

## TEMPORAL LOBE EPILEPSY WITH PERSONALITY AND BEHAVIOUR DISORDERS CAUSED BY AN UNUSUAL CALCIFYING LESION

REPORT OF TWO CASES IN CHILDREN RELIEVED BY TEMPORAL LOBECTOMY

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It has long been realized that "a particular variety of epilepsy", neither petit mal nor grand mal, but characterized by a "dreamy state" and often accompanied by a crude sensation of an olfactory, gustatory, or epigastric nature, commonly arises as a result of lesions of various types in the temporal lobe (Jackson, 1888, 1889, 1898). This type of epilepsy, which has been described as epilepsy with intellectual aura, uncinat epilepsy, psychical epilepsy, and in recent years as psychomotor epilepsy, can frequently be shown by electroencephalography to be associated with a focus of spike discharge in the anterior part of one or both temporal lobes (Penfield and Erickson, 1941; Gibbs, Gibbs, and Fuster, 1948; Lennox, 1951). Often its victims also show marked personality disorders and even psychosis (Lennox, 1951).

Temporal lobe epilepsy, however, is usually considered to be rare in children. The following two case histories are therefore of interest, not only because epilepsy of this type associated with a personality disorder occurred in childhood, but also because in both patients the symptoms were caused by an unusual focal collection of astrocytes, oligodendrocytes, calcospherites, and capillaries, apparently similar to the lesion described by Penfield and Ward (1948) as a "hemangioma calcificans". Further, in both patients these symptoms were relieved by temporal lobectomy. It is possible that lesions of this kind are not as uncommon as would at first sight appear, for, while in the cases that have been reported the lesion has generally come under notice as a calcified shadow visible on x-ray films, in one of our patients the lesion was not discerned radiologically. Indeed, even the presence of the lesion was unsuspected until histological studies were completed, and pre-operatively

the electroencephalographic findings were the sole guide post to the affected temporal lobe.

### Case Histories

**Case 1.**—S.S., a girl aged 2 years 2 months, was referred in November, 1950, by Dr. J. G. Hamilton to the Children's Department of the Maudsley Hospital (Dr. K. Cameron) for investigation of fits and a behaviour disorder.

An only child of healthy parents, she was born in September, 1948, after a normal confinement, and until 14 months old she seemed a normal, placid child. At 12 months she started walking, and began saying a few words. Within two months, however, she became irritable and fretful, developed marked likes and dislikes, and often would stamp and scream for periods of up to a minute without apparent reason. Then, when nearly 2 years old, she began suffering from minor epileptic seizures, which soon increased in frequency up to six or eight a day. In a typical attack she would suddenly stop playing and look vacant for one or two seconds. Sometimes during this period she would extend her right arm, and either gaze at it or make plucking movements as if trying to pick up a non-existent object. Next moment coarse nystagmoid movements of the eyeballs would appear, and her head would fall forward for a few seconds with pupils dilated and eyeballs turned upwards. Her head would then right itself momentarily, only to fall forwards once more, this sequence going on for perhaps a dozen times. The whole attack might last two or three minutes before she would regain consciousness, but for several more minutes afterwards she would seem confused and bewildered.

Once these attacks began, her temper tantrums ceased, but her mental development halted, and she made no further progress in walking or talking. Her parents sought advice in several quarters. A provisional diagnosis of an infantile affective disorder with breath-holding spells occurring against a background of over-anxious parents was entertained, and led to her admission to the Maudsley Hospital.

Examination revealed a physically healthy child, affectionate, but very restless and difficult to manage. When 2 years 5 months old her mental age as judged from some items of the Merrill-Palmer scale appeared to be less than 18 months (Miss M. Newell). She was thus retarded more than a year. She would say a few words and hum the tune of "Boys and girls come out to play". The only abnormal neurological sign was a very slight weakness of the right lower face. The skull was of average size for her age (maximum circumference, 19 inches). The cerebrospinal fluid was normal and the blood Wassermann reaction negative. An attack of measles shortly supervened, halting until January the following investigations:—

*Radiographs of the Skull.*—These disclosed an irregular area of calcification (5.5 cm. long by up to 2.5 cm. in depth) situated in the temporo-occipital region of the left cerebral hemisphere (Fig. 1). The bony outlines of the skull were normal.



FIG. 1.—Case 1: Lateral radiograph of skull showing calcification in left temporal region.

*Ventriculography.*—This showed both lateral ventricles in their normal situations without any shift or local indentation to indicate an expanding lesion. Unexpectedly the trigone and temporal horn of the left lateral ventricle were slightly dilated with the calcified shadow appearing just beneath them (Fig. 2). Indeed, had the calcified shadow not been present, the appearances might readily have been mistaken for those of focal cerebral atrophy.

*Electroencephalography.*—Records were obtainable only with the child deeply asleep after the administration of "seconal", gr.  $1\frac{1}{2}$ , by mouth. The most obvious abnormality was relative flattening of electrical activity in the left temporal and occipital regions (Fig. 3). Normal sleep fast and slow waves were seen over the right hemisphere and the left frontal region. In addition, epileptic discharges of irregular spike-and-wave type were observed over both cerebral hemispheres, right more than left. The E.E.G.s were interpreted as suggesting a large destructive lesion of the more posterior part of the left cerebral hemisphere which was causing epileptic discharges to appear over the intact cortex of the opposite cerebral hemisphere, as well as over the anterior part of the left hemisphere.

While in hospital the child was given sodium phenytoin, gr.  $\frac{3}{4}$  t.i.d., and tridione, 0.3 g. b.i.d., for some weeks, but her attacks continued. It was clear, particularly on the x-ray findings, that some structural abnormality was present. Several diagnoses were conjectured, including calcified choroid plexus papilloma and cerebral calcification epilepsy (Geyelin and Penfield, 1929). The epileptic attacks were of an akinetic type, seemingly associated with a dreamy state and perhaps also a visual hallucination. The asymmetrical movements of the right arm and the variability of the attacks were not in keeping with true petit mal seizures, but were consistent with their being of temporal



FIG. 2.—Case 1: Encephalograms, postero-anterior and lateral, showing slight dilatation of the trigone and temporal horn of the left lateral ventricle with the calcified shadow beneath.

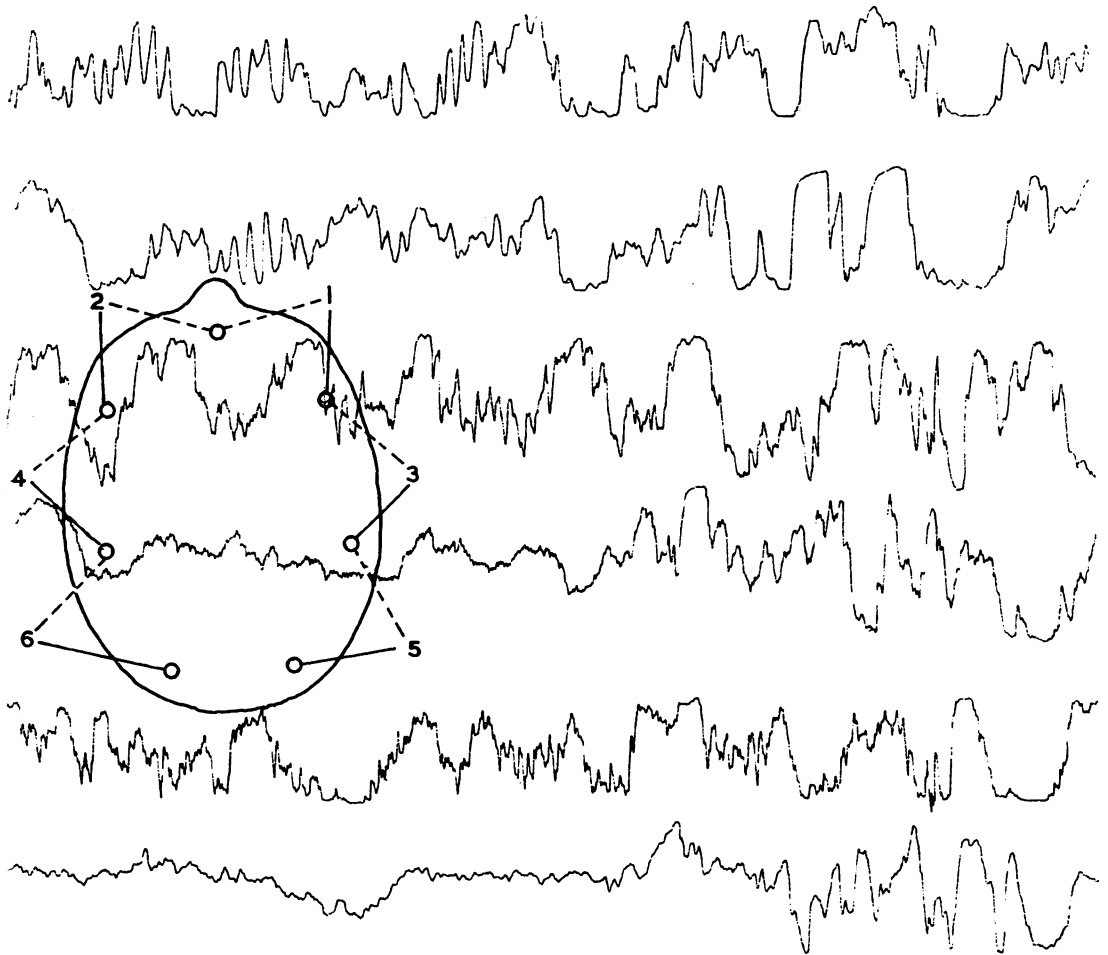


FIG. 3.—Case 1: Electroencephalogram taken with child asleep under “seconal” before operation (Dec. 12, 1950). Note the asymmetry in the posterior channels with flattening of the left side, but both hemispheres show irregular sharp waves and spikes (same paper speed as in Fig. 4).

lobe origin, although the electroencephalographic findings suggested a more widespread lesion. However, the focal character of the radiological lesion was undeniable, and neurosurgical intervention was decided upon.

*Operation.*—Under general anaesthesia a lateral craniotomy was performed, the lesion being located but not removed at this session. A week later the craniotomy was re-opened. A small transverse incision was made in the temporal lobe immediately below the superior temporal sulcus, and the temporal horn and vestibule of the lateral ventricle were laid open. A reddish-brown mass could be seen lying within the brain substance beneath the floor of the ventricle. It appeared circumscribed. Consequently a block dissection of the inferior

half of the temporal lobe and the adjacent occipital lobe was carried out, including the whole of the floor of the temporal horn and vestibule and part of the hippocampus. At the completion of the removal, the temporal pole and the superior temporal convolution were still *in situ*, as was the uncus. The operation cavity extended up to the hiatus tentorii.

*Pathology.*—The specimen consisted of the inferior part of the left temporal lobe, which had been severed by a horizontal cut through the middle temporal gyrus. It included the inferior temporal and fusiform gyri and the hippocampus but not the cortex of the pole or the uncus. It measured 7 (sagittal plane)  $\times$  5 (coronal plane)  $\times$  3 cm. and weighed 64.6 g. On coronal section the cortex to the naked eye was unduly deep and did not present the usual margin to the white matter. The latter

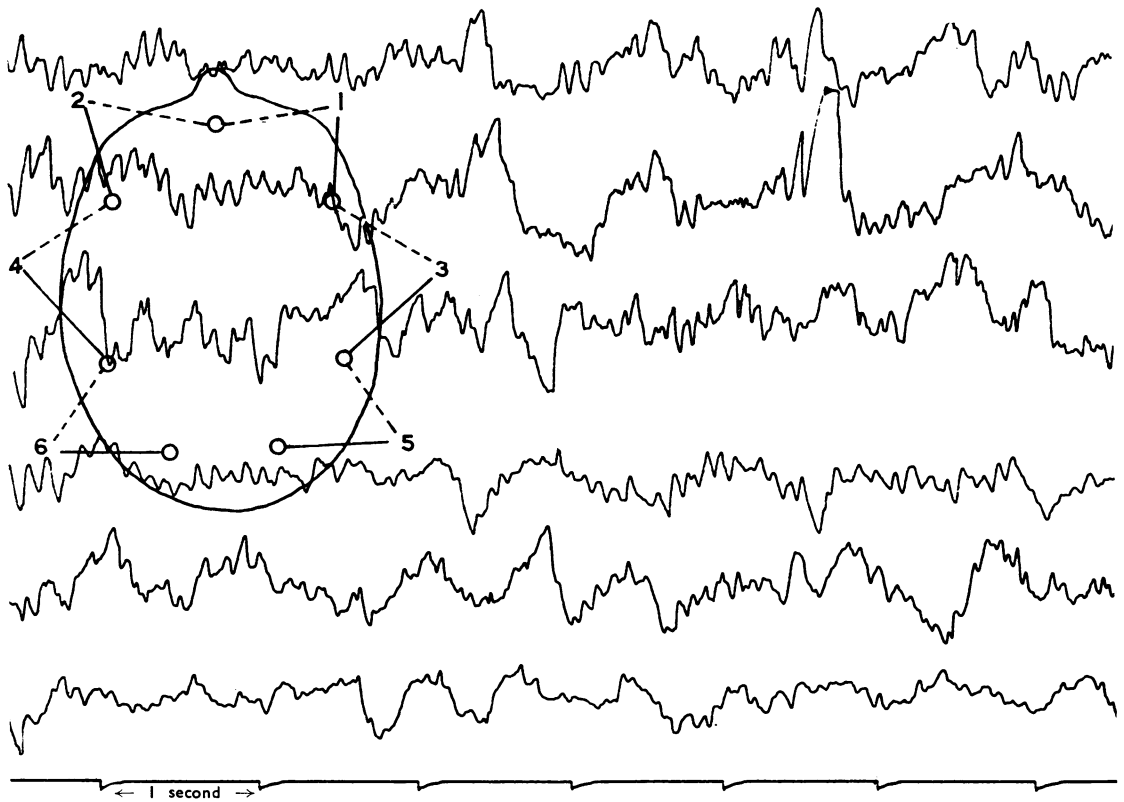


FIG. 4.—Case 1 : Post-operative (April 3, 1952) sleep record in the same position. Note that the right hemisphere is now quite normal. The posterior part of the left hemisphere shows normal sleep spindles, but there is some diminution of the high voltage very slow waves of deep sleep.

was largely replaced by a tissue which showed extensive microcyst formation giving a honeycombed appearance (Fig. 5). Innumerable calcospherites were scattered throughout the white matter (Fig. 6). A fairly dense fibrous gliosis was also present, and was particularly marked around many of the cystic spaces (Fig. 7) and around the calcospherites. In preparations stained with Cajal's gold sublimate astrocytes were occasionally seen to arrange themselves radially around calcospherites (Fig. 8).

Many of the cysts contained a fine reticular network of collagenous fibres in which there were found a few scavenger cells and debris of blood corpuscles. A few of the scavenger cells gave the iron reaction with Turnbull's stain. Those cysts containing blood superficially resembled an angiomatous malformation, but nowhere was an endothelial lining or evidence of other coats of a vessel wall seen. Occasionally capillaries and calcospherites were found lying within the cystic spaces.

The sclerosed tissue between the cysts contained numerous proliferating astrocytes and oligodendroglial cells although their proportion varied in different parts

of the lesion (Fig. 9). A number of nerve cells of normal appearances were found in the less affected cortex, and an occasional nerve cell and a few nerve fibres could be seen even in parts showing dense glial proliferation.

*Postoperative Course.*—The child's convalescence was fairly smooth, and no further fits were observed. She returned to the Maudsley Children's Department on the twelfth postoperative day, at which stage she was alert, played contentedly with her toys, and was much quieter than before. She could still see objects to her right side, although theoretically a right-sided upper quadrantic homonymous hemianopia must be present. She was, however, too young to test on the perimeter. There was still slight weakness of the right lower face, but no other neurological signs were detected. When discharged to her home on the seventeenth day she seemed a naturally contented child and was talking more freely. Sodium phenytoin, gr.  $\frac{3}{4}$  twice daily, was prescribed for a prolonged period.

*Follow-up.*—She has since been re-examined on several occasions, the last being 14 months after operation

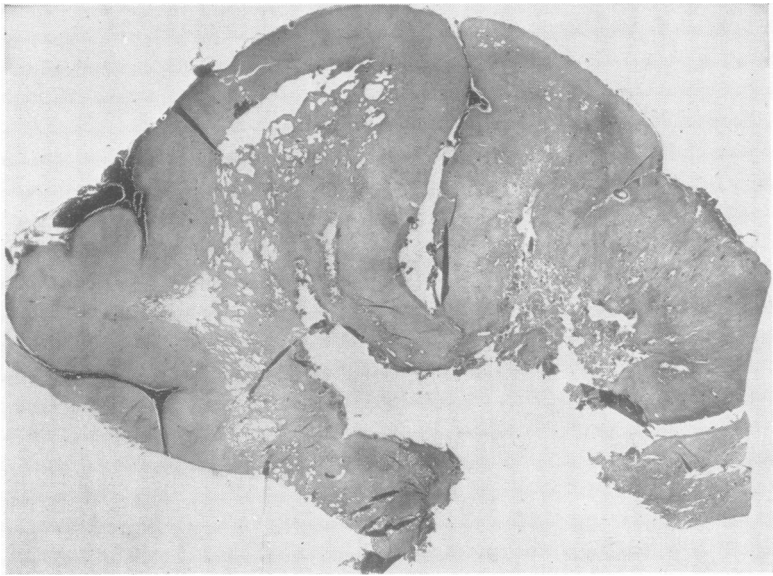


FIG. 5

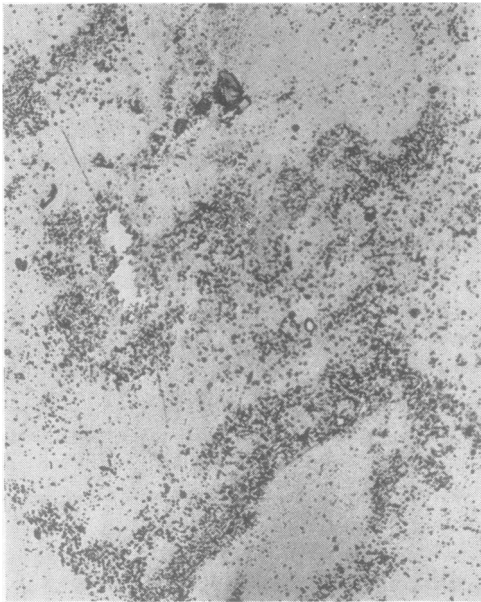


FIG. 6

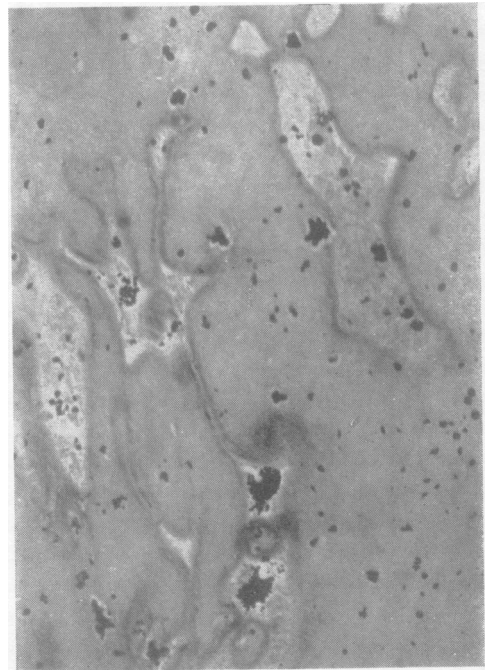


FIG. 7

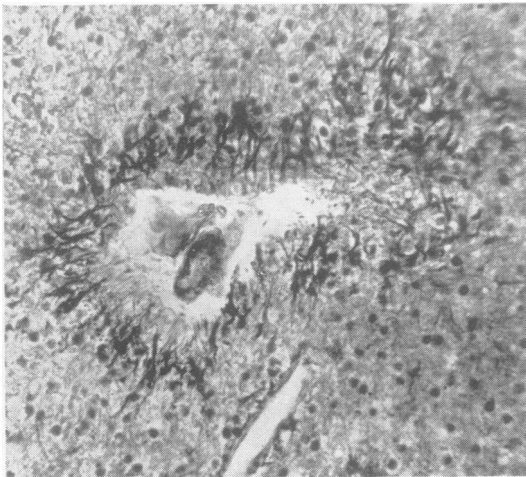


FIG. 8

FIG. 5.—Case 1: Coronal section through specimen showing honeycombed appearance owing to numerous microcystic spaces in the white matter. Haematoxylin-eosin,  $\times 3$ .

FIG. 6.—Case 1: Numerous calcospherites in the centre of the tumour. Haematoxylin-eosin,  $\times 5$ .

FIG. 7.—Case 1: Fibrous gliosis particularly marked around microcystic spaces which contain a fine reticular network. Holzer,  $\times 75$ .

FIG. 8.—Case 1: Radial arrangement of astrocytes around calcospherite. Cajal's gold sublimate,  $\times 235$ .

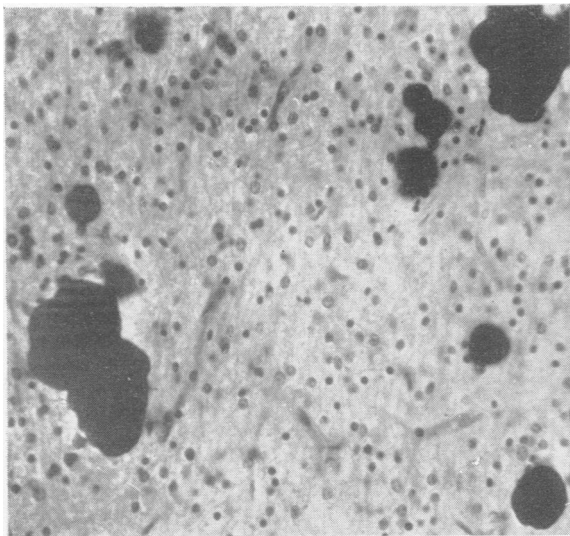


FIG. 9.—Case 1: Calcospherites, capillaries, and proliferating astrocytes and oligodendrocytes. Haematoxylin-eosin,  $\times 300$ .

Radiographs show no return of intracranial calcification. She has been completely free of epileptic attacks. Cared for by anxious parents and an attentive nanny, she is somewhat indulged, but on acquaintance she becomes friendly and responsive. She expressed herself clearly with short phrases and sentences, would name objects, and sing several tunes. Psychological tests were repeated at intervals, and Miss Newell found that when she was 3 years old her mental age on the Merrill-Palmer scale had risen to 2 years 1 month, and when she was 3 years 7 months her social age on the Vineland Social Scale was 2 years 6 months. Her mental age and social behaviour thus appear to be progressing at a constant rate, but are still approximately a year behind the average for her age. A letter from her parents two years after operation says she is still improving.

*Electroencephalography.*—E.E.G.s performed shortly after operation showed a normal appearance of the right cerebral hemisphere (Fig. 4). The area of relative inactivity still persisted in the left posterior inferior region and a few small spikes were seen on the edge of this area. A sleep record 14 months after operation showed only very doubtful flattening of the left posterior temporal region, and the record could be passed as within normal limits.

Clinically this child has been relieved of her epilepsy and temper disorders. However, she is still a difficult child to manage although her behaviour is better. Her mental standards are improving at a fairly normal rate, although her scoring is still well behind that of a normal child of her age.

The pre-operative electroencephalographic examination was very difficult in this child and conse-

quently was limited. The general impression gained was one of widespread and bilateral epileptic disturbance. Subsequently the restoration of an almost normal electrical rhythm to both cerebral hemispheres, after such a limited excision, is particularly intriguing, and adds substance to the view expressed by Krynauw (1950) on the basis of his hemispherectomy studies that the pathological activity of a limited area of abnormal tissue may have a profoundly disturbing influence on the functioning of the surrounding normal brain.

Case 2.—P. K., a girl aged 14 years, was referred to the Children's Department of the Maudsley Hospital (Dr. D. A. Pond) in March, 1951, by Dr. R. W. Tibbetts for investigation of an "atypical epilepsy and behaviour disorder". The investigations were interrupted several times because exacerbations of this behaviour disorder rendered her unmanageable, leading to her temporary transfer elsewhere. She was finally admitted to the Neurosurgical Unit for operation in June, 1953.

Her birth and early development had been normal, and there was no family history of epilepsy. She had been seduced at the age of 9 years, and on this her parents blamed her subsequent troubles, for shortly afterwards she had her first fit. In this one day she suddenly clutched her mother, said, "I feel funny", and went red in the face, there was no loss of consciousness, and a few seconds later she seemed all right again. During the next five months she had further similar attacks about twice weekly, and then more severe seizures also appeared. She herself said that these attacks usually began with a strange feeling in her abdomen which she found difficult to describe. Her parents said that she might cry out and appear dazed at their onset, and then within a few seconds she would lose consciousness and remain unresponsive for from one half to three minutes. Often she lay still, but at other times she would keep kicking her legs. Sometimes smacking movements of the jaws were observed, and sometimes also she was incontinent of urine. She was usually flushed in the early stages of the attack and pale towards the end. The patient added that if immediately the epigastric sensation appeared, she could concentrate her thinking on something else, she could often abort an attack. A potent way of doing this was by laying her head on her father's shoulder. Indeed she became unduly attached to her father, and he to her, and several psychiatrists who saw her throughout subsequent years felt this relationship in some way had much to do with her various symptoms.

About a year after their onset her fits reached a frequency of 15 to 20 major attacks a day, but then shortly afterwards lessened to one every few months. About this time her behaviour deteriorated markedly. She would become aggressive and violent for hours on end, a contrast to her former friendly and affectionate nature. She also became wilful, insolent, and vulgar, and at times would expose herself. As a result the family situation also deteriorated with much rivalry

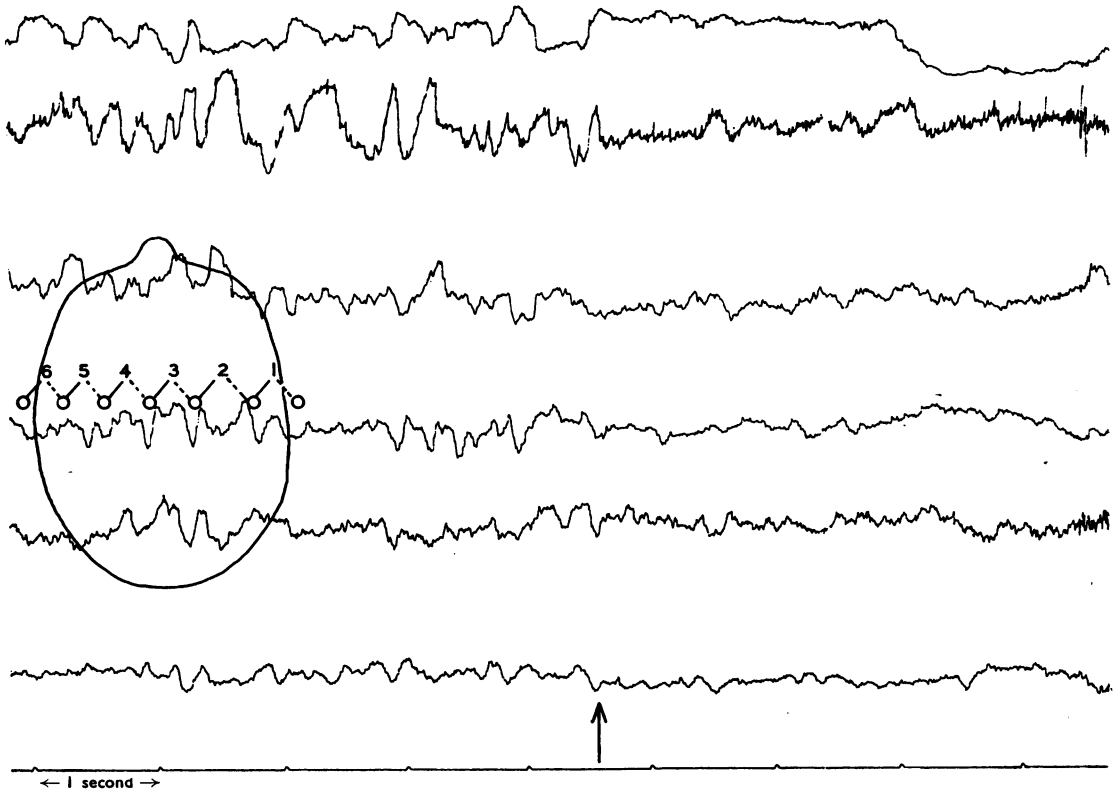


FIG. 10.—Case 2: Preoperative E.E.G. showing a focus of irregular slow waves in the inferior part of the right mid-temporal lobe. Sharp waves are also recorded from the same area. The arrow marks the onset of a brief seizure during which the patient flushed, gripped her throat, and exhibited irregular movements of her limbs. The seizure lasted approximately 30 seconds, the record and the patient's condition both rapidly returning to normal. Note the symmetrical generalized flattening of the record at the onset of the attack.

among the siblings and inconsistent handling by the parents. She was seen by many doctors and even had a short period in an epileptic colony. Hysteria was frequently considered, and the diagnosis of epilepsy was not settled for several years because her attacks were of a type unusual in childhood, were often precipitated by emotional outbursts, and were relatively unaffected by anticonvulsant drugs.

From April, 1951, she spent most of her time in various wards of the Bethlem Royal and Maudsley Hospitals, with periods at home to which she was taken by her father, who often allowed himself to be persuaded by the child that she was being maltreated. However, he always came begging us to take her back after a few days of her behaviour at home. On the wards her behaviour was at times aggressive and unpredictable but mostly she had an air of injured innocence and hostility, and she displayed a great capacity for playing off staff and patients against each other. Several unavailing attempts were made at individual psychotherapy. The only factor apparently related to her behaviour was the number of seizures, for a group of attacks such as she frequently experienced just before menstruation always

caused her to be subdued and manageable for a few days, whereas a single attack would have little effect. She averaged two or three attacks weekly in spite of regular and intensive anticonvulsive drugs.

The physical and neurological examinations were normal as were the visual fields, the cerebrospinal fluid, and the Wassermann reactions in blood and cerebrospinal fluid. Psychological tests showed that the Wechsler full-scale I.Q. was 79 (verbal 87 and performance 74). Nothing suggestive of organic disease was found in the sub-test score matter, nor in the Rorschach and other psychological tests.

*Electroencephalography.*—Several E.E.G.s had been taken in other hospitals before admission to the Maudsley Hospital. The outstanding characteristic of these records was the great variability in the degree of abnormality. Some records were quite normal, but those that were abnormal showed fundamentally the same focal disturbance, namely irregular sharp-and-slow-wave complexes in the right inferior fronto-temporal region. Fig. 10 shows the usual pattern obtained from the standard vault electrodes, as well as the onset of one of the

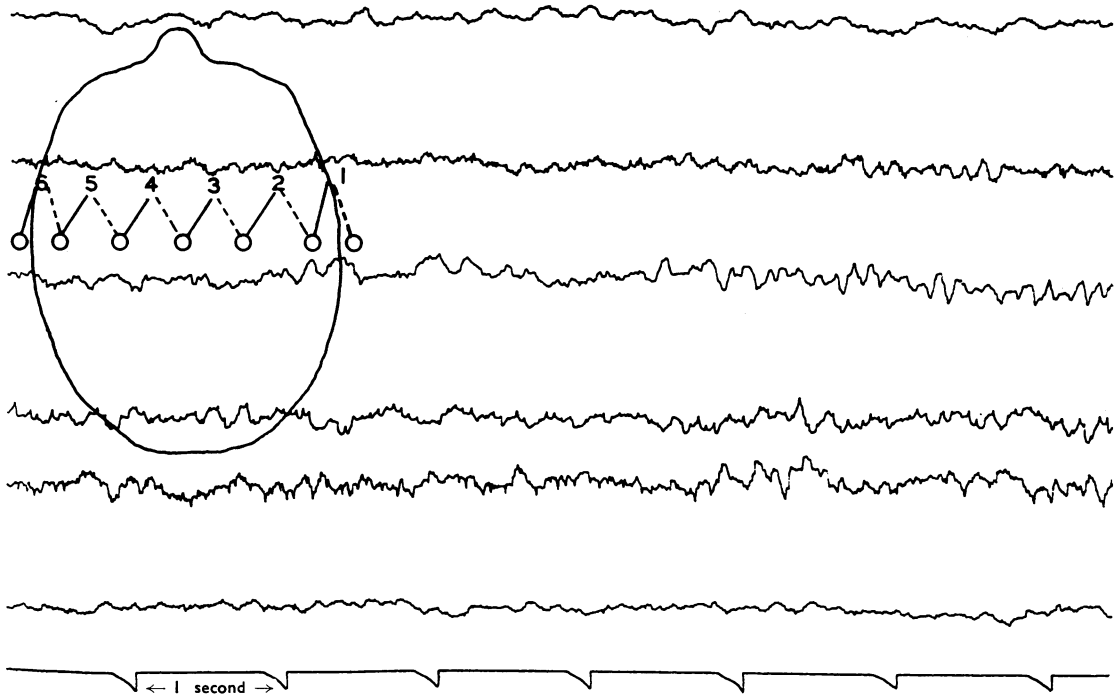


FIG. 11.—Case 2 : Post-operative record in the same position as the pre-operative one. Note the disappearance of the irregular slow activity and the increased amount of alpha rhythm.

typical short seizures in which the autonomic phenomena were so predominant. Besides the standard electrode positions, other placements with additional electrodes on the zygoma and over the temporal and frontal regions were tried in an effort to localize the area. The sharpest waves were obtained from the inferior surface of the temporal lobe by means of needle electrodes placed under the foramen ovale according to a new standardized technique ("sphenoidal leads"). Owing to her non-cooperation this particular examination was performed under light pentothal anaesthesia in the E.E.G. Department at Guy's Hospital just before the operation.

*X-ray Studies.*—Radiographs of the skull were normal, while a right carotid arteriogram showed a normal vascular pattern. Cisternal encephalography, however, showed that while both lateral ventricles were in their normal positions without any evidence of a shift or other abnormality, the right lateral ventricle was slightly smaller than the left (Fig. 12). The temporal horns, however, were symmetrical.

Here then was a young girl with epilepsy, often of akinetic type, but characterized by a "dreamy state" and associated with an epigastric aura.

Her fits and personality disorder were in a sense antithetic: when the fits became infrequent her behaviour disorder became worse. There were no focal neurological signs, the air-encephalographic findings were inconclusive, and the only definite clues implicating the right temporal lobe were the various electroencephalographic findings. The girl was often unmanageable at home, and she was facing certification and permanent retention in an institution. On the strength of the electroencephalographic findings we therefore decided to remove the right temporal lobe, performing the widest removal possible without incurring any disabling symptoms.

*Operation.*—Under general anaesthesia a right lateral craniotomy was performed, exposing the convexity of the temporal lobe and the adjacent portions of the frontal and parietal lobes. The surface appearances of the brain, including the inferior aspect of the temporal lobe, seemed normal. The temporal lobe anterior to Labbé's vein was then removed in one piece, the specimen including the temporal pole, the anterior 3 cm. of the superior temporal convolution, the inferio-lateral border of the lobe for a distance of 8 cm., the uncus, and the



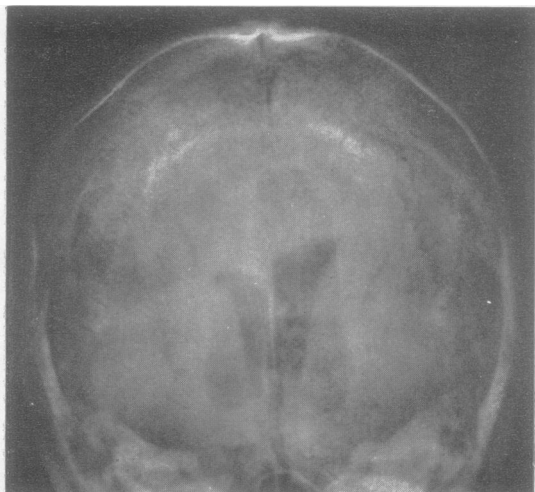


FIG. 12.—Case 2: Encephalogram showing that the right lateral ventricle is slightly smaller than the left.

anterior 3 cm. of the hippocampus (Fig. 13). The insula and roof of the temporal horn of the ventricle were left undisturbed.

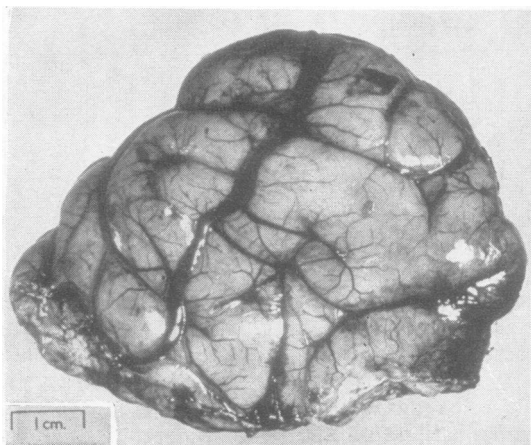


FIG. 13.—Case 2: Lateral aspect of removed temporal lobe. The specimen is inverted with the temporal pole on top.

*Pathology.*—The specimen was divided into three blocks and then was embedded in celloidin. An investigation by silver impregnation methods for which frozen sections were required was thus impossible. No abnormality was noted on macroscopic inspection.

Histologically the most striking change was seen in the region of the uncus. This region contained a lesion 1½ cm. in length and ¾ cm. in breadth containing numerous calcospherites and capillary blood channels, some of which were in the process of calcification (Fig. 14). Between the blood vessels and calcospherites which gave the Kossa reaction for calcium but did not stain with Turnbull's iron stain, there was seen a

tumour-like proliferation of astrocytes and oligodendrocytes (Fig. 15). Holzer-stained sections revealed considerable fibrous gliosis throughout the lesion, being particularly marked around the calcospherites and in the sub-pial layer. Many nerve cells of normal size

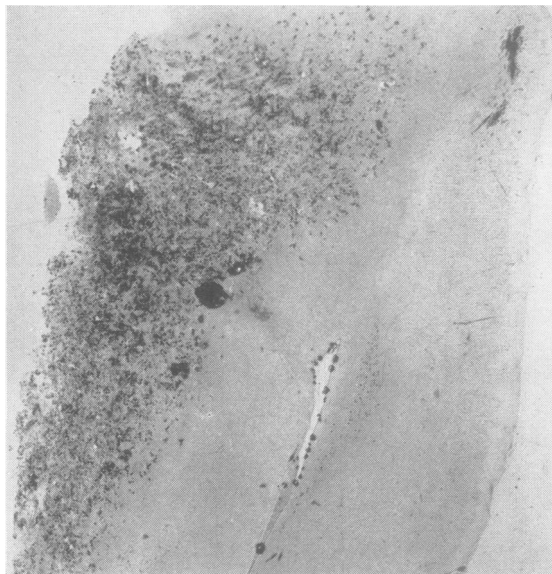
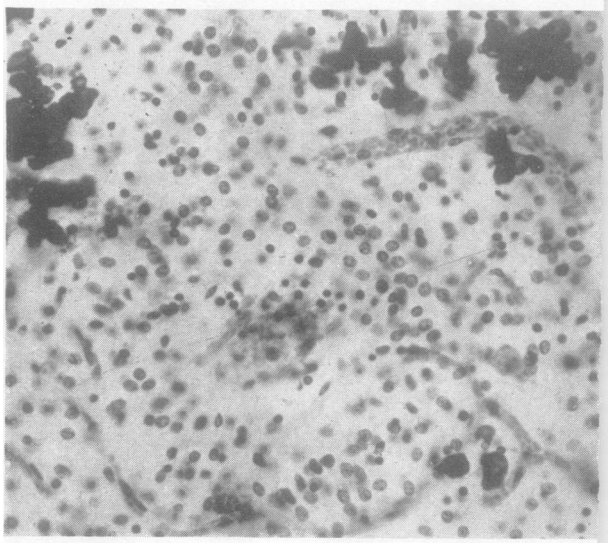


FIG. 14.—Case 2: Area in uncus region occupied by calcified lesion. Haematoxylin-eosin, × 6.

and appearance were dispersed throughout the lesion. Maldeveloped nerve cells and glial cells of the type found in tuberosc sclerosis and ganglioglioma were nowhere seen. Outside the main lesion the histological changes consisted of a patchy marginal gliosis and occasional calcification of capillaries. There was no sclerosis of Ammon's horn. The cortex showed no appreciable outfall of nerve cells, but the usual biopsy artefacts. The meninges were slightly fibrosed

FIG. 15.—Case 2: Calcospherites, capillaries, and proliferating astrocyte oligodendrocytes. A few normal nerve cells. Haematoxylin-eosin, × 30



and some of the meningeal blood vessels showed slight calcification.

*Post-operative Course.*—For some days the patient exhibited a partial ptosis of the right upper eyelid, evidently from some interference with the third cranial nerve, but no abnormality of oculomotor movements or of the pupillary resections. On the fourth post-operative day she had four transient focal seizures involving the left side of the face. Subsequently she had further focal fits on the sixth, seventh, and eighth post-operative days. Apart from these features her manner and behaviour were little different from that expected after a routine craniotomy.

By the second week a striking improvement was observed in her manner, for she had become quiet and cooperative. She now described the aura to her old attacks as a sequence of thoughts which would pass through her mind in rapid and jumbled succession for a few seconds before she lost consciousness. Sometimes these thoughts used to come and pass off without any loss of consciousness. She also denied ever having had the epigastric aura which pre-operatively she had described. Visual field testing showed that she had developed a slight left-sided, upper quadrantic, homonymous hemianopia; otherwise the neurological examination was still negative. Psychometric testing by Miss S. M. Cox showed a definite improvement in her intellectual abilities.

Test	Pre-operative	Post-operative
Wechsler : Verbal I.Q. . . . .	87	93
Performance I.Q. . . . .	74	87
Full scale I.Q. . . . .	79	89
Porteus maze . . . . .	89	107

She was transferred back to the Maudsley Hospital from the Neurosurgical Unit on the twentieth post-operative day, and soon afterwards she returned home with advice to take phenytoin, gr. 1½, twice daily.

*Follow-up.*—She was seen again nine months after operation, and reports were received from her parents, doctor, school teacher, and social service worker. All these testified that she was now a normal girl, well-behaved, getting on well at school, and completely free of any suggestion of epilepsy.

*Post-operative Electroencephalography.*—For the first few weeks after operation a considerable asymmetry was present with a focus of fairly regular slow waves in the posterior temporal region which could be reasonably ascribed to the operative interference. Over-breathing increased the asymmetry and the response to photic stimulation was also asymmetrical. Eight months later the record had become more normal (Fig. 11), and the effects of photic stimulation symmetrical. At no time have any of the anterior temporal sharp-and-slow-wave complexes been seen.

This child, like the first, has recovered from her epilepsy and temper disorders, and is now developing as a normal girl. It is interesting that in both cases there was the same histological lesion, the

only difference in the clinical findings being that in the first the lesion showed as a calcified shadow visible in x-ray films, whereas in the second case the calcification was not sufficiently dense to be so visualized. Indeed, in the second case no clue to the nature of the lesion was forthcoming from the history, clinical examination, pre-operative investigations, or even from the operative findings. The underlying pathology was not realized until histological studies of the resected temporal lobe had been made.

**Discussion**

These two case histories present several points of interest, particularly those concerning the nature of the tumour, its clinical effects, and the response to surgery. They raise the question as to how frequently other patients, children as well as adults, may not harbour the same causal lesion, and how this lesion can be recognized and diagnosed.

*Nature of Tumour.*—The interpretation of the pathological change is difficult. The combination of intensive calcification, increased vascularity, and glial sclerosis recalls the “hemangioma calcificans” described by Penfield and Ward (1948). These authors described five cases of a circumscribed calcified lesion occurring in one temporal lobe of patients whose ages varied from 21 to 63 years, and who had suffered from epilepsy for from one and a half to 30 years. In four of the cases the lesion was successfully removed at operation with relief of seizures, while the fifth case was studied at necropsy. Penfield and Ward made the point that these lesions were either very slowly growing or stationary and that, although they might indent the temporal horn of the ventricle, they did not produce a shift of the ventricular system as visualized by air-encephalography. In all their five cases the lesion was visible in plain x-ray films, and they stressed this fact as one of diagnostic importance. However, they mentioned an additional case, in which a small mass, that histologically was of the same type, had been removed from the temporal lobe of a man aged 39 years. Although this mass likewise had been causing epilepsy, they eliminated the case from further consideration because no calcification had been detected radiologically.

In Penfield and Ward’s cases evidence of angiomatous malformation was described, but this is missing in our cases unless it has been obscured by intensive calcification. It is noteworthy that Fig. 2 of Penfield and Ward’s paper somewhat resembles Fig. 7 of our paper, which we have interpreted as microcyst formation and not as an angiomatous malformation. There is a certain resemblance

between these lesions and the lesions which Geyelin and Penfield (1929) and more recently Alexander (1953) have described as "endarteritis calcificans cerebri". The main difference is that the latter condition has a familial incidence and usually leads to multiple lesions. Again, especially in our first case, there were also points of resemblance to astrocytoma and oligodendroglioma, in particular to their combination described by de Busscher and Scherer (1942) as oligodendro-astrocytoma. The particular tumour which they described occurred in a patient aged 57 and took its origin from the septum pellucidum growing into the anterior horn of the lateral ventricle. However, as no further similar observations have been recorded the nosological position of this tumour is not clearly defined.

In our two cases there was no positive evidence that the lesions had been caused by inflammation, by vascular disturbance, or by trauma. However, as Penfield and Ward considered their concept of "hemangioma calcificans" as of rather tentative value, allowing a provisional grouping of cases with somewhat unusual lesions, we feel it is permissible to add our two new cases to this group.

**Clinical Aspects.**—The clinical effects of the lesions seen in our two cases were those of epilepsy arising within the temporal lobe, and were obviously the result of their local situation and not of their pathological characteristics. In this respect their effects were similar to those of other lesions of the temporal lobe, such as scars, areas of softening, neoplasms, and racemose angiomas (Jackson, 1888 and 1898; Penfield and Erickson, 1941). As is so common with temporal lobe epilepsy (Lennox, 1951) both our patients showed conspicuous personality and behaviour disturbances, and indeed it was largely on account of these disturbances that they were referred for operation.

The paper of Penfield and Ward (1948) indicates that calcified lesions of various sorts are not uncommon with temporal lobe epilepsy. Besides the five cases of "hemangioma calcificans" already quoted, they encountered during the course of six years seven other cases of calcified lesion including racemose angioma, calcified intracerebral haematoma, and calcified tuberculoma. Their experiences therefore support the view that every patient with chronic epilepsy should have the skull radiographed, especially if the clinical picture is that of temporal lobe epilepsy. The disclosure of a calcified intracerebral shadow should then point the way to further investigations, and to consideration of surgical treatment if adequate treatment with

anticonvulsant drugs fails to control both the seizures and the behaviour disorder.

Our second case, however, as well as the additional case of Penfield and Ward (1948), shows that these lesions may not be sufficiently calcified to be visualized in plain x-rays, neither may they be demonstrated by air encephalography or by cerebral arteriography. The clinical picture then is that of temporal lobe epilepsy without any clinical, neurological, or radiological evidence of a structural lesion. We do not know how frequently this occurs.

It is in such cases that electroencephalographic investigation becomes of paramount importance, for it may be on the electroencephalographic evidence alone that the surgeon is called upon to remove the offending portion of the brain. The problem of diagnosis in such cases is part of the larger problem of recognizing in unicate or psychomotor epilepsy which temporal lobe contains the epileptic focus. Special electroencephalographic techniques are often required, including sleep records and special placement of the electrodes. Our second patient was one of a dozen subjects with intractable epilepsy in whom a unilateral or predominantly unilateral "spike-discharging" focus was demonstrated in the anterior temporal region and then was removed at operation. The other cases will be reported separately, but in each some histological change was found in the temporal lobe. However, we have no certain information as to the frequency with which the calcified lesions described in this paper occur and are responsible for symptoms.

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