EPILEPSY DUE TO SMALL FOCAL TEMPORAL LESIONS WITH BILATERAL INDEPENDENT SPIKE-DISCHARGING FOCI

A STUDY OF SEVEN CASES RELIEVED BY OPERATION

BY

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It is widely known that in epilepsy of temporal lobe origin the epileptic discharges may be widespread, affecting both temporal lobes and even more widely both cerebral hemispheres (Gibbs, Gibbs, and Fuster, 1948; Jasper, Pertuiset, and Flanigin, 1951; Ajmone Marsan and Stoll, 1951), but what is not so well recognized is that a unilateral temporal lobe lesion can also on occasion give rise to bilateral temporal lobe discharges. It is the purpose of this paper to report seven cases in which small focal lesions (glial hamartomas, angiomas, or other neoplasms) too small to distort appreciably the ventricular contours on pneumoencephalography were yet associated with bilateral, seemingly independent, temporal lobe discharges, and to describe the changes which occurred in the E.E.G. patterns after operation. The various cases are listed according to the site of the lesions. These cases were culled from 75 consecutive cases of intractable temporal lobe epilepsy operated on before the end of 1957, as described in a previous communication (Falconer, Hill, Meyer, Mitchell, and Pond, 1955).

Case Histories

Case 1: STA.* (Hamartoma with Oligodendrogliomatous Characteristics).—A 19-year-old boy, referred by Dr. D. Hill, had suffered from the age of 5½ years from sudden losses of consciousness often beginning with an epigastric sensation and a taste. Occasionally an organized visual sensation was also present. Such attacks usually lasted 10 to 30 seconds, recurred once or twice a day, and had proved resistant to drugs. Staring, fidgeting, and masticatory movements had been observed during attacks. He never had major convulsive seizures (grand mal). His birth had been an instrumental one, and his intellectual progress had always been retarded. At the age of 12½

years he was placed in an institution because of backwardness and antisocial behaviour. No abnormality was found on neurological examination. Air encephalography disclosed an asymmetry of the two temporal horns, such that the right horn was slightly reduced (Dr. R. D. Hoare), but in many centres the appearances would have been considered within normal limits. The cerebrospinal fluid was normal. His I.Q. measured by the Wechsler full scale was rated at 68 (V.S. 80, P.S. 63). The family history was negative, and both parents were highly intelligent persons.

Several pre-operative E.E.G. studies with routine scalp electrodes and sphenoidal electrodes under Pentothal narcosis showed three independent foci of spiking activity, a major one over the right temporal convexity and two minor ones from the left and right sphenoidal electrodes. In addition there was some reduction of barbiturate-induced fast activity beneath the right temporal lobe. The left sphenoidal focus sometimes spread to the left convexity.

At operation in February, 1956, a 7 cm. right anterior temporal lobectomy was performed, after confirmation by electrocorticography that there were spike discharges in the temporal lobe below the Sylvian fissure but not above this. He made a good post-operative recovery, apart from a series of focal motor seizures involving the left face in the second post-operative week. The lesions in the resected temporal lobe have been described by Cavanagh (1958, Case 4) as a multifocal glial lesion (hamartoma with oligodendrogliomatous characteristics) involving the uncus, amygdala, and the hippocampal and fusiform gyri.

Since operation this patient has been followed for four years. He exhibits a complete left homonymous hemianopia. He has had no fits or auras since leaving hospital, and he has been off drugs for two years. He has become cheerful and social, and he dresses impeccably. He works in a book store, and goes to dances.

The post-operative E.E.G.s were interesting. One month after operation there was some bilateral wave-and-spike activity, while the contralateral sphenoidal electrode still showed independent spiking. One year later the contralateral spiking had disappeared, but ipsilateral sharp waves were present in the posterior temporal

*Our colleagues in publishing their several papers dealing with various aspects of our material on temporal lobe epilepsy have taken as a convention that each case is designated by the first three letters of the patient's name. This convention facilitates references from one paper to another.

FIG. 1.—Case 2. Pre-operative sphenoidal E.E.G. under Pentothal narcosis shows frequent independent spike discharges at both sphenoidal electrodes. Barbiturate-induced fast activity reduced on the right side.

FIG. 2.—Case 2. Post-operative sphenoidal E.E.G. under Pentothal narcosis six years later fails to show spike activity. The presence of fast activity in channel 1 is due to the craniotomy.

region behind the area of excision. Four years later no spike activity was present.

Case 2: MUN (Hamartoma with Oligodendrogliomatous Characteristics).—A 35-year-old man, referred by Dr. J. P. Dewsbury, while serving as a regular army officer during 1942, first developed attacks of psychomotor epilepsy characterized by epigastric and cephalic sensations with derealization lasting only a few seconds. Masticatory movements as well as confusion had been noted by witnesses, and he averaged one to two attacks a day in spite of medication. He had never had grand mal. A mild psychosexual character disorder developed, and in 1949 he was invalided out of the army. Thereafter he was unemployable, in spite of high intelligence (Wechsler F.S. 138, V.S. 137, P.S. 139) and in spite of several attempts at rehabilitation. The neurological examination, cerebrospinal fluid, radiography of the skull, air encephalography, and right carotid angiograms were all normal.

The pre-operative E.E.G. studies showed frequently firing spike discharges at the right sphenoidal electrode conducted to the convexity. There were also less frequent but independent spike discharges at the left sphenoid electrode (Fig. 1). In addition the barbiturate-induced fast activity was reduced beneath the right temporal lobe (sphenoid-ear channel) and over the right temporal convexity. A Metrazol-induced seizure was inconclusive.

At operation under local analgesia in November, 1954,

electrocorticography showed frequent firing in the inferior temporal region with some reduction of normal activity (Dr. G. Pampiglione). Depth electrodes in the region of the roof of the temporal horn were also very active. A 7 cm. right anterior temporal lobectomy was performed. Subsequently he made a good post-operative recovery, with only a minimal left upper-quadrant homonymous hemianopia. Dr. J. B. Cavanagh (1956, Case 3) reported that the resected temporal lobe contained multifocal hamartomas with oligodendrogliomatous characteristics in the inferior and fusiform temporal gyri and in the amygdala and uncus.

Since operation he has been followed up for six years. Throughout he has been free of fits except for one isolated psychomotor attack in the sixth year after he had been off drugs for several years. He now holds a responsible post on the Stock Exchange and has married.

A post-operative E.E.G. made one month after operation disclosed no spike activity in either cerebral hemisphere, but at 12 months an isolated, ipsilateral sharp wave was observed. Subsequent records up to six years after operation have been satisfactory (Fig. 2).

Case 3: SIN (Oligo-astrocytoma).—A 2½-year-old girl, referred by Dr. K. Cameron, of normal birth and negative family history, had been subject to outbursts of temper for no apparent reason since the age of 14 months. Shortly afterwards minor turns characterized by vacancy, fluttering of the eyelids, nystagmus, and sometimes in-

continence, appeared. These could last from a few seconds to 10 minutes, and could recur as frequently as six to eight times daily in spite of medication. Simultaneously with the appearance of fits, her intellectual development ceased. She had no attacks of grand mal. Neurological examination showed slight weakness of the right lower face, but was otherwise negative. The cerebrospinal fluid was normal. Intelligence testing proved impossible. Radiographs of the skull showed an irregular mass of calcification about $5 \times 3 \times 2$ cm. in the posterior inferior temporal region, while air encephalography showed this to be situated beneath a somewhat dilated but not otherwise disturbed temporal horn. The pre-operative E.E.G. under Seconal-induced sleep showed irregular wave and spike discharges over both cerebral hemispheres, right greater than left, but barbiturate-fast activity was clearly reduced over the left hemisphere.

At operation in February, 1951, a partial temporal lobectomy ($7 \times 5 \times 3$ cm.) was carried out including some of the surrounding brain tissue as well as the tumour. The tumour itself was described as an oligo-astrocytoma by Dr. A. L. Woolf, and later reported as such by Cavanagh (1958, Case 7). Her post-operative course has since been one of steady progress. A battery of intelligence tests in the two and a half years subsequent to operation gave I.Q.s ranging from 63 to 77. The psychologist reported two and a half years after operation that she was potentially above an educationally sub-normal level. We have been unable to obtain recent formal intelligence testing, but she is now at a normal school, although working with girls a year younger. She exhibits a complete right homonymous hemianopia.

Follow-up examination after nine years has disclosed a cheerful, alert girl, who has had no fits or auras since operation. Electroencephalographs performed one year, two years, and nine years after operation have failed to show any spike activity.

Case 4: LEW (Capillary Angioma).—A 43-year-old woman, referred by Dr. D. Hill, started experiencing fits 12 years earlier. Her initial attacks were grand mal. Later attacks consisted of dazedness for from one to two minutes, accompanied by clonic movements of the right lower face and right upper limb. She said that they were ushered in by an epigastric sensation and occasionally by a nasty smell. After attacks she sometimes performed such automatisms as dressing and undressing, rearranging of flowers, etc. The frequency of attacks varied, but averaged two or three a week. Over the years she had become paranoid and aggressive. Neurological examination was negative. Skull radiography, cerebrospinal fluid, and air encephalograms were normal. Wechsler full scale I.Q. was 101 (V.S. 104, P.S. 99). The pre-operative E.E.G.s showed an actively firing spike focus at the left sphenoidal electrode conducted to the convexity, and a less frequent but independent focus at the right sphenoidal area. An E.E.G. performed about four years earlier, however, had shown a purely left-sided focus.

In February, 1953, a 6 cm. left anterior temporal lobectomy was performed, after electrocorticography had confirmed the presence of a focus in the left inferior temporal region. The resected temporal lobe was

reported by Prof. A. Meyer as showing a small capillary angioma in the uncus region. She subsequently had a smooth convalescence, and has been followed up for seven years. In her seventh year she had two akinetic fits, associated with loss of consciousness, incontinence, tongue-biting. She had been off medication for many years. Her husband reported her personality as much improved.

Her E.E.G.s repeated five days after operation showed no spike activity, and frequent E.E.G.s over the next seven years have failed to show paroxysmal activity.

Case 5: STE (Angioma).—A 40-year-old woman, referred by Sir Charles Symonds, with a normal birth and family history, had suffered from frequent psychomotor attacks since the age of 16 years. Often they were ushered in by epigastric and sometimes also by olfactory sensations. In most attacks she would stare, retch, and fumble, being afterwards amnesic for the attack which might last up to one to two minutes. The earliest attacks were of the grand mal variety. Her present attacks varied in frequency, occurring up to four times daily during her menstrual periods, but with remissions which were seldom longer than two or three days. For the past seven years she had displayed numerous paranoid aggressive outbursts towards her husband and son, as well as towards strangers, and she had had frequent conflicts with the police. Two years before her present admission, at another hospital, radiographs disclosed a shell of patchy calcification deep in the left temporal lobe. (A left carotid angiogram was normal as was a ventriculogram.) Neurological examination was negative, and E.E.G. examinations reported as unsatisfactory because of the patient's poor cooperation. A left craniotomy was performed and an incision 3 cm. long made in the line of the inferior temporal convolution. A mass $6 \times 5 \times 4$ cm. was removed composed of calcified material, fresh black clot, and discoloured brain tissue. It was subsequently reported as an angiomatous malformation. Her surgical recovery from this operation was satisfactory, but no improvement in fits or personality occurred. Her husband left her, and she became a serious social problem. When admitted to our unit her personality was as described. Her fits seemed frequent. No neurological abnormality was found. Radiographs of the skull no longer showed abnormal calcification. Air encephalography, however, showed some dilatation of the left temporal horn, but the left carotid angiograms were still normal.

Pre-operative E.E.G.s, repeated on several occasions, showed bilateral temporal lobe disturbances. Sharp waves and spikes with some slow-wave activity were seen at the left sphenoid electrode spreading to the convexity, but the most active spiking was at the right (or contralateral) sphenoidal area, also spreading to the convexity. This latter activity was independent of the right side. No obvious asymmetry of barbiturate-fast activity was noted, but assessment was difficult owing to the previous craniotomy.

As a lesion had previously been found on the left side the craniotomy was reopened in October, 1953, disclosing a large operative cavity in the left temporal lobe, even

FIG. 3.—Case 6. Pre-operative Pentothal-sphenoidal E.E.G. shows majority of spikes at right sphenoidal electrode (channels 13 and 14). Some are reflected to channel 9.

although the more active spiking was on the other side. The remains of the anterior 8 cm. of the temporal lobe were removed, including the uncus, anterior 3 cm. of the hippocampus (Ammon's horn), and the amygdala. Professor A. Meyer reported that there were still remnants of angiomatous malformation in the fusiform and hippocampal gyri, and some proliferation of macroglial cells in sectors h3-h5 of the hippocampus. During the post-operative period she exhibited a slight nominal dysphasia, and she required considerable psychiatric after-care. Her personality improved considerably, as did her liability to epileptic attacks. Her home situation has mended itself, and her husband states that she now has not more than four epileptic attacks in any one year, even although she takes no drugs. The follow-up period has now extended over seven years.

A post-operative E.E.G. one month after operation

FIG. 4.—Case 6. (a) The upper trace three weeks after operation shows no spikes, but some 3 to 4 c.p.s. activity in the contralateral right fronto-temporal region, as well as some post-operative 1 to 2 c.p.s. activity in the left posterior temporal region. (b) The lower trace three months later is normal.

failed to reveal any spikes. Similar findings were obtained six years later.

Case 6: PIN (Hamartoma with Oligodendrogliomatous Changes).—A 23-year-old man, referred by Dr. D. Hill, had suffered from psychomotor epileptic attacks since the age of 13 years, averaging from five to 20 attacks every month in spite of medication. The aura was one of undue familiarity with his environment and also micropsia, and was followed by pallor, staring, humming, and fidgeting with his hands for 30 to 60 seconds. He had also had about 10 grand mal attacks. He spoke with a stammer

but was not dysphasic. Neurological examination was normal. The Wechsler full scale I.Q. was 112 (V.S. 118, P.S. 111). Radiographs of the skull and air encephalograms revealed a small calcified mass in the left temporal lobe situated beneath a normal temporal horn about 7 cm. behind the temporal pole.

Several E.E.G. examinations were performed, and these revealed bilateral temporal independent spike activity in both inferior temporal areas, the right or contralateral side being much the more active (Fig. 3). Each focus of spikes would at times spread to the convexity. Two epileptic attacks were induced, one with intravenous Metrazol and one with intravenous Megamide, and on both occasions the fits appeared to arise in the left temporal lobe.

The problem then arose as to which temporal lobe was at fault. The right temporal lobe was the more active electrically. However, the fact that the induced attacks arose in the left temporal lobe, which was the site of a calcified nodule, led us to undertake a left-sided craniotomy. At operation electrocorticography demonstrated electrical firing to be most pronounced in the anterior temporal region, including the amygdalar region. Electrical stimulation in the region of the calcification produced no clinical effect, but stimulation of the amygdala produced an amnesic attack lasting seven and a half minutes. An 8 cm. anterior temporal lobectomy was performed. Subsequently, he had a smooth post-operative convalescence, and Dr. J. B. Cavanagh reported the lesion as a small gliomatous hamartoma with oligodendrogliomatous characteristics occupying parts of the fusiform and hippocampal gyri. He was left with an upper quadrantic field defect.

FIG. 5.—Case 7. Pre-operative Pentothal-sphenoidal E.E.G. shows multiple spikes at both temporal areas, left more than right.

FIG. 6.—Case 7. Post-operative Pentothal-sphenoidal E.E.G. three years later shows no spike activity. Reduction of activity in channels 5, 10, 13, and 14 is due to the temporal lobe resection.

Since then he has been followed up for nearly two years. During that time he has been on medication, and has had an occasional minor seizure. The E.E.G.s taken three weeks after operation showed some unexplained slow activity on the right side, but no spikes. When repeated three months and one year after operation, the E.E.G.s were normal (Fig. 4).

Case 7: GUL (Meningioma of Meckel's Cave).—A 48-year-old woman, referred by Dr. D. Hill, for 10 years had had frequent sudden attacks of pallor and confusion lasting a few minutes, in which she would sometimes repeat stereotyped phrases that bore no relation to her surroundings. There was no remembered aura. Such attacks occurred three to four times a week, and were

resistant to anticonvulsants. There was also a rather vague deterioration of memory. Neurological examination was negative, and air encephalograms suggested some mild compression of the right temporal horn. Intelligence testing showed a Wechsler full-scale I.Q. of 107 (V.S. 107, P.S. 107).

Electroencephalograms showed spike foci at both inferior temporal areas, left more than right, and in addition there was some irregular slow-wave activity at 1 to 2 c.p.s. in the right temporal and right central regions (Fig. 5). This led us to operate on the right side.

At operation in July, 1955, a glioma was suspected, especially as the convolutions towards the temporal pole seemed slightly swollen, and corticography here disclosed slow waves. An 8 cm. anterior temporal lobectomy was therefore performed. After the lobe was excised a small conical meningioma 2.3 cm. diameter by 1.5 cm. high was disclosed coming from Meckel's cave and burrowing into the anterior parts of the hippocampal and fusiform gyri. The cortex of the resected temporal lobe showed Alzheimer type of glial nuclei in the inferior temporal and fusiform gyri (Dr. J. B. Cavanagh).

Subsequently, she was followed up for four years, and during that time a slow but progressive dementia has become apparent. Doubtless she now has Alzheimer's disease. Fits, however, have been infrequent. Since operation E.E.G.s over a period of three years have shown no spike activity at the right sphenoidal area (Fig. 6), and on one occasion only one or two doubtful sharp waves at the left sphenoidal region.

Discussion

The salient features of these seven cases are summarized in Tables I and II. They are all examples of long-standing temporal lobe epilepsy with bilateral temporal lobe spike discharges and have been either cured or substantially relieved by a unilateral temporal lobectomy. Each case had a small focal lesion (gliomas or glial hamartomas, four; angiomas, two; meningiomas, one), and, after temporal lobectomy, spike activity in the post-operative E.E.G. records disappeared on both sides. The lesions involved various parts of the temporal lobe with the possible exception of the hippocampus (Ammon's horn) and the superior and middle temporal gyri. The structures chiefly involved were the fusiform and hippocampal gyri and the amygdala and uncus. It is attractive to believe that these focal lesions were the epileptogenic agents. Favouring this view are the improvement that followed surgical removal in all cases and the fact that in the resected specimens no alternative pathological cause was apparent. The question, therefore, arises as to how these lesions led to bilateral spike discharges, and the mechanisms involved. It is of course well known that unilateral lesions or electrical stimulations of one cerebral hemisphere can give rise to electrical activity with epileptic characteristics in the opposite cerebral hemisphere. The most striking example is afforded by

cases of infantile hemiplegia associated with epilepsy when the presumably normal hemisphere as well as the damaged one are the sites of abnormal electrical rhythms (Krynauw, 1950). The removal of the diseased hemisphere by hemispherectomy is usually followed both by cessation of fits and by a return of normal electrical patterns in the remaining hemisphere. It is not, however, widely realized that small lesions of the type we have described in this paper can also cause bilateral discharges, although this has been previously reported (Kennedy and Hill, 1958; Falconer and Cavanagh, 1959). Yet there is also experimental evidence that this can happen.

We understand that Gastaut (1953) has observed the development of bilateral, seemingly independent temporal lobe spikes in cats in which alumina cream has been injected into one temporal lobe. Vigouroux, Naquet, and Gastaut (1953) in one particular cat injected the alumina cream in the left amygdala, provoking epileptic seizures within two months, and producing a left temporal lobe spike focus and later a right temporal focus as well. The left temporal area, including the lesion, was then excised and the seizures ceased, but the right temporal focus remained for several weeks. Similar observations were made by Penfield and Jasper (1954) in monkeys in which alumina cream had been injected into the motor cortex on one side. They pointed out that spikes which were transmitted from the primary focus were abolished by section of the corpus callosum, but that secondary independent spikes persisted, suggesting that a true autonomous focus had been established in the homologous area of the opposite hemisphere, presumably due to long persistent bombardment from the primary side. In one particular monkey careful excision of the primary focus containing the alumina cream did not immediately arrest the firing on the opposite side, but a month later the firing was found to have disappeared.

Frost, Baldwin, and Wood (1958) and also Poblete, Ruben, and Walker (1959) have studied in monkeys the propagation of after-discharges between the temporal lobes after electrical stimulation. The first group of authors showed that repetitive electrical stimulation of the second temporal gyrus or of the amygdalar complex was frequently followed by after-discharges in the contralateral amygdala or contralateral second temporal gyrus, but that this propagation could be interrupted by section of the anterior commissure. Poblete and his colleagues came to somewhat similar conclusions. There is thus experimental evidence of close connexions between homologous areas of the two temporal lobes.

Most authors who have operated on patients with temporal lobe epilepsy have stressed that the results are better if before operation the abnormal electrical

TABLE I

CLINICAL AND PATHOLOGICAL DATA OF CASES ARRANGED ACCORDING TO SITUATION OF LESION (PRE-OPERATION DATA IN PRESENT SERIES)

Case	Sex Age	Duration of Epilepsy (years)	Pathology	Situation of Lesion							Type of Epilepsy		I.Q.
				Middle Tempo- ral Gyrus	Inferior Tempo- ral Gyrus	Fus- gyrus	Hipp- gyrus	Amyg- dala	Uncus	Am- mon's Horn	Grand Mal + Psycho- motor	Auras	
1 STA	M 15	14	Oligo- dendrogloma	○	○	■	■	■	■	○	—	Epigastric, organized visual, taste	68
2 MUN	M 35	11	Oligo- dendrogloma	○	■	■	○	■	■	○	—	Epigastric, cephalic, strangeness	138
3 SIN	F 2½	1	Oligo- astrocytoma	Not available	■	■	Not available	Not available	Not available	Not available	—	? Vertiginous	277
4 LEW	F 43	12	Angioma	○	○	○	○	Not available	■	○	+	Epigastric, smell	101
5 STE	F 40	24	Angioma	○	○	■	■	○	○	■	+	Epigastric, smell	—
6 PIN	M 23	9	Oligo- dendrogloma	○	○	■	○	○	○	○	+	Microspia, familiarity	112
7 GUL	F 48	10	Meningioma	○	○	■	■	○	■	○	—	Nil remembered	107

TABLE II

E.E.G. OPERATIVE, AND THERAPEUTIC DATA (OPERATION OR RESULTS IN PRESENT SERIES)

Case	Pre-operative E.E.G. Data					Operation			Post-Operative E.E.G. Data (Spikes)	Therapeutic Result		
	Ipsilateral			Contralateral								
	Convexity (Spikes)	Sphenoidal (Spikes)	Barbiturate Fast	Convexity (Spikes)	Sphenoidal (Spikes)	Extent (cm.)	Side	Follow-up (Years)		Epilepsy	Person-ality	Work
1 STA	++	+	Reduced	±	+	7	R	4	Nil	5	4	4
2 MUN	++	++	Reduced	+	+	7	R	6	Nil	4.5	4	5
3 SIN	+		Reduced	++		7	L	9	Nil	5	4	4
4 LEW	+	++	?	-	+	6	L	7	Nil	4	3	5
5 STE	±	+	—	±	++	8	L	7	Nil	4	3	5
6 PIN	±	+	—	±	++	8	L	2	Nil	4	4	4
7 GUL	±	+	—	±	++	8	R	5	Nil	4	4	4

In assessing therapy, 5 ranks as excellent, 4 as very good, and 3 as improved.

activity is confined to the resected temporal lobe. The literature on the subject has recently been reviewed by Bloom, Jasper, and Rasmussen (1959-60), and this has also been our own experience (Falconer *et al.*, 1955). Bloom and his colleagues at Montreal, for instance, showed that unilateral temporal lobectomy in a group of 29 cases with bilateral discharges produced successful results, *i.e.*, complete or almost complete abolition of seizures, in only 24% of the cases, whereas in a much larger group of cases with strictly unilateral temporal lobe spiking two out of every three cases were benefited.

Yet the problem remains that certain cases with bilateral spiking can be benefited by unilateral operation, and also that cases with unilateral temporal lobe lesions can be associated with bilateral spiking. Bilateral spiking, therefore, does not necessarily indicate bilateral pathological changes as it is unlikely in the cases we have described that similar lesions were on the other side. This appears to hold true even for mesial temporal lobe sclerosis with Ammon's horn lesions. Thus out of our first 50 cases of temporal lobe epilepsy, 21 presented Ammon's horn

sclerosis (Falconer *et al.*, 1955; Kennedy and Hill, 1958) and of these 10 presented bilateral independent temporal lobe discharges, and in eight of them the seizures were abolished (Falconer and Kennedy, unpublished data).

Recently our group have shown that out of 70 cases of temporal lobe epilepsy submitted to unilateral temporal lobectomy for epilepsy on E.E.G. criteria in the presence of normal or only minimally abnormal air encephalograms (which in many centres would have been reported as normal), no less than 15 cases (21%) were due to small unsuspected glial malformations, other tumours, or vascular malformations (Falconer and Cavanagh, 1959). The present seven cases described in this paper (with one exception, Case 6) are taken from that series and illustrate some of the difficulties that arise in the diagnosis and localizations of temporal lobe epilepsy. The disappearance of the contralateral spiking may be delayed (Cases 1, and 7).

It is clear that even when there are bilateral independent spike-discharges in the temporal lobes there may be only a single lesion on one side. In such

cases the patient's seizure may be relieved by a unilateral operation. However, we have as yet no E.E.G. criteria that enable us to forecast with high accuracy where a unilateral lesion is present. There are, however, some pointers. Thus in three of the seven cases described here (Cases 1, 2, and 3) there was definite diminution of barbiturate-induced fast activity on the side of the lesion. In this respect these cases resemble the findings previously described by Kennedy and Hill (1958) in cases with predominantly unilateral Ammon's horn sclerosis. The predominance of spiking by itself is no guide, for in four of our cases (Cases 3, 5, 6, and 7) spikes were more frequent on the unoperated side, and yet the patients were benefited. Three of these cases, however, were selected because radiographs had shown a small calcified lesion on the side which was operated (Cases 3, 5, and 6). The remaining case (Case 7) had irregular slow-wave activity on the operated side. Even radiological evidence of unilateral temporal lobe atrophy is no certain guide, for in a case recorded by Falconer and Cavanagh (1959, Case WAK) there was a very active spike focus on the right side, slight but definite dilatation of the left temporal horn outside the range of the controls. In this case we chose to operate on the E.E.G. rather than the radiological data with a fortunate result in finding a small unsuspected oligodendroglioma. (This patient has been free of fits for six and a half years.) Probably the observance of a spontaneous fit during an E.E.G. examination is of lateralizing value, but such fits may rarely occur. Metrazol-induced activity in our experience can be fallacious, although sometimes helpful (unpublished data). Thus so far the best indication of a unilateral lesion when the radiographs are not conclusive (as in Cases 1 and 2) is a diminution of barbiturate-induced fast activity. In one case the presence of unilateral slow activity was the key to the lateralization (Case 7), while in another E.E.G.s taken several years earlier had shown a unilateral discharging focus (Case 4). With further experience of temporal lobectomy when there are bilateral discharges the criteria for anticipating a unilateral lesion and subsequent relief of seizures may become better crystallized.

Summary

The case histories are recorded of seven patients with intractable temporal lobe epilepsy, generally of long duration, whose electroencephalograms

disclosed bilateral, seemingly independent spike discharges in both temporal regions. The air encephalograms showed little or no abnormality, although in three cases a tiny calcified lesion was seen. Each patient was submitted to a unilateral temporal lobectomy for reasons given in the text, and in each a small focal lesion (glioma, hamartoma, capillary angioma, or meningioma) was later disclosed. Subsequently each patient was either cured or very greatly benefited as regards epilepsy, while the spike discharges disappeared, although sometimes after a period of delay. It is presumed, therefore, that the small focal lesions were the epileptogenic agents, and the possible mechanisms by which they caused bilateral spiking activity are discussed. This small group of cases represent an exception to the general rule that unilateral temporal lobectomy fails to benefit more than a minority of patients with intractable temporal lobe epilepsy and bilateral temporal lobe discharges, and therefore the recognition preoperatively of similar cases is considered. Provisionally, it seems that without radiological evidence of a lesion, diminution of barbiturate-induced fast activity using sphenoidal leads for recording is one of the best indications of a focal lesion, although unilateral slow activity may also be helpful.

We wish to thank the physicians who have referred their cases, Dr. Denis Hill for his cooperation in the E.E.G. data, Professor A. Meyer and Dr. J. B. Cavanagh for the pathological data, and the Medical Research Council for a grant for the assistance of several research associates. We also wish to thank the Department of Medical Illustration, Guy's Hospital.

REFERENCES

- Ajmone Marsan, C., and Stoll, J. (1951). *A.M.A. Arch. Neurol. Psychiat.*, **66**, 669.
- Bloom, D., Jasper, H., and Rasmussen, T. (1959-60). *Epilepsia (Amst.)*, 4 ser., **1**, 351.
- Cavanagh, J. B. (1958). *Brain*, **81**, 389.
- Falconer, M. A., and Cavanagh, J. B. (1959). *Ibid.* **82**, 483.
- , Hill, D., Meyer, A., Mitchell, W., and Pond, D. A. (1958). *Lancet*, **1**, 827.
- Frost, L. L., Baldwin, M., and Wood, C. D. (1958). *Neurology*, **8**, 54.
- Gastaut, H. (1953). *Epilepsia (Boston)*, 3 ser., **2**, 59.
- Gibbs, E. L., Gibbs, F. A., and Fuster, B. (1948). *Arch. Neurol. Psychiat. (Chicago)*, **60**, 331.
- Jasper, H., Pertuiset, B., and Flanigin, H. (1951). *A.M.A. Arch. Neurol. Psychiat.*, **65**, 272.
- Kennedy, W. A., and Hill, D. (1958). *J. Neurol. Neurosurg. Psychiat.*, **21**, 24.
- Krynauw, R. A. (1950). *Ibid.* **13**, 243.
- Penfield, W., and Jasper, H. (1954). *Epilepsy and the Functional Anatomy of the Human Brain*. Churchill, London.
- Poblete, R., Ruben, R. J., and Walker, A. E. (1959). *J. Neurophysiol.*, **22**, 538.
- Vigouroux, R., Naquet, R., and Gastaut, H. (1953). *Rev. neurol.*, **88**, 574.