Significance of surgery for temporal lobe epilepsy in childhood and adolescence

MURRAY A. FALCONER, M.CH., F.R.C.S., F.R.A.C.S.
Director, The Neurosurgical Unit of Guy's, Maudsley, and King's College Hospitals, London, England

The problem of childhood temporal lobe epilepsy is reviewed and illustrated from three cases in which the patients were freed from fits by temporal lobectomy. The pathological lesion (mesial temporal sclerosis) is discussed and the likelihood that many adult cases have gone unrecognized in childhood is emphasized.

PENFIELD and Flanigin,45 with their report in 1950 of some 68 patients submitted to operation, are acknowledged as the pioneers of the surgical treatment of temporal lobe epilepsy. Although they did not expressly say so, their patients were clearly all adults.

The fact that temporal lobe epilepsy commonly originates in childhood has only recently been recognized,1,4,16,40 and little has been written about its surgical treatment in children. In this paper I will survey the subject, indicating how the condition may present itself in childhood and emphasizing the possibilities of surgical treatment if the epilepsy should become intractable or be associated with disabling, behavioral, or educational disorders.

The Problem

By definition, temporal lobe epilepsy means epilepsy originating within one or both temporal lobes.9,13,23,44 This definition is important because psychomotor seizures can arise in other parts of the limbic system, such as the inferior frontal and cingulate regions, although they occur most often with temporal lobe lesions.18,23,28,49,50 Temporal lobe epilepsy appears difficult to recognize in childhood. My own experience of anterior temporal lobectomy has been mainly with adults, but most of them have been epileptic since the first decade of life.20,23,54 From time to time I have stressed the importance of earlier diagnosis, pointing out that had I been able to operate on these patients while they were still in their school years, and presuming that operation would have achieved the same beneficial results in seizure relief and social adaptation as it did in adult life, much physical and personal suffering would have been spared them and their families.14-16,20 When one looks back at the old records of these patients from childhood on, one realizes all too well the difficulties of diagnosis at an early age.11 The following case history viewed in retrospect illustrates these points.

Case 1

A 16-year-old boy, small for his age, was referred by D. W. Liddell, M.D., F.R.C.P., and G. W. Fenton, M.R.C.P., with intractable epilepsy and uncontrollable aggression. All his life he had attended the pediatric departments of various London hospitals for manifestations of epilepsy. There was no family history of this disease. His birth had been a difficult one, requiring resuscitation;
his birth weight had been 2.8 kg. At the age of 5 months he had some febrile convulsive attacks. An electroencephalogram (EEG) was reported as an “unsatisfactory record,” but was not repeated. However, the child was lively and active and passed the milestones of childhood at the normal times. At 4 years of age he had “a non-febrile convolution.” A subsequent EEG was again reported as normal.

When he began school at 5 years of age he was reported as backward; his behavior was often difficult and his attendance irregular. When 7 years old he witnessed the death of his brother by drowning. Four months later he began to show frequent “epileptic fits” and episodes of uncontrollable behavior. The fits were apparently major convulsions, with incontinence of urine and feces; they often occurred daily in spite of anticonvulsant drugs.

When 8 years old the patient was admitted to a hospital for 3 weeks of observation. The EEG was once more normal. The hospital records stated: “No fits witnessed, but he would sometimes fall to the ground, close his eyes, and twitch his limbs, then look under his lids to see if he was being watched.” A psychiatrist wrote: “Hyperkinetic and anxious, ruminating on the death of his brother.” He was discharged on anticonvulsants. His “fits” and rages continued, and he had to be sent to a special school for disturbed children.

When 10 years old the patient was investigated in another children’s psychiatric department. The verbal I.Q. was 81, and performance I.Q. 86 (WISC). His behavior improved after phenobarbitone was withdrawn, and he remained only on phenytoin. In interviews he described “a cold feeling in the stomach before an attack.” The EEG was again reported as normal (Fig. 1). Serious doubts were entertained about the genuineness of his epilepsy. At that time his seizures were described as characterized by sudden falls to the ground with slight frothing of the mouth and a slight change of color. Their duration was only 1 to 2 seconds, and they occurred about twice a month. It was noted that he had never hurt himself in an attack.

When he was 15 years old he was referred to the adult department of the Maudsley Hospital. His attacks were now more clear-cut. All began with an epigastric aura, and in the more severe ones convulsive movements of the left limbs were noted for a few minutes followed by a period of sleep for up to half an hour. In the less severe attacks he simply lay still for 2 to 3 minutes. He averaged at least one attack a week. There had also been many episodes of sudden severe rage, including one which took four policemen to subdue him. On account of his aggression he was under a Court Order and living in a special hostel, for his parents could not cope with him and he was unemployable. Various drug regimes had proved ineffective.

Neurological examination was normal.
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Routine EEG’s now disclosed a spike focus in the left temporal lobe (Figs. 2 and 3), while sphenoidal lead studies under thiopentone narcosis disclosed spike discharges originating in the left inferior temporal region (Fig. 4). Skull films were normal, but the pneumoencephalograms showed that the left temporal horn was slightly smaller than the right. The I.Q.’s were FS 86, VS 90, PS 83 (Wechsler).

A left anterior temporal lobectomy was performed in June, 1968, revealing mesial temporal sclerosis. Convalescence was smooth, and the patient acquired a slight right upper quadrantic homonymous hemianopia but no other evident sequel to his operation.

From that time onward he has been fit-free and has exhibited an entirely different personality. The aggression has gone, and he is now at home living happily with his parents, who have removed him from the Court’s jurisdiction. He has been working in light employment. Several postoperative EEG’s, including sleep records, are normal.

The follow-up period at the time of writing is admittedly only 16 months. One cannot help wondering, however, if the correct diagnosis had been made earlier and the operation undertaken, whether this patient, his parents, and the State would have been saved much trouble and distress.

Difficulties of Diagnosis in Childhood

The first point to remember is that epilepsy in infants is common, and that its incidence decreases as adolescence approaches. In other words, most epileptic children appear to “grow out of their epilepsy.” Thus, Cooper, in following from birth to 15 years of age 5000 children of all social classes born in Great Britain in the year 1946, found a total incidence of about 25 infants per 1000 with some epileptic manifestations during the first 2 years of life, and possibly 30 per 1000 during the first 5 years of life.

Fig. 2. Case 1. Tracing taken with patient awake at age 15 years, is normal at the beginning of the record. A sharp wave with pulse reversal then appears between Channels 5 and 7, and simultaneously appears in phase in Channels 11 and 12. It is followed by slow activity in the left temporal area and possibly both front areas.
Another comparable study also from Britain reports 40 infants per 1000 with epileptic manifestations during the first 5 years of life. Cooper also showed that the incidence of fits decreased steadily as the children grew older. Indeed, 218 children per 1000 experienced their one and only fit after the age of 2 years, and of the 26 children in his total series (approximately 5 per 1000) who had continued fits after the age of 2 years (the chronic epileptic group), many had not had their initial seizure until after infancy. From the statistics in these studies, it may be assumed that the incidence of epilepsy in the population of Great Britain as a whole is 4 to 6 persons per 1000. The study of Kurland regarding the prevalence of epilepsy in different age groups of the resident population of Rochester, Minnesota, is not relevant to these points, for he deliberately excluded febrile convulsions.

Cooper made no attempt to ascertain what proportion of his children with epilepsy suffered from temporal lobe seizures, a figure that is very difficult to determine. In particular, recognition is difficult because the temporal lobe seizures may vary from auras, to "absences" and psychomotor seizures, to grand mal. The auras resemble those experienced by adults in psychomotor seizures, and it is difficult for a young child to describe epigastric, cephalic, perceptual, or oral factory and gustatory auras as well as complex experiences. It is also rare for a mother to appreciate that her child is suffering from them. Indeed, Ounsted says that quite frequently children with these auras will hide them from their parents lest they be regarded as "mad." Furthermore, the clinical severity of the seizures may range in the same child from mild absences to grand mal seizures at different stages in his life, as in our Case 1.

A second difficulty stems from the lack of continuity in investigations and management of children, who are often looked after by one set of authorities (pediatricians, educational authorities, juvenile courts) until adolescence (about 15 or 16 years), when they are handed over to another group (neurologists and psychiatrists, labor exchanges, ordinary courts of justice).

A third factor is the widely recognized difficulties in interpreting the EEG records of childhood and equating them with those of adult life. The anterior spike-discharging focus used as a criterion in the EEG diagnosis of adults cannot be readily applied to children, in whom the abnormalities on routine examination take other forms such as generalized dysrhythmias or occipital and posterior temporal spikes. Gibbs has discussed the migration of foci with advancing years, stating that a posterior focus is seen in infancy, a midtemporal focus in the

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**Fig. 3. Case 1.** Tracing with patient under quinalbarbitone sodium (Seconal) narcosis at age 16 years, shows frequent left anterior and mid temporal spikes (Channels 5, 6, and 7).
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school-age period, and an anterior temporal focus in the adult years. However, he realized that the problem is not as simple as that, stating that approximately half of the children who had a midtemporal focus at 10 years of age will have a normal EEG at 15 years and cease to have epilepsy, while about 15% will have developed anterior spikes. In our own experience with patients from childhood onward, it commonly seems that not until adolescence does an anterior temporal focus crystallize. Yet we have seen a typical anterior spike-focus in a child as young as 8 years.

The following case illustrates some of the difficulties of diagnosis. In this patient, it was not his seizures, which had gone unrecognized, but his educational difficulties that led to his referral for operation. Other cases of a similar nature can be cited. Ounsted and his colleagues on the first page of their monograph gave a good example with epigastric and autonomic phenomena, semipurposeful movements, and an organized visual experience, all of which had been unrecognized by the child's mother and family doctor.

Case 2

A 12-year-old boy, referred by P. R. Evans, M.D., F.R.C.P., had had some sort of "fit" at the age of 8 years, when he had lain unconscious on the ground by the side of a football field for some 20 minutes. He was treated with anticonvulsant drugs for several years by one of my senior pediatric colleagues who, because no further periods of unexplained loss of consciousness had subsequently occurred, was on the point of recommending that medication be discontinued. The boy, however, was falling behind in his class work, which was worrying his parents who could not account for it. When 12 years old he developed nasal sinusitis, and had his skull x-rayed to show the nasal sinuses. The

Fig. 4. Case 1. Tracing with patient under thiopentone (Pentothal) narcosis at 16 years, shows similar spiking over left temporal convexity with frequent and sometimes independent spiking with phase reversal at the left sphenoidal electrode (Channels 11 and 12). There is no diminution of barbiturate-induced fast activity.
Fro.

5.

Case

2.

X-ray films taken to show the nasal air sinuses reveals a small calcified mass projected through the right orbit.

films revealed a small, spherical calcified shadow in the anterior part of the right temporal lobe less than 1 cm in diameter (Figs. 5 and 6). A pneumoencephalogram showed a normal ventricular system, but EEG studies including sphenoidal leads under thiopentone (Pentothal) narcosis disclosed a spike-discharging focus in the right anterior temporal region (Fig. 7). Further questioning of his mother revealed that, although there had been no further “fits” since the episode on the football field, the boy for several years had been experiencing frequent “shivering spells” lasting a few seconds in which he would run up to her for comfort clutching his abdomen.

Operation was decided upon even though the “shivering spells” seemed mild, in the hope of improving his educational difficulties. Following a right anterior temporal lobectomy he has been followed for 2 years and has been free of these “shivering spells,” his schooling has improved, and medication has been withdrawn. The underlying lesion proved to be a hamartoma in the hippocampal gyrus (Figs. 8 and 9).

Comment

It would thus appear that in the generality of epileptic children temporal lobe epilepsy is comparatively rare and other forms of epilepsy, possibly transient, relatively common. In contrast, according to DeJong, statistics can be produced showing that anywhere from 30% to 80% of adult epileptic patients suffer from it. Yet, as my colleagues Pond and Bidwell pointed out, most adult patients with temporal lobe epilepsy in our institutions usually developed epilepsy in childhood. However, in their extensive survey of some 14 general practices in Great Britain, they were not able to make a clinical diagnosis of temporal lobe epilepsy in any of their 45 epileptic children under the age of 10 years and in only 4 out of 23 other children between the ages of 10 and 14 years.
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(17%). Pond and Bidwell, however, did not make intensive EEG studies of their cases. In 1962, Chao and his associates pointed out the frequency of temporal lobe epilepsy in childhood, saying that it was occurring in 15% of their epileptic children under the age of 15 years. In 94 children they observed both psychomotor seizures and a spike or sharp wave temporal EEG focus; in 66 children they observed psychomotor seizures but could not find an EEG focus; and in a further 94 children they obtained a temporal lobe focus, but most of these children evidently had fits of a type other than psychomotor. In 24 of their 90 children with a clinical and EEG diagnosis of temporal lobe epilepsy, the onset of seizures occurred before the age of 3 years, and childhood infections and febrile convulsions appeared relatively common. They thought that with medication most of these children were well controlled, and that as their epilepsy disappeared the EEG also became normal. The pathological changes underlying their cases were discussed only briefly.

Ounsted's Contributions

In 1966 a most important contribution to the recognition of temporal lobe epilepsy in children came from Ounsted, et al. Working in Oxford, England, they collected and reported 100 children ranging in age from...
TABLE 1

Etiological groups in 100 children
with temporal lobe epilepsy

<table>
<thead>
<tr>
<th>Group No.</th>
<th>No. of Cases</th>
<th>Median Age at Onset of First Fit (yrs)</th>
<th>Median Age at Onset of Temporal Lobe Epilepsy (yrs)</th>
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<tr>
<td>1. Cerebral insult*</td>
<td>35</td>
<td>3</td>
<td>5½</td>
</tr>
<tr>
<td>2. Febrile convulsions or status epilepticus</td>
<td>32</td>
<td>1½</td>
<td>3½</td>
</tr>
<tr>
<td>3. No related antecedent</td>
<td>33</td>
<td>4½</td>
<td>8</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>2</td>
<td>5</td>
</tr>
</tbody>
</table>

* The acute cerebral insults comprised: birth injury, 10; head injury, 6; meningitis of various types, 11; encephalitis, 5. The chronic cerebral insults comprised: Schilder's disease, tuberous sclerosis, astrocytoma, phenylketonuria, 1 each.

under 3 to 15 years in whom a firm diagnosis of temporal lobe epilepsy was made both on clinical and EEG grounds, in 17 of whom the diagnosis was made while still under the age of 3 years. Their 100 children were drawn from about 1000 children referred to various hospitals in their catchment area with seizures of all sorts, and their figure of 1 in 10 epileptic children referred to hospital is probably the closest approximation to the prevalence of temporal lobe epilepsy in childhood. All their 100 patients apparently suffered from psychomotor type of seizures, and all but 12 of them from grand mal as well.

In their monograph, these authors stressed that repeated EEG studies were often required (many of their patients having had more than 10 records), as were sleep records and various activation techniques including Megamide and Pentothal records. Details of their techniques have not yet been published. However, personal communications with Ounsted have emphasized the need for performing the examination while the child is lying peacefully and tranquilly. Sleep records may be required. If need be, medication may have to be withdrawn until a spike focus is detected. By such means Ounsted and his colleagues demonstrated spike or spike-and-wave foci in all their 100 cases, and found that in two thirds of them the focus was unilateral. They point out that a single record performed on a frightened child is of no value for the child is so "alerted" that the focus is suppressed.

Ounsted and his colleagues divided their 100 cases into three distinctive etiological groups: cerebral insult, febrile convulsions, and no detectable origin (Table 1). Nine of their patients had gross neurological abnormalities, such as hemiplegia, generalized hypotonia, and choreoathetosis; although it is not clearly stated, these particular patients came mainly from their cerebral insult group. The remaining 91 patients had no such signs of gross cerebral damage.

Each group was then analyzed to ascertain the influence of several possible etiological factors. The possible effect of birth injury was sought. The authors matched their 10 cases of known birth injury with controls, and also in their whole series compared birth weights and prematurity; they found that birth injury was not a significant factor in the statistical sense, although it may sometimes contribute to the initial seizure. This supports our own expressed views.

Ounsted and his colleagues, however, were impressed by the influence of antecedent febrile convulsions and of status epilepticus in their children. Febrile convulsions in infancy are common, and most pediatricians regard them as of little consequence. However, experience teaches that they should be regarded in a different light. These convulsions are rare before the age of 6 months, reach their peak incidence about 18 months, and again are rare after 5 years. The more severe the febrile convulsions the more likely it is to be prolonged into status epilepticus which, as is well known, causes cerebral damage particularly to the mesial temporal structures, such as the hippocampus, amygdala, and uncus. It is often difficult to determine from the history just how severe a febrile convulsion has been. Ounsted and his colleagues were clearly impressed that, in their epileptic children with an antecedent febrile convulsion, these episodes usually occurred before the age of 3 years (median age, 1 yr 4 mos), while the median age of onset of temporal lobe seizures in this particular group was also earlier (median age, 3 yrs 10½ mos) than in their other two groups. Furthermore, they observed that...
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patients in this “febrile convulsion” group had more frequent seizures of both grand mal and psychomotor type than did patients with no evident antecedent cause, tended to have lower I.Q. levels, and were more likely to exhibit the “hyperkinetic syndrome,” 37 or to show catastrophic rage phenomena. 40 In these various respects, their “brain insult” group appeared comparable to their “febrile convulsion” group. However, in one additional respect their “febrile convulsion” group stood out, for the risk of epileptic phenomena occurring in siblings was very high indeed (30%), a significant figure three times as high as in the “antecedent-free” group. We shall return to these various points later.

Ounsted and his colleagues argued that their “febrile convulsions” group probably had developed Ammon’s horn sclerosis as the epileptogenic lesion. 40 They did not attempt to name the underlying lesion in their “antecedent-free” group. All this has greatly interested us, for our observations may be able to supply the missing key regarding the pathology, which unlocks the problem of etiology of temporal lobe epilepsy in childhood and adolescence.

Pathological Findings in Surgical Patients

To date we have now published various findings following unilateral anterior temporal lobectomy for epilepsy in two somewhat overlapping series of cases, each comprising 100 consecutive patients. 17,20,21,23,54 The criteria of selection of cases, pathological findings, and the results of surgery both with regard to seizure relief and social adaptation have already been stated. In practically all cases a part of the hippocampus and also nearly all of the amygdala were included in the resected specimen. 21 We arbitrarily tended to exclude patients who were also mentally defective (I.Q. below 70) but most of our patients exhibited also psychiatric abnormalities. In about half the patients in both series, the lesion now known as “mesial temporal sclerosis” was found at operation (Fig. 10). Basically, this is the same lesion as “Ammon’s horn (hippocampal) sclerosis,” but the former term is preferable because the sclerotic process usually extends into other structures in the mesial part of the temporal lobe, such as the amygdala, the uncus, and the hippocampal gyrus as well. In a fifth to a quarter of the cases, hamartomas were found; these are small, benign, developmental malformations, usually of gial origin, and too small to deform the pneumoencephalograms (Figs. 9 and 11). These lesions tend to cluster in the region of the amygdala and the hippocampal gyrus. In a tenth of the cases we observed only scars and infarcts, lesions which might have preceded the epilepsy or resulted from it. In the remaining quarter of our patients we were unable to demonstrate any significant lesion affecting neurons in the resected specimen, although some of the patients subsequently showed improvement in both their epilepsy and social adjustment. Subpial gliosis or white matter gliosis by itself was not regarded as significant, for these lesions are common in routine autopsy findings without overt neurological disease.

Reports on the etiology and significance of Ammon’s horn (mesial temporal) sclerosis have recently been reviewed 17,18,21,52 from the time of its first description in 1825 at autopsy by Bouchet and Cazauvieihl. 5 Its histological appearances are illustrated in Fig. 10. It is the commonest single lesion found at postmortem in the brains of institutionalized epileptics, occurring in about half of the patients, usually unilaterally (80%). 54 Unilaterality, important when surgical treatment is considered, cannot be determined from the often quoted papers of Spielmeyer and Scholz, 52 for it would appear that at autopsy they only examined one cerebral hemisphere in detail, and then generally the dominant hemisphere. 7,62 While there is often a genetic factor evidenced by a high family history of epilepsy (about 14%) we believe this is an acquired lesion created usually by an asphyxial episode in infancy, such as a febrile convulsion or status epilepticus. 21,52 The nerve cell damage then sustained leads to a scarring process which in its turn becomes an epileptogenic lesion. Mesial temporal sclerosis appears to be the most common single lesion responsible for drug-resistant and chronic epilepsy that originates in the first two decades of life. The fact that it can also occur in young mentally defective patients who have not yet exhibited epilepsy does not rule out the epileptogenic role of this lesion. 8 Occasionally, patients are encoun-
Fig. 10. Photomicrographs of Ammon's horn (mesial temporal) sclerosis. Top: Normal hippocampus. Cresyl violet, ×13. Bottom: Hippocampus of 16-year-old girl in specimen removed at operation shows gross loss of cells in the Sommer sector (H1) and in the end-folium (H3-5) with some preservation of cells in the resistant sector (H2). The patient had developed frequent epilepsy at age 5 years after a status epilepticus at the age of 2 years; following operation she has been completely fit-free for over 3 years. Cresyl violet, ×13.

tered who have had a status epilepticus in infancy but whose epilepsy did not appear until adult life.21

The best results from surgery have occurred when mesial temporal sclerosis has been encountered at operation. In our second series of 47 patients with this lesion, 24 (51%) were rendered completely seizure-free, eight (17%) were experiencing only occasional seizures, while only two (4%) had no seizure relief.22 In social adaptation, as well, the best results occurred with this lesion.24 Aggression in particular appears to be related to mesial temporal sclerosis (personal observations), occurring as it does most frequently in patients whose epilepsy began at an early age.21 Somewhat comparable results in the relief of epilepsy occurred
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whenever a hamartoma was found in the resected specimen, although this point is not clearly brought out in our second series. However, improvement in social adaptation inexplicably is not as marked when hamartomas are found as when mesial temporal sclerosis is present. Indeed, several of our cases with persisting psychosis, usually of schizophrenic type, had hamartomas. The results of operation when only scars and infarcts or no lesions at all were found, although gratifying, were not as good as whenever mesial temporal sclerosis or a hamartoma was present.

Comparison of Ounsted’s Cases with Our Surgical Cases

Our cases showed many striking and probably significant similarities to those of Ounsted and his colleagues. In our two series of 100 cases, a history of antecedent infantile convulsions or status epilepticus was forthcoming in 30% to 40% of the cases whenever mesial temporal sclerosis was subsequently demonstrated. In contrast, infantile convulsions were rare in our other pathological groups. Again, in our patients with mesial temporal sclerosis the age of onset of habitual seizures was generally younger than 10 years, and frequently before the age of 2. Thus, in this respect, our two series of cases strongly resemble the “febrile convolution” group of Ounsted. Our patients, like Ounsted’s, all had psychomotor seizures, and several also had grand mal attacks. Other points of resemblance are that our cases of mesial temporal sclerosis, like Ounsted’s “febrile convolution” group, had a high incidence of familial epilepsy (14% of near relatives in our cases compared with 30% in Ounsted’s), while in neither our patients nor those of Ounsted’s did birth trauma appear to play a significant part. Birth injury, however, may still be a factor, for Weller and Norman have reported a case of mesial temporal sclerosis in one of a pair of identical twins, who suffered from birth trauma whereas the twin did not. However, the injured twin, who died at the age of 3 yrs 2 mos, also had had epileptic convulsions during life, and it is difficult to sort out which lesions were due to birth trauma and which to infantile convulsions. The healthy twin had a normal EEG at 4 years and had never had seizures. It seems likely in this instance that birth injury was responsible for the infantile epilepsy that in its turn produced the sclerotic lesion. It is also difficult to rule out a trivial head injury as the trigger that set off the status epilepticus in the case of Small and Woolf.

Ounsted’s group of patients whose brains were damaged by meningitis or encephalitis is not represented in our case material. One reason for this is that a third of his cases in this group were mentally defective, and would probably have been excluded by us on this ground. Again, the cerebral damage to his patients usually occurred during the first months of life, and presumably the resultant lesions were patchy and widespread with particular involvement of the temporal lobes. They subsequently might have shown diffuse multifocal EEG abnormalities which would be deemed a contraindication to a unilateral temporal lobectomy. Thus some of

![Image](https://example.com/image.jpg)

**Fig. 11.** *Left:* Resected temporal lobe of 20-year-old man with 8-year history of epilepsy shows hemorrhage with a hamartoma of the fusiform gyrus. The patient has been completely fit-free since operation (15 years). *Right:* Section showing recent localized hemorrhage in hamartoma. Nissl, ×1.
these patients showed gross neurological signs such as a hemiplegia, for which a hemispherectomy rather than a temporal lobectomy would have been required. Febrile convulsions apparently were not observed in this group, and the median age of onset of temporal lobe epilepsy was 5 yrs 3 mos, slightly later than in the febrile convulsion group.

It is the possible substrate in Ounsted’s third or antecedent-free group of epileptic children that is particularly intriguing. In his monograph, Ounsted gave no inkling of what he considered was the etiology in this group that formed a third of his cases, but in a later paper he hinted that hamartomas may have been responsible for some of them. Indeed, the resemblances to the hamartoma cases which constituted a quarter of our material are many and strong. Thus, the median age of onset of temporal lobe epilepsy in this group of Ounsted’s was 8 years, which was later than in his other two groups. The later onset of epilepsy seems true of our hamartoma material. Again, both in Ounsted’s third group and in our hamartoma cases the incidence of a positive family history of epilepsy was low. Considering the frequency with which these hamartomas occur in surgical case material, one can only conjecture that they must have occurred in some of Ounsted’s cases also, and are represented in his third group.

Personal Operative Experiences

My personal experience with surgery in children with temporal lobe epilepsy is limited, but it does indicate that some of these children should be considered for operation and can probably be benefited. In our recent series of 100 consecutive patients, there were nine children operated on under the age of 15 years. Yet most of the other patients in that series had developed epilepsy before that age. Of the nine children, five were boys and four were girls, and the relevant clinical and pathological data are found in Table 2. All suffered from psychomotor seizures, while in Cases 1, 2, 6, and 8, from grand mal as well. Undoubtedly eight of the nine children were pubescent (ages 11 to 14). One patient (Case 11) was grossly mentally defective, and probably on this account should never have been operated on. All nine except one (Case 6) also displayed severe behavioral disorders characterized chiefly by aggression, and undoubtedly this was an important reason for referring them for surgery. Five were regarded as unmanageable even in institutions (Cases 1, 2, 3, 8, and 9), while postoperatively two had to return to broken homes.

Postoperative improvement in epilepsy was striking, but not quite as gratifying as that in behavior and social adaptation. All except the mentally defective patient (Case 11) were improved with regard to epilepsy, four being rendered completely fit-free (Cases 3, 7, 8, and 9), while four others (Cases 1, 4, 5, and 6) were also markedly relieved. Mesial temporal sclerosis was disclosed at operation in seven, and all these were improved with regard to epilepsy.

However, the improvement in social adaptation was not so striking. Neither patient with non-specific lesions was benefited. Of the seven with mesial temporal sclerosis, two made a normal adjustment, including the one who was normal beforehand (Cases 5 and 6). The other five were benefited to some degree. Four of the patients with mesial temporal sclerosis are at least in fairly regular employment and one is a satisfactory student. One, although better, is still institutionalized since his home circumstances are bad.

The results of surgery on the whole are encouraging, particularly since five of the children, all pubescent, were unmanageable due to behavior problems before operation (Cases 3, 4, 7, 8, and 9). Also eight of the nine were pubescent. One naturally wonders what would have been the outcome had they been operated on earlier. The youngest case in our first series has already been reported. She was 2 yrs 2 mos at the time of operation, and our last information 10 years later was that she was still free of fits. The youngest patient in our second series (Case 5, Table 2) is an example of what surgery can achieve, and is reported below.

Case 3

An 8-year-old boy (Case 5, Table 2) was referred by P. R. Evans, M.D., F.R.C.P., with a history of psychomotor seizures dating back 4½ years. He was the older of two children. There was no family history of epilepsy, but there was one of psychopathy on
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<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age, Sex</th>
<th>Antecedent factors</th>
<th>Age at onset of epilepsy (yrs)</th>
<th>Side</th>
<th>Postoperative Results</th>
<th>Social Adjustment</th>
<th>Follow-up (yrs)</th>
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<tbody>
<tr>
<td>1</td>
<td>13 M</td>
<td>difficult birth</td>
<td>11</td>
<td>L</td>
<td>non-specific</td>
<td>almost fit-free</td>
<td>continued intractable</td>
</tr>
<tr>
<td>2</td>
<td>13 F</td>
<td>difficult birth</td>
<td>infancy</td>
<td>R</td>
<td>non-specific</td>
<td>not improved</td>
<td>remained an imbecile</td>
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<tr>
<td>3</td>
<td>13 M</td>
<td>infantile convulsions</td>
<td>7</td>
<td>R</td>
<td>mesial temporal sclerosis</td>
<td>fit-free</td>
<td>improved, left “cerebellar” tremor</td>
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<tr>
<td>4</td>
<td>14 F</td>
<td>difficult birth</td>
<td>8</td>
<td>R</td>
<td>mesial temporal sclerosis</td>
<td>almost fit-free</td>
<td>neurotic</td>
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<tr>
<td>5</td>
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<td>birth asphyxia; at age 6 febrile convulsions</td>
<td>3½</td>
<td>R</td>
<td>mesial temporal sclerosis</td>
<td>occasional fit</td>
<td>normal, poor verbalization</td>
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<td>6</td>
<td>14 F</td>
<td>febrile convulsions</td>
<td>9</td>
<td>R</td>
<td>mesial temporal sclerosis</td>
<td>occasional fit</td>
<td>normal</td>
</tr>
<tr>
<td>7</td>
<td>13 F</td>
<td>febrile convulsions</td>
<td>1½</td>
<td>R</td>
<td>mesial temporal sclerosis</td>
<td>fit-free</td>
<td>improved; occasional temper tantrums</td>
</tr>
<tr>
<td>8</td>
<td>11 M</td>
<td>unknown</td>
<td>9</td>
<td>R</td>
<td>mesial temporal sclerosis</td>
<td>fit-free</td>
<td>much improved</td>
</tr>
<tr>
<td>9</td>
<td>14 M</td>
<td>head injury</td>
<td>5</td>
<td>R</td>
<td>mesial temporal sclerosis</td>
<td>fit-free</td>
<td>much improved</td>
</tr>
</tbody>
</table>

TABLE 2

Results of anterior temporal lobectomy in nine cases of temporal lobe epilepsy

the maternal side. His birth was prolonged and difficult, and he was born asphyxiated. His birth weight was 4.6 kg. He passed the milestones of childhood normally, except that he did not talk until he was 3½ years old. From this period onward he suffered from epileptic attacks characterized by sudden loss of consciousness with falling and preceded by some epigastric sensation that he found difficult to describe. In the actual attacks he would stare ahead without convulsing, and make mouthing movements with some salivation. After 20 to 30 seconds he would regain consciousness, but would remain confused and sometimes aggressive for a further 5 minutes. Sometimes he was incontinent of urine during the seizures. Their frequency varied from 1 to 20 a day with an occasional day’s freedom. Medication with various drugs such as phenobarbital, hydantoin, primidone, and beclamide had not controlled the seizures. Between attacks he showed cyclic episodes of aggression and disobedience in which he would do the opposite of what he was told. The epileptic attacks, the incontinence, and his rages had seriously interfered with his schooling, and he was consequently backward. A recent EEG had shown an unstable alpha rhythm at 6–7 cps with at times some “doubtful sharp waves in the right temporal and posterior regions” (Dr. G. Pampiglione).

Examination. The patient was “a dull boy who spoke in a monotonous and slow way, and who not infrequently asked that questions be repeated. The range of his general knowledge was very poor." His I.Q. levels (WISC) were F.S. 95, V.S. 100, P.S. 90 (Dr. V. Meyer). Neurological examination disclosed slight incoordination of the left arm and an inability to walk a straight line. How many of these features were due to his illness

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or to his medication could not be determined. Caloric tests of vestibular function showed no directional preponderance (Dr. R. Hinchcliffe). X-rays of the skull showed that the right temporal region bulged slightly compared with the left, while the pneumoencephalograms revealed that the right temporal horn was also slightly smaller (Fig. 12). Several EEG studies, including records with sphenoidal electrodes under thiopentone narcosis, disclosed frequent spike discharges in the right temporal area especially at the sphenoidal electrode, radiating to the temporal convexity and inferior frontal areas. There was also diminution of barbiturate-induced fast activity between the right sphenoidal and right ear electrodes (Figs. 13 and 14).

In view of his youth, operation was delayed for 18 months while further drug regimes were tried by his pediatrician. These proved unavailing.

Operation. In May, 1961, when he was 9 years old, a right craniotomy was performed under light intratracheal anesthesia using nitrous oxide and oxygen with suxamethonium chloride (Scoline) as a relaxant (Dr. S. Barker). Electroacorticography confirmed the presence of spike discharges in the anterior temporal region together with a marked and unusual absence of electrical activity from the inferior surface of the temporal lobe (Dr. M. V. Driver). A 6 cm right anterior temporal lobectomy was performed. In the fresh specimen the region of the uncus was palpably hard. A post-excision corticogram showed an occasional spike in the region of the insula and in the inferior frontal region just above the Sylvian vein. The histological report described "a severe amygdaloid and Ammon's horn sclerosis of long standing" (Dr. C. Treip).

Postoperative Course. Convalescence was straightforward. The patient showed a minimal left upper quadrantic homonymous hemianopia, but no longer any incoordination of his right arm or gait. He has since been followed regularly for 8 years. During this time he has been attending a normal school, and there have been no further incidents of aggression or incontinence.

However, he still has occasional minor epileptic attacks characterized by twitching of the left limbs for a matter of seconds up to 3 minutes and associated with dazedness but without loss of consciousness. He remains solitary, but his hobby is mathematics. He is still at school but in the highest form of a
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secondary modern school. He appears to communicate better with his schoolmates and his brother than with his parents. He has passed his General Certificate of Education in physics and mathematics (a university prerequisite) but not in English, chemistry, or history. He shows reticence to speak, and direct questioning usually elicits one word answers. His sentences are very simple, seldom consisting of more than six words. Yet he can quote the value of $\pi$ (the ratio of the circumference of a circle to its diameter) to eight places of decimals. His present I.Q. levels are F.S. 119, V.S. 126, and P.S. 108 on the Wechsler scale (Miss E. Drew). Attempts have been made at speech therapy. Sphenoidal EEG at 1 year after operation showed no spiking activity (Dr. M. V. Driver). It is felt that there has been a very gratifying decrease in epilepsy, a marked improvement in behavior and schooling, a marked improvement in intellect, but that verbal speech difficulties that preceded the epilepsy have not been influenced either way by operation. Current medication is hydantoïn (100 mg) and primidone (250 mg) both twice daily.

**Natural Outcome of Temporal Lobe Epilepsy**

It seems that temporal lobe epilepsy in childhood is likely to be carried on into adult life. There are no substantial statistics to support or refute this view, but it is an impression held by Ounsted who tells me that, with a few exceptions, most of his temporal lobe epileptic patients on reaching adult life continue to lead useful lives with only an occasional seizure while on medication.89 He does not know for certain whether any one of his cases has become completely seizure-free. In this respect his views conflict with those of Chao and his colleagues who tended to regard most cases of temporal lobe epilepsy in children as comparatively minor. My own experience tells me that the clinical severity of the psychomotor and grand mal seizures that characterize temporal lobe epilepsy in childhood bear no constant relationship to the behavioral or educational disturbances. Thus, in our Case 1, the seizures observed when the boy was 10 years old seemed trivial being “absences” lasting only a second or two. Yet the behavioral disorder was gross. Similarly in Case 2, and again in Case 3, the “shivering spells” were mild and without loss of consciousness, but the educational difficulties associated with them were appreciable.

**Present Position Regarding Incisural Sclerosis**

One of Penfield’s many contributions to the understanding of temporal lobe epilepsy was the introduction of the concept of “incisural sclerosis.”12,43 This was based not on detailed histological studies of the whole resected temporal lobe, as was done by Profes-

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**Fig. 13.** Case 3. Tracing of patient at age 8 years, while awake, shows spike discharges with phase reversal between Channels 1 and 2 and between Channels 1 and 3.
Fig. 14. Case 3. Tracing of patient under thiopentone (Pentothal) narcosis shows the same convexity spike focus as well as a focus at the right sphenoidal electrode (Channels 9 and 10). There is also a reduction of barbiturate-induced fast activity in Channels 9 and 10 compared with 11 and 12. A similar reduction is seen in Channels 2 and 3 compared with 6 and 7.

Sor Alfred Meyer and Dr. J. A. N. Corsellis and their colleagues in our material, but on "piece-meal" biopsies of the mesial temporal structures. Essentially, the distribution of lesions in the concept is the same as that found in mesial temporal sclerosis, but our ideas of its etiology differ. Penfield held that it was due primarily to birth injury, and that during parturition the fetal head was molded, so that the anterior choroidal and posterior cerebral arteries impinged against the free edge of the tentorium leading to anoxic changes in these mesial temporal structures, which as the child grew older "ripened" into an epileptogenic lesion. For a while the views of Lindenborg that these arteries could be selectively compressed during raised intracranial pressure leading to tentorial compression appeared to support Penfield's views.

Our experience, however, has led us away from the concept of birth injury to support instead one of some asphyxial episode during infancy, such as a febrile convolution or a status epilepticus as the principal cause, although we concede that birth injury somehow may "spark off" the febrile convolution. Most of the evidence has already been reported but it can be summarized here. First, in our two large series there was the same incidence of difficult birth in cases with mesial temporal sclerosis as in cases without it. Second, in cases of mesial temporal sclerosis there was a high incidence (30%-40%) of recorded infantile convulsions, but such convulsions were rare in other cases. Third, recent damage to the hippocampus and other mesial structures has frequently been observed at postmortem examination after status epilepticus in children, but apparently not in adults. Fourth, mesial temporal sclerosis is not found at autopsy in the brains of patients who developed epilepsy in adult life, as after frontal lobotomy or electroconvulsive therapy performed without relaxants.
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Fifth, certain structures supplied by the anterior choroidal and posterior cerebral arteries regularly escape sclerosis, such as the pallidum and the calcarine cortex.

To these objections can now be added the observations of Ounsted and his colleagues that birth injury appears to have played little part in the development of temporal lobe epilepsy in their patients, and the comment of Norman that the histological change in the Ammon's horn does not resemble an infarct. There is also Norman's further observation that birth asphyxia tends to damage the H2 (resistant) sector of the hippocampus and not the H1 and H3–5 sectors, which is contrary to what is found in mesial temporal sclerosis (Fig. 15).

Furthermore, there is the evidence furnished by Small and Woolf and Brierley that neither status epilepticus nor cerebral anoxia in children causes brain swelling or tentorial herniation, evidence that negates Lindenberg's hypothesis.

In support of the view that the hippocampus might be selectively vulnerable to anoxia in infancy but not in adult life, Ounsted and his colleagues have pointed to the analogy of the Fring's strain of mice, which are specially bred because they display audiogenic seizures that are limited to the period between the 17th and 25th days of life. They also give other analogies.

Recently, McLardy has produced Ammon's horn sclerosis experimentally in guinea pigs by inducing repetitive seizures while the animals are under hypoxia or hypoglycemia, and are also hyperthermic. This latter point seems essential, and to achieve it he heated his animals in a glass-doored oven until their rectal temperatures were 43°–45°C. He made the further observation that, if the animal's head was on one side during the seizures, the Ammon's horn on the dependent side was always more affected than in the overlying hemisphere.

Current Views Regarding Pathogenesis

My colleagues and I were not the first to equate Ammon's horn (mesial temporal) sclerosis with anoxia, but we are the first to report the favorable prognosis that follows temporal lobectomy whenever this lesion is encountered, and is presumed unilateral. At the Colloquia on Temporal Lobe Epilepsy held in Marseille in 1954 and in Washington in 1957, both Norman and Malamud besides ourselves raised this hypothesis. Subsequently, Gastaut in summing up the observations reported at these two colloquia wrote, both in French and in English versions, that the Marseille school was in agreement with the Montreal school in ascribing temporal sclerosis "to an ischemia secondary to vascu-

Fig. 15. Case 3. Photomicrographs to contrast with Fig. 10. Left: Ammon's horn after birth asphyxia in a patient with no epilepsy. Long-standing lesions are seen in the Sommer sector (H1) and in the resistant sector (H2). Carbol azure, ×6.3. Right: Another case of birth asphyxia with gross old lesions in H2 and in the end-folium (H3–5) but with sparing of the Sommer sector (H1). Carbol azure, ×6.3. (Reproduced from Ounsted, C., Lindsay, J., and Norman, B. M., Biological Factors in Temporal Lobe Epilepsy, London: Heinemann Medical Publishing Company, 1966, by kind permission of the authors and publisher.)

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lar compression in the course of an intracranial hypertension occurring during or after birth."\textsuperscript{24,25} He then went on to write "it is certain that the views of Malamud and Norman, if confirmed, would completely disrupt the concepts of modern epileptology; for, from the etiological standpoint, one would have to admit that convulsions accompanied by cerebral anoxia could cause lesions later producing temporal lobe epilepsy."\textsuperscript{24,25} Ounsted, \textit{et al.},\textsuperscript{40} and ourselves say just that, for it is the only concept which is in harmony with the facts. Gastaut went further and argued that it would be illogical to perform a temporal lobectomy as this would respect only "secondary lesions leaving the primary focus intact."\textsuperscript{24,25} Yet at the same time he wondered how Penfield managed to cure more than half his cases by limiting his intervention to an anterior temporal lobectomy. Gastaut's astute words written in admonition have proved prophetic.

\textbf{Conclusions}

It would appear that temporal lobe epilepsy, which all authorities agree occurs frequently in adults, commonly has its origins in childhood, but is often unrecognized as such at this period of life. Its possible incidence is 10\% of all children who are referred to hospital with epileptic manifestations. While the majority of children with more common epileptic manifestations will grow out of them as they reach adolescence, it seems likely that temporal lobe epilepsy will continue into adult life, although it may not be disabling.

From the etiological viewpoint, patients with temporal lobe epilepsy in childhood appear to be divisible into three approximately equal groups:

\textbf{Group 1.} Patients with cerebral damage due to acquired illnesses in childhood, such as meningitis, encephalitis, cerebral tumors; or to birth injury, head injury, or congenital abnormalities such as subnormality, or phenylketonuria. In this group there are few patients with a family history of epilepsy.

\textbf{Group 2.} Patients with febrile convulsions or status epilepticus. The underlying lesion here is one of mesial temporal sclerosis. In this group patients with a family history of epilepsy are common.

\textbf{Group 3.} Patients without evident antecedent factors. The underlying lesion in some of these is probably hamartoma, although in many no definite histological lesion may be demonstrated in the temporal lobe. There is sometimes a family history of epilepsy.

The place of surgical treatment in temporal lobe epilepsy in childhood has not yet been established. Examples have been given of cases in which it proved of benefit, particularly when mesial temporal sclerosis was the underlying lesion. As epilepsy in some children will prove to be lifelong, I feel that pediatricians confronted with the problem of epilepsy should take a long-term view of what is going to happen to their patients when they grow up and have to leave home and face the world. Surgery may prove applicable to only a few of these patients but, if an accurate diagnosis of temporal lobe epilepsy can be made, and the outlook after an arbitrary period of intensive medication of, say, a year still appears bleak, the natural reluctance shown by many pediatricians to consult their neurosurgical colleagues should be overcome. Even then surgery should only be undertaken if the clinical and EEG data indicate a focal origin for the seizures.

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