actually gives an erroneous impression because, according to our

*This does not include the operative deaths. One such patient had had 4 operations. Two had 2 operations. There was another patient who had 2 operations but is excluded because he could not be followed up.

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TABLE IX.

RESULTS IN 51 PATIENTS FOLLOWED UP FOR 5 TO 7 YEARS

Group 4.	Perfect result.....	12	(23%)	Success 41%
Group 3.	Nearly perfect result	9	(18%)	
Group 2.	Satisfactory result..	18	(35%)	Good 35%
Group 1.	Slight improvement	6	(12%)	Failure 24%
Group 0.	No improvement...	6	(12%)	

method of recording, a patient who does badly for a few months can never get into the successful groups, even if his attacks should stop completely.

When the whole group is considered it is recognized that some patients drop out of the success groups because of some recurrence, perhaps transient, but others become seizure-free in later years, making for a compensating balance.

When each of the years that follow operative excision is taken by itself without regard to other years, it appears that there is a reasonably steady quota of patients who are completely seizure-free. Table X shows that this quota varies between 40% in the first postoperative year and 48% in the sixth.

TABLE X.

FREEDOM FROM SEIZURES YEAR BY YEAR

Year after excision	*Total patients followed up*	*Complete freedom from attacks*
1st	203	80 — 40%
2nd	173	77 — 45%
3rd	124	50 — 41%
4th	86	35 — 41%
5th	51	21 — 42%
6th	25	12 — 48%
7th	11	5 — 45%

L. EFFECTS OF EXCISION ON FUNCTION AND EFFICIENCY

It is not enough to know whether a radical surgical procedure has stopped attacks or not. We must know its effect upon the patient's ability to work, to hold a job, to study; the effect on physical and mental function, the effect on behaviour and on the happiness of patient and friends. When all the features of his life are considered, it still remains for the physician to ask the final question: In the opinion of the patient and of those who love him, was the operation a success or a failure? This is answered in part by Table VIII, but we may examine the question in more detail.

ECONOMIC RESULTS

An economic analysis is not easy. Before operation 79 out of the 203 patients were fully occupied in spite of seizures. Not infrequently it was the threat of loss of employment that brought the patient to seek operation. Five of them are not now employed and two are in mental or epileptic homes. On the other hand, 51 not previously employed are now on a satisfactory full-time work or educational schedule. There are 38 patients out of work but possibly employable when last heard from.

PHYSICAL EFFECTS

The child who is hemiplegic, epileptic and progressively more and more retarded mentally seems to have a great deal to gain and little to lose. If the attacks are stopped, mental progress and behaviour improved and spasticity of the paralyzed extremity lessened, then it is obvious that everything is achieved that could be hoped for. But if only one of these things is accomplished, if the attacks stop and the child does better in school, the parents may call the procedure a splendid success although the paralysis is unaltered.

If a man who is physically and mentally normal has his seizures stopped but forfeits the use of an arm so that he cannot work or develops a serious defect in memory, he may consider the procedure a failure. On the other hand, there are other bodily disabilities he would gladly accept to escape from the curse of seizures, for example a hemianopic defect or a cortical somatic sensory loss.

Clinical cases are personal as much as they are scientific problems, and the clinician must often make the best compromise with perfection that he can. He must understand the patient and his hopes before he presumes to decide on treatment. But this is, after all, the secret of the art in the practice of medicine.

Frequency of attacks after operation is obviously not the only measure of success although it is usually the most important.

Hemiplegia or hemiparesis, aphasia and memory loss are the serious defects that a neurosurgeon fears as a sequel to cortical removal or the end result of some untoward complication of operation. Cortical sensory loss alone or a de-

fect in the homonymous visual field is usually considered an unimportant deterrent when there is a reasonable chance of stopping seizures.

Hemiplegia or hemiparesis as an untoward complication may result from manipulation of the middle cerebral vessels, especially when scarred cortex is removed above the fissure of Sylvius as well as below it. It may be difficult to decide whether this has produced arterial thrombosis or simple spasm. There is also some danger of trauma to the internal capsule during the process of removal of the cortex of the insula or the cortex deep within the fissure of Sylvius. Then there is, of course, always the danger of hemiplegia that may be caused by postoperative bleeding unless reopening is carried out in time. But this is a complication that all neurosurgeons must guard against after every craniotomy.

Out of 159 patients who had normal or almost normal motor and sensory function before operation 15 were found to have somatic motor or sensory abnormalities at the time of follow-up assessment. Six of these abnormalities were considered to be severe. Two were hemiplegias. In all, 26 patients had some degree of motor or sensory disability not present before operation, often minimal and usually anticipated. In all but five the excision was carried out in the central area.

On the other hand, in our analysis of these cases there would seem to be eight patients whose paralysis or weakness was improved by operation. Six were converted from a severe to a moderate defect and two from a moderate defect to almost normal.

Among these eight patients who claimed improvement in somatic motor function, four had removals of part of the central or Rolandic cortex, two had temporal lobectomies, one had intermediate frontal (supplementary motor) removal and one had extensive removal of central, parietal and temporal cortex.

This improvement seems, at first sight, difficult to understand. In some cases there was "paradoxical improvement" in the existing hemiplegia (as described by Welch and Penfield, 1950) resulting from reduction in spasticity when a useless precentral gyrus was removed. In others it seems possible that there was great weakness secondary to a bout of seizures before operation and that this weakness may have cleared up or improved as the result of cessation of seizures.

CHANGES IN VISUAL FIELDS

Thirty-two patients developed a visual field defect as a result of operation, or a pre-existing defect was increased by operation. As one would expect, these followed excisions in the posterior temporal and occipital regions. Temporary lobectomy frequently gave an upper quadrantic defect, but the patient did not report this as a disability. The only complete hemianopic defects were produced by occipital lobectomy.

Temporal lobectomy was followed by a field defect (upper quadrant anopia) in 18 patients, and it is of interest to correlate the extent of lobe removal with the visual defect. The surgeon usually measured the length of the amputation from the anterior pole of the lobe, so that figures are available. The figures must be taken with some reservation, of course, as it is the injury to the deeper optic radiation which is important and not the extent of superficial cortical excision.

Excision of the anterior 4 cm. of the lobe produced a defect in only one patient out of 14. When the removal amounted to 5 cm. or a little more, 5 out of 20 had postoperative field defects. Four out of 12 had defects if 6 to 7 cm. were removed, and 3 out of 8 when the ablation extended 7 to 8 cm. All three of those with more than 8 cm. of the temporal lobe removed had a field defect. In two cases the extent of lobectomy is not known.

It must be admitted that completely accurate visual field examinations were not carried out on all patients of this series, but more detailed recent studies have provided interesting results which will be published eventually.

ASPHASIA

After cortical excision carried out in the dominant hemisphere, aphasia made its appearance frequently during the postoperative period, at the usual time for neuroparalytic œdema, about the 3rd to the 5th day after operation. It cleared up about the 10th to the 14th day. Such transient effects will not be referred to again.

In all cases, excisions of this type were carried out under local anæsthesia so that the patient could continue to talk. When no aphasia appeared while the patient was on the operating

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table, its subsequent appearance proved to be of little moment.*

Aphasia was present when the patient was discharged from hospital (14th to 21st day) in the case of 15 patients, and it persisted as a disability in only three patients.

It is possible that the remaining 12 patients discharged with a definite aphasia would show some changes at the present time if they were available for careful examination, but they themselves and their relatives do not recognize any difficulty in speech, nor is there any complaint of defect in reading or writing.

Two of the patients were lefthanded but all had speech localization in the left hemisphere. Eleven of the 15 speech defects were produced by temporal removals as follows: one after a 4 cm. removal, two after 4 to 5 cm. removal, seven after more than 5 cm. removal. In the 11th case the extent of removal was not accurately recorded. In three patients the excision was in the left lower intermediate frontal region, and the remaining excision was in the left lower centro-parietal junction.

INTELLIGENCE

No special study of the mental state before and after operation was made. But some idea of mental change may be had from the clinical notes. Psychological and psychiatric studies are in progress now and will be published. Memory loss was discussed above under the heading of Partial Temporal Lobectomy.

It would appear that, in all, 24 patients have deteriorated since cortical excision. None of them was in the success group 4 and only three were in the success group 3. Thus the intellectual deterioration occurred largely in patients who were not relieved of their seizures and one may surmise that it was associated with the continuing seizure process.

M. SUMMARY NOTE

This is a report of the results of the surgical treatment of 234 patients who had focal cerebral seizures together with a discussion of the reasons for success and failure. This series includes all such patients operated upon in the six-year period that closed in 1951, excluding cases of neoplasm. When added to the two preceding follow-up studies, 470 such cases may now be reconsidered.

Before operation none of these patients had been able to lead happy, efficient lives, even with the help of the best regimens of conservative therapy. The procedure of cortical excision, which was recommended in the first place as a counsel of despair, has come to be applied to patients in a rapidly expanding field. Mechanisms of cause and techniques of study and treatment are described and the chances of success evaluated.

For three-quarters of the patients the operation proves to be a real benefit, and half of the patients continue to be completely free of attacks year by year, at least as far as our analysis goes, into the seventh postoperative year.

Epileptogenic cortex is nociferous or harmful cortex because of its general influence on brain function. Successful removal, when this is possible, may result in better intellectual performance and more normal behaviour as well as in freedom from seizures. There are the dangers of operation and the risks of functional deficits to be considered. But in spite of this the treatment of focal epilepsy by surgery is now established as a legitimate form of therapy.

Success demands special training, and the neurosurgeon must have clinical and scientific co-operation in a well-equipped hospital. But there is no field in medicine where the rewards are greater in terms of human happiness and efficiency.

*A method of speech localization exploration has been used which will be reported in detail with our associate Dr. Lamar Roberts. A stimulating electrode is applied to the cortex to produce local interference with function. If aphasia appears during stimulation, and if there is no evidence of spreading after-discharge when the electrode is removed, the convolution stimulated is considered essential to speech and is carefully avoided during excision unless there is other strong evidence in favour of its removal.

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RÉSUMÉ

Le but de cette communication est d'évaluer le traitement de l'épilepsie par les excisions corticales. La nature même de l'irritation qui rend la matière grise épileptogène est encore inconnue. D'après Hughlings Jackson, chaque crise épileptique commence par une décharge neuronale de la formation grise. Si, théoriquement, cette aire est enlevée, les crises devraient cesser. Cependant, la difficulté consiste à identifier et à enlever tout le cortex épileptogène, mais non plus. Le foyer est l'endroit où les potentiels électriques sont les plus élevés entre les attaques; cependant, le cortex epilepto-

gène peut être beaucoup plus vaste que ces indications nous permettraient de le soupçonner. Ce foyer peut être localisé cliniquement d'après la manifestation initiale de la crise, ou à l'aide de l'électro-encephalographie. A l'opération, on peut aussi quelquefois reconnaître le cortex anormal à la simple inspection. La stimulation électrique du cerveau même peut reproduire la manifestation initiale de la crise. Les lésions pathologiques peuvent être une tumeur, un kyste, une zone de destruction résultant d'une hémorragie, d'une thrombose, ou d'un traumatisme produit à la naissance, lésant le cortex adjacent. L'électro-corticographie est la baguette de coudrier du neuro-chirurgien à la découverte de zones épileptogènes. L'entité pathologique la plus fréquemment vue est la "sclérose par l'incisure" de l'hippocampe et de l'uncus (vide infra). La dureté du cortex épileptogène proviendrait du changement des astrocytes protoplasmiques en astrocytes fibreux et de la prolifération de ces cellules. Une particularité commune à toutes ces lésions semble être une insuffisance de la circulation, minime mais persistante. L'importance de ce désordre n'est pas encore bien établie. Un trouble du métabolisme de l'acétylcholine par le tissu épileptogène a été incriminé par Elliott, et paraît relié à certains degrés d'anoxie.

L'auteur présente une série de 234 malades ayant subi une craniotomie pour le traitement de l'épilepsie, entre 1945 et 1950. Aucun d'entre eux ne présentait de tumeur intra-cranienne. Tous les malades ont été suivis pendant au moins un an; certains d'entre eux l'ont été pendant 7 ans. Cent quatre-vingt-dix d'entre eux répondirent à un questionnaire écrit; bon nombre de leurs proches parents y ajoutèrent leurs propres commentaires.

Les résultats montrèrent une distribution pour les hommes de 65%, et de 35% pour les femmes. Cet excédent ne s'applique pas seulement à l'épilepsie post-traumatique mais bien aussi aux accidents de la naissance et à l'anoxie néo-natale. Dans 4.4% des cas, il fut possible de trouver des antécédents familiaux de désordres convulsifs. Les tendances héréditaires peuvent donc être laissées de côté. Sous le titre d'accidents de la naissance furent réunis les traumatismes par instrumentation, les hémorragies intra-craniennes, l'anoxie cérébrale et l'herniation du cortex temporal par l'ouverture de la tente. La "sclérose par l'incisure" est décrite par l'auteur comme étant la lésion produite par la compression temporaire des artères qui franchissent l'échancrure de la tente pour irriguer l'uncus, l'hippocampe et le cortex adjacent, par l'engagement des circonvolutions temporales vers la fosse postérieure. Les convulsions produites dans un état fébrile ne jettent aucune lumière sur la nature de la lésion cérébrale puisque la fièvre ne peut servir qu'à démasquer la présence d'une lésion atrophique, préalablement occulte. Les crises du lobe temporal commencement le plus souvent entre les âges de 5 et 20 ans. Les premiers-nés se retrouvent plus souvent dans les malades souffrant d'épilepsie focale causée par un accident de la naissance que le reste de la population en général. Dans les cas douteux de lésion traumatique post-natale, la présence d'hémiparésie, de déficit sensoriel ou d'aphasie datant du temps de la blessure, fut prise comme preuve de lesions localisées du cerveau. Le tableau no. 4 montre que l'intervalle entre le traumatisme post-natal et le début des crises habituelles est d'environ un an après les blessures pénétrantes de la tête et d'un peu moins d'un an pour les blessures non-pénétrantes. Dans les lésions intra-craniennes suppurées, les crises apparurent entre 2 et 13 ans après la phase aiguë de l'infection.

Les sept angiomes de la série furent tous excisés, ainsi que les circonvolutions adjacentes, sauf un de la région centrale droite dont l'enlèvement aurait pu causer une grave hémiplégie. La dégénérescence des hémangiomes calcifiants est prononcée et leur vascularité est beaucoup diminuée. Les 4 cas de cette série se trouvaient dans le lobe temporal.

Sous l'en-tête "Divers" furent réunies 18 lésions disparates telles que: l'ischémie post-natale, l'encéphalite du plomb, la malaria cérébrale, la sclérose tubéreuse de Bourneville, l'urémie, les encéphalites (probablement à virus), la maladie hémorragique du nouveau-né, les possibilités de tumeur cérébrale, les méningites, et l'artériosclérose.

L'auteur désire attirer l'attention sur le délai considérable dans le début des crises répétées causées par les lésions temporales datant de la naissance. Chez les 73 malades dont les crises commencèrent entre les âges de 10 et 20 ans, la cause se retrouve indifféremment entre le traumatisme de la naissance, le traumatisme post-natal, etc. Pour servir aux fins de cette étude, l'attaque d'épilepsie devait comprendre au moins une des conditions suivantes: mouvements convulsifs, perte de connaissance ou perte de mémoire. Aucun des malades ne reçut de médication anti-convulsive après l'opération, sauf du phéno-barbital. Ceux qui durent recourir à la phénytoïne (dilantin) furent considérés comme des échecs chirurgicaux. Des 203 cas ayant subi une craniotomie et qui purent être suivis par après, 57 n'eurent plus jamais d'attaque et 34 n'eurent que trois attaques ou moins avant que les crises ne disparaissent. L'intervention peut donc être considérée comme un succès dans 45% des cas. Des résultats satisfaisants furent obtenus dans un autre 20%. Des 68 malades ayant subi une lobectomie temporale partielle, 47% bénéficièrent d'un bon résultat. La comparaison avec des séries semblables publiées antérieurement montre une proportion croissante de résultats excellents que l'on peut attribuer à l'expérience acquise. Deux facteurs nouveaux sont cependant apparus: une plus grande fréquence des lésions du lobe temporal et une plus grande importance attachée à l'électro-corticographie. Les bons résultats furent plus nombreux chez les femmes que les hommes. Cette différence a été particulièrement marquée dans les cas de lésions relevant de traumatismes natals et post-natals. La reprise des attaques tôt après l'opération est de mauvais augure. Trois semaines ou plus après l'intervention, l'électro-encéphalographie offre un indice assez satisfaisant des résultats à venir. Lorsque ceux-ci ne sont pas satisfaisants, une deuxième intervention peut être pratiquée dans des cas choisis. Un certain nombre d'échecs devinrent ainsi de brillants succès et, dans 60% des cas, cette deuxième opération s'avéra utile.

Au point de vue économique, 51 malades sont maintenant à l'ouvrage et 38 autres sont en mesure de l'être. De 159 malades ayant une fonction motrice ou sensorielle normale ou quasi-normale avant l'opération, 15 subirent une perte motrice ou sensorielle, 6 d'entre eux à un degré marqué. Par contre, la paralysie ou la faiblesse pré-opératoire de 8 malades fut améliorée par l'intervention. Les défectuosités du champ visuel, résultant de l'opération, n'ont pas semblé nuire aux sujets qui en furent atteints. Une aphasie passagère s'est souvent manifestée vers le 3e ou 5e jour après une intervention intéressant l'hémisphère dominant; deux semaines plus tard, elle était disparue dans la plupart des cas. Quinze malades présentaient encore de l'aphasie a leur départ de l'hôpital; elle ne persista que chez trois d'entre eux. Une détérioration mentale s'est produite chez 24 malades ayant obtenu peu ou pas d'amélioration de leur opération. Il est possible que cette détérioration soit en rapport avec la persistance des attaques. M.R.D.

MEDICAL PRACTICE IN MANITOBA

The Students' Guide number of the *Lancet* (August 27) contains a nice piece of Canadian enterprise in the form of an article by Drs. McLandress, Martin and Briggs of Winnipeg, describing medical practice in Canada with special reference to pædiatrics in Manitoba.