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The Electroencephalogram in Parasagittal Lesions

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Parasagittal lesions of the cerebral cortex present a number of particularly difficult problems to the electroencephalographer. Epileptogenic foci, tumors, or traumatic lesions which leave scars on the mesial surface of the hemispheres are not only hard to localize by means of the EEG but may cause electrographic disturbances at a distance, making possible a false localization or interpretation. In other cases they may be strangely silent. Working in close association with Dr. Penfield has made it possible for us to observe not only the EEG preoperatively in a number of such patients but to verify the lesion and to obtain electrocorticographic findings which may shed some light on the cause of ambiguous EEG results.

In previous reports Penfield and Jasper (1946) and Jasper (1951) have drawn attention to the bilaterally synchronous rhythmic discharges which may appear in the EEG in patients with epileptogenic foci on the mesial surface of one hemisphere. These electroencephalograms may resemble closely those of the petit mal or combined petit mal ~ grand mal epilepsy. Similar EEG findings have recently been reported by Dell and Hecaen (1951) as probably arising from subcortical structures. Lennox and Robinson (1951) have recently drawn attention to the importance of the anterior cingulate region in the genesis of the wave and spike discharge, and some of the functional characteristics of petit mal seizures of the akinetic or myoclonic form. All of these studies point to the importance of the cortical areas on the mesial surface o the hemisphere (pericollosal) in the genesis of seizures which might otherwise be considered of subcortical or non-focal origin.

In this report we wish to summarize our preoperative clinical and electroencephalographic findings, and the results of operative exploration, electrical stimulation of the exposed cortex and electrocorticographic findings on selected patients with verified epileptogenic lesions involving the mesial surface of one hemisphere.

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MATERIAL and METHODS

This report is based upon detailed studies of 31 cases with parasagittal epileptogenic lesions. The ages of these patients ranged from 3 1/2 to 46 years; the majority (78 percent) were over 16 years of age. There were five cases between 6 and 15 years of age and only two cases under 5 years. Age of onset of attacks ranged from 1 to 44 years ~ the majority beginning after adolescence.

Electroencephalographic and electrocorticographic studies were carried out with the usual standard procedures using Grass ink-writers, but with specially built amplifiers for the work in the operating room. The 10-20 electrode position system of the International Federation was used for all electroencephalographic studies as shown in figure 1. Standard bipolar and ear reference derivations were used in addition to special placements of electrodes when indicated. Activation procedures included Metrazol, photic stimulation, hydration, sleep and hyperventilation as needed.

The etiology of seizures, as determined by the history and operative findings in these cases may be summarized as follows:

Etiology or Pathological Findings	*No*
Birth injury	6
Head injury	9
Brain tumor	7
Cortical atrophy or cicatrix, unknown cause	7
Calcification and gliosis unknown cause	2

In 17 of the 31 cases in this series there were no physical or neurological signs to aid in the localization of the lesion. In those with lesions in the vicinity of the sensorimotor cortex or intermediate frontal region hyperactive reflexes or paresis, awkwardness, or sensory deficit in the contralateral limbs pointed to the side of the lesion, while apraxia or hemianopsia was present in those cases with lesions in the parieto-occipital region.

SEIZURE PATTERNS

The most important advance in our understanding of seizure patterns to be expected from epileptic discharge aris-

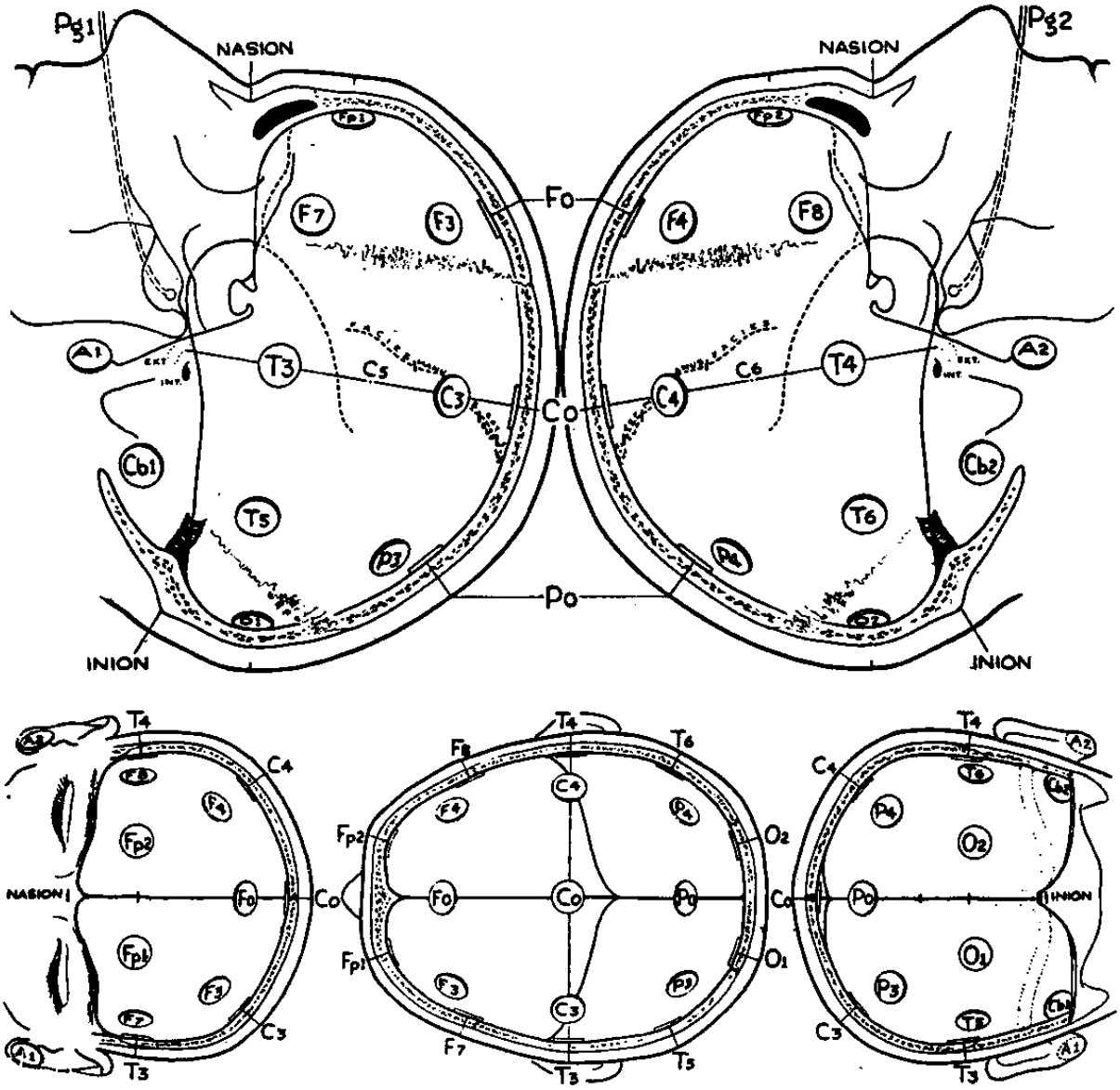

Fig. 1. Standard Electrode positions used throughout the present studies. This is the 10-20 system of placements of the International Federation which has now been adopted in many laboratories.

ing on the mesial surface of one hemisphere is the recent description of the functional characteristics of the supplementary motor region by Penfield and associates (1950-1951), and confirmed by Woolsey and Erickson (1951). In this area, lying largely on the mesial surface of the intermediate frontal region, just anterior to the precentral leg area, there is a separate motor representation of the body which is bilateral to some extent. From this same region or a portion of it, there is an area from which arrest of speech and voluntary movement can be elicited, or interrupted vocalization produced by electrical stimulation. Some forms of vague sensation referrable to the body as a whole, or to the autonomic system (palpitation etc.) appear also to arise from this area of cortex.

More recently it is becoming apparent that there may exist a supplementary sensory zone in the mesial parietal region, just posterior to the postcentral leg area. These areas, together, might be called the supplementary sensorimotor cortex, situated about as outlined in figure 2. Knowledge of the functional characteristics of these areas has aided greatly in the localization of epileptogenic foci in the cases under consideration here.

The initial phenomena and pattern of onset of seizures in these cases depended upon that portion of the mesial surface of one hemisphere which was the focus of onset of the attacks. For descriptive purposes we have divided the mesial cortex into five zones, omitting the temporal region since it is not under consideration in this study. These are: (1) the anterior frontal; (2) intermediate frontal; (3): Rolandic sensorimotor; (4) parietal, and (5) occipital areas, as shown diagrammatically in figure 2. The initial phenomena and subsequent patterns of attack which were most characteristic of each of these areas in the patients studied may be summarized as follows:

The above characteristic clinical features of seizure pat-

SEIZURE PATTERNS ARISING FROM PARASAGITTAL CORTEX

Initial Phenomena	Pattern
1. Anterior Frontal	
Lapse of consciousness, Staring. Possibly initial feeling of "dizziness" or light headedness with mental confusion before loss of consciousness in some cases.	Brief stereotyped irrational automatic behaviour or unconscious adversive tonic-clonic generalized convulsion.
2. Intermediate Frontal	
Sensory: Cephalic, throat, epigastric palpitation, general body sensation, subjective paralysis. Motor: Conscious adversion of head and eyes, tonic-postural movements, vocalization, aphasia or arrest of speech or movement with staring, forced thinking or confusion of ideas.	Contralateral tonic adversive movement followed by generalized tonic-clonic seizure.
3. Rolandic Sensorimotor	
Clonic movements of lower extremity or shoulder (1 case) Sensation in lower extremity.	Clonic movements of lower extremity progressing to contralateral then bilateral convulsion.
4. Parietal	
Bilateral sensation in extremities or body.	Generalized seizure or Retropulsion (1 case).
5. Occipital	
Visual sensation. Conscious adversion eyes and head	Generalized seizure

tern have aided greatly in the interpretation of the electroencephalograms in the cases under consideration. Examples will be given after consideration of EEG findings.

ELECTROGRAPHIC STUDIES

The most prominent feature of the electroencephalograms in 26 out of 31 cases was the appearance of bilaterally synchronous bursts of rhythmic spike and wave complexes. In most cases these appeared spontaneously, without activation. Although usually of somewhat irregular form and frequency, these EEG patterns often resembled closely enough the wave and spike pattern of petit mal epilepsy to cause difficult in differential diagnosis.

For purposes of discussion we shall assume that the wave and spike of petit mal epilepsy is a primary bilateral synchronous discharge, that is to say, it appears not to be related to a unilateral cortical focus, but may be of subcortical origin, projected symmetrically and synchronously to homologous areas of the two hemispheres. This type of discharge we shall call primary bilateral synchrony. On the other hand, a bilaterally synchronous discharge which can be shown to arise from a unilateral cortical focus we shall call secondary bilateral synchrony. The mechanism whereby a cortical focus can fire into subcortical structures and set off a projected secondary bilateral synchrony has been discussed with Penfield (1946) and by Jasper (1951). The basis for differentiation between primary and secondary bilateral synchrony will now be considered.

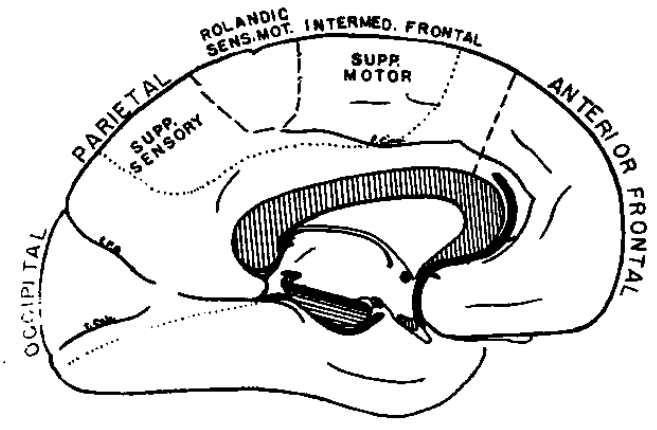

Fig. 2. Schematic diagram illustrating the subdivisions of the parasagittal area (broken lines). The supplementary sensory and motor areas outlined by dotted lines.

In these cases with verified parasagittal lesions the bilaterally synchronous discharges can usually be differentiated from primary bilateral synchrony by the frequency, form, regularity, symmetry, and order of successive bursts of activity. The waves in each burst are not usually repeated in a regular sequence of 3/sec. rhythm as is usualy seen in the classical petit mal discharge, though it may be similar. The rhythm may vary between 2 and 3.5 per sec., most commonly at 2-25 per sec. At times, frequencies of 4-6 per sec. appear. The form and relationship of the spike to the wave component of a spike and wave complex is less regular. Consistent asymmetry of voltage and wave form is usually observed over the homologous areas of the two hemispheres. Also there may be isolated focal discharges from one side alone, preceding a burst of bilaterally synchronous waves. Although we have observed a short burst of regular 3 per sec. spike and wave discharge in some cases, the slow spike and wave form, or sharp and slow wave complex was more common.

1. A Single Phase Reversal at the Midline

In addition to the above features, detailed localization studies reveal important differences in the EEG of cases

THE ELECTROENCEPHALOGRAM IN PARASAGITTAL LESIONS

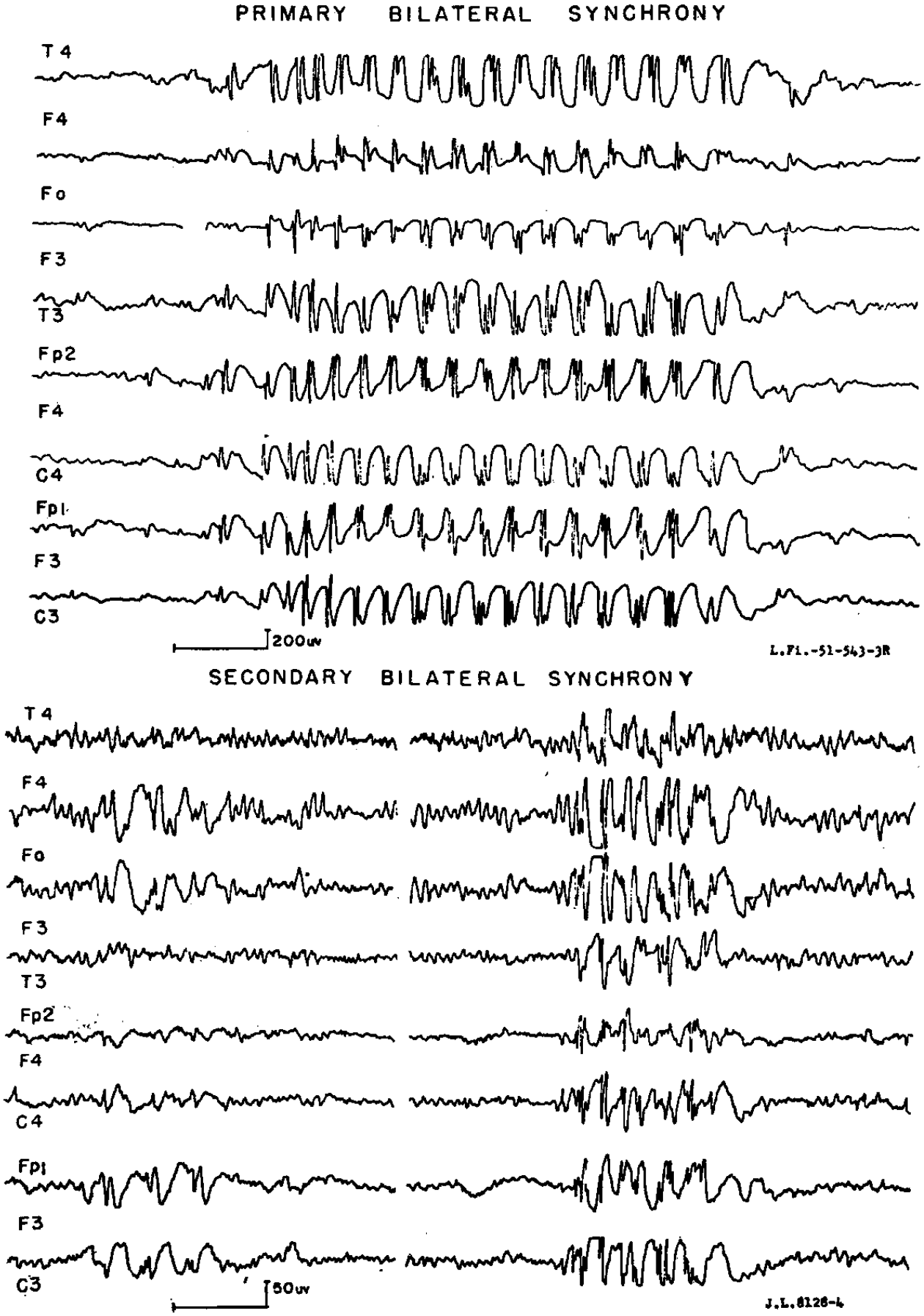

Fig. 3. Primary and secondary bilateral synchrony as observed in a coronal frontal line of electrodes right to left (first 4 lines) and A.P. lines of electrodes (right and left) in second 4 lines. Above: EEG of a 12 year old boy with petit mal epilepsy. Regular 3/sec. spike and wave activity symmetrical over both hemispheres with a double phase reversal over right (F4) and left (F3) frontal regions, resulting in a pseudo-reversal at the frontal vertex (F0). Note the two equipotential zones over F4, F0 and F3 - F0. Below: Irregular, asymmetric, spike and wave activity of case J.L. recorded over the same areas as above. The only phase reversal is observed over vertex at F0.

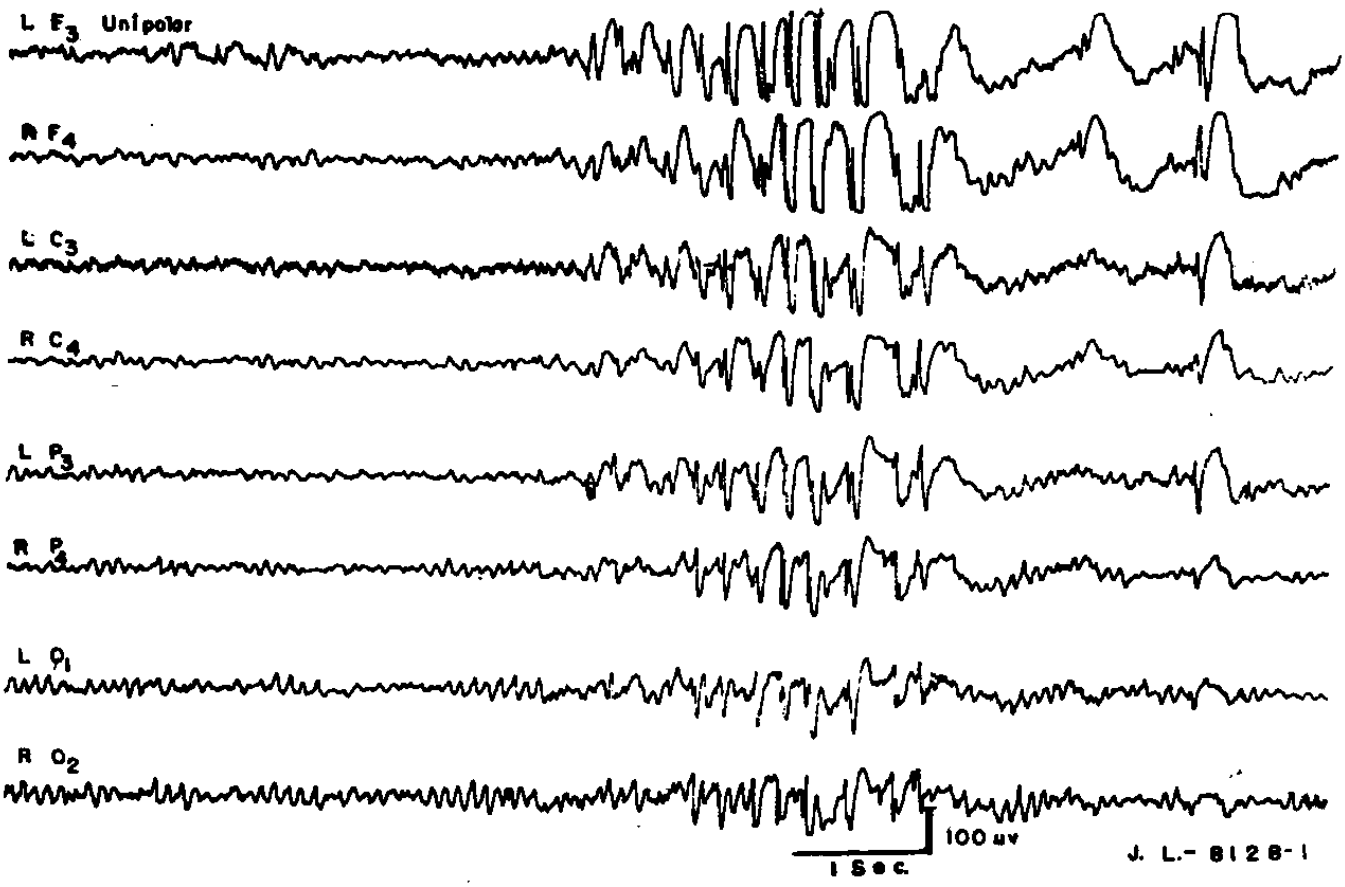

Fig. 4. Case J.L. Unipolar recording of bilaterally synchronous spike and wave activity. Note the spontaneous abnormality at F3 (left frontal) preceding a bilaterally synchronous burst.

with secondary as opposed to primary bilateral synchrony. A coronal line of closely spaced electrodes connected in successive pairs for phase reversal studies has proven to be a most useful method of examination when a parasagittal lesion is suspected. An example of the differentiation between primary and secondary bilateral synchrony wih this method is shown in the following case.

Case JL: 23 years, was admitted with complaints of seizures beginning at the age of 5 years. His birth and development was normal, there was a severe illness at the age of 4 years, probably an encephalitis.

The seizures began with a feeling which the patient described as a epigastric "faiblesse". This would be followed by "shadows" in the right visual field and adversive movements of the head and eyes to the right. The patient then would lose consciousness and experience a major seizure.

The preoperative EEG showed several bursts of paroxysmal high voltage spike and wave complexes synchronously over both hemispheres and maximal over frontal regions. Frequencies in these paroxysms varied from about 2.5 to 4/sec. Phase reversal studies showed the highest voltage activity to reverse at frontal vertex, over the standart electrode position Fo (Fig. 3). Spontaneous sharp wave activity, at times preceded a bilateral synchronous discharge over the left frontal region (Fig. 4). The presence of spontaneous bursts of irregular paroxysmal spike and wave activity with one phase reversal at the midline suggested a parasagittal lesion. The occasional sharp wave activity from the left frontal region, preceding some of the bilateral discharges was thought to indicate a focus in the left intermediate frontal region.

Neurological examination did not reveal any objective finding as to lateralization or localization of this focus.

Pneumograms revealed a general dilatation of the left lateral ventricles, chiefly in the parietal region.

A large osteoplastic craniotomy was done. Corticographic studies showed two foci of sharp wave activity over the intermediate frontal and parietal regions discharging independently of each other.

Electrical stimulation in the parasagittal region of the parietal cortex over an area where the convolutions were atrophic, produced part of the patients habitual seizures. After discharge, in the form of irregular spike and wave actvity, was also produced by stimulation of this area.

The excision was confined to the parietal area on the basis of corticographc findings, stimulation studies and gross pathologic findings.

The patient did not experience any attacks until he was discharged. However, the post-operative EEG showed the same paroxysmal activity as preoperatively, but no sporadic sharp waves were observed over the left frontal region.

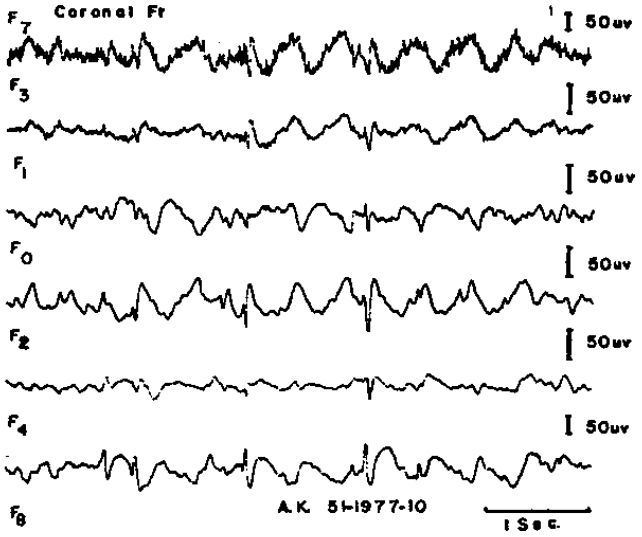

Fig. 5. Case A.K. Low voltage zone over F2-F4 in a case with a large atrophic lesion in the right intermediate frontal region. Maximal reversals were observed over frontal vertex and lateral frontal regions at F4.

The patient returned two years later with generalized seizures which were no longer preceded by a visual aura. The EEG showed irregular bifrontal wave and spike discharge, probably arising from the remaining atrophic cortex over the mesial surface of the left frontal lobe.

In this case the pneumoencephalogram and the objective findings at operation indicated a definite atrophic lesion of the cortex, most marked in the left parietal lobe but extending forward into the mesial frontal region. With such a diffuse lesion of the cortex of the left hemisphere following encephalitis one cannot rule out the possibility of a lesion also involving subcortical structures, which may have complicated the problem in this case.

2. Equipotential Area over Parasagittal Lesion

In the coronal line of electrodes the true petit mal wave and spike shows a double phase reversal, one over each midfrontal region, resulting in a pseudo phase reversal at the mid-line as shown in figure 3. There may be relatively equipotential zones on both sides of the mid-line, i.e., between F0 and F3 and between F0 and F4. In cases of bilateral synchrony secondary to parasagittal focus there is either a phase reversal at the mid-line (shown in the above example, J.L) or the one side of the mid-line near the site of the lesion. An equipotential zone usually lies on only one side of the mid-line directly over the parasagittal lesion.

An example of an equipotential zone over the site of an epileptogenic lesion in the right intermediate frontal region is as follows:

Case K.A.- 33 years. Had major and minor seizures since the age of 13 years following a head injury. In this major seizures there was first, raising of the left arm to the level of the shoulder, jerking of the left face and hand, followed by adversive movements of the head and eyes to the left. He then developed a major seizure, with tonic and clonic movements.

In his minor seizures, which he experienced up to seven times a day, there was staring, followed by repeating the words "pretty near".

The preoperative electrogram showed frequent spontaneous bursts of bilaterally synchronous 2-2.5/sec. slow waves and slow spike and wave complexes, maximal over frontal regions. Phase reversal studied over coronal frontal runs with closely spaced electrodes showed a maximal reversal at F4 and at times at Fo, with an equipotential area between F2 and F4 (Fig. 5). This was interpreted as evidence of an epileptogenic lesion including parasagittal portion of the right intermediate frontal region.

Pneumographic studies revealed a slight diffuse dilatation of ventricles, more marked on the right hemisphere and maximum in the right frontal region. Neurological findings revealed minimal left facial weakness, hyperactivity of the tendon jerks on the left. There was a relative smallness of the left foot and a scar on the right forehead.

An osteoplastic craniotomy was performed. A large severely atrophic area was found in the right intermediate frontal region. Corticographic studies revealed a sharp wave focus on the borders of the atrophic area. The habitual seizures were reproduced by stimulation. Some sharp wave activity was also observed over the convexity of the frontal lobe inferiorly.

The excision was restricted to the parasagittal region where maximal corticographic abnormality was observed and seizures were reproduced.

Post-excision records showed some slow waves with occasional low voltage spike components from the convexity where previously sharp waves were obtained.

The patient did not experience any seizure during the post-operative course. The patient was aphasic and there was a questionable akinetic state with perserveration and slowness of the movements of the left hand. However, there was complete recovery by the time the hyperventilation patient was discharged.

The post-operative EEG showed some 3/sec. bifrontal slow waves with hypenventilation, not considered of definite significance in the early post-operative period.

3. Low Voltage Area over Destructive Lesions

In some cases what may seem to be an equipotential area is actually a low voltage area due to the existance of an inert lesion such as a brain tumor lying beneath the recording electrodes. In such cases there may appear a spike focus adjacent to the electrically silent area, as shown in the following example in a patient with an astrocytoma in the left intermediate frontal region.

Case H.K., a 39 years woman with seizures since the age of 17. The seizures began wih a cephalic sensation. This was followed by flushing, the head and eyes then turned to

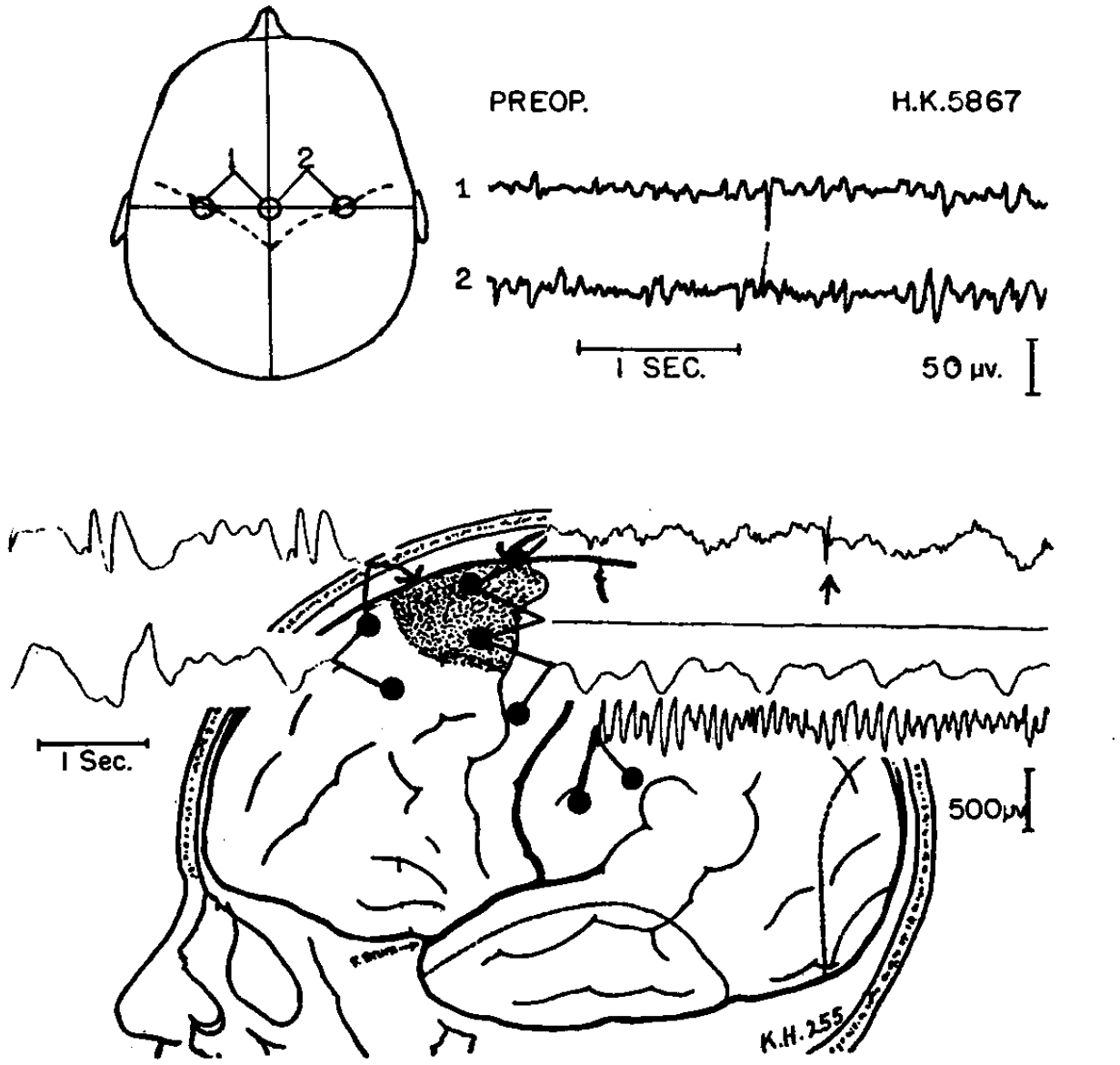

Fig. 6. C.H. electrographic and corticographic studies of parasagittal tumor. Above rapid spikes as seen in the EEG, reversing over central vertex. The broken lines represent the central fissure. Below: the location of the tumour is represented by the dotted area. The arrow points to the rapid spike activity as observed in the cortical electrogram. Delta waves were from the parasagittal intermediate frontal region around the borders of the tumour. The record over the tumour itself was silent.

the right while the right arm was raised to the level of the shoulder. During this period the patient felt as if her right limbs were paralyzed. This however, was not confirmed by examination during a seizure.

Two preoperative electrograms did not reveal a clear epileptogenic focus. In a third attempt, fine rapid spikes were observed reversing over the standard electrode position Co (Fig. 6) and the activity over C3 was relatively of lower voltage as compared with the homologous area of the right hemisphere.

At operation a tumor was found over the left intermediate frontal region. Electrocorticogram revealed a spike focus on the mesial surface of the cortex adjacent to the tumor. The record over the tumor was found to be electrically silent and slow waves and delta waves were observed over the cortex surrounding the tumor (Fig. 6). The patient's habitual seizure was reproduced with stimulation of the area where the spikes were recorded.

During the early post-operative period the patient was aphasic and a hemiparesis was observed. Both signs however, progressively improved. The patient did not experience any seizures in the post-operative course and the post-operative EEG showed no epileptiform activity.

Re-examination for years later showed a normal EEG and the patient has had no further attacks.

4. Higher Voltage Activity Controlateral to Focus During a Clinical Seizure

The asymmetry of bilateral synchronous abnormality and

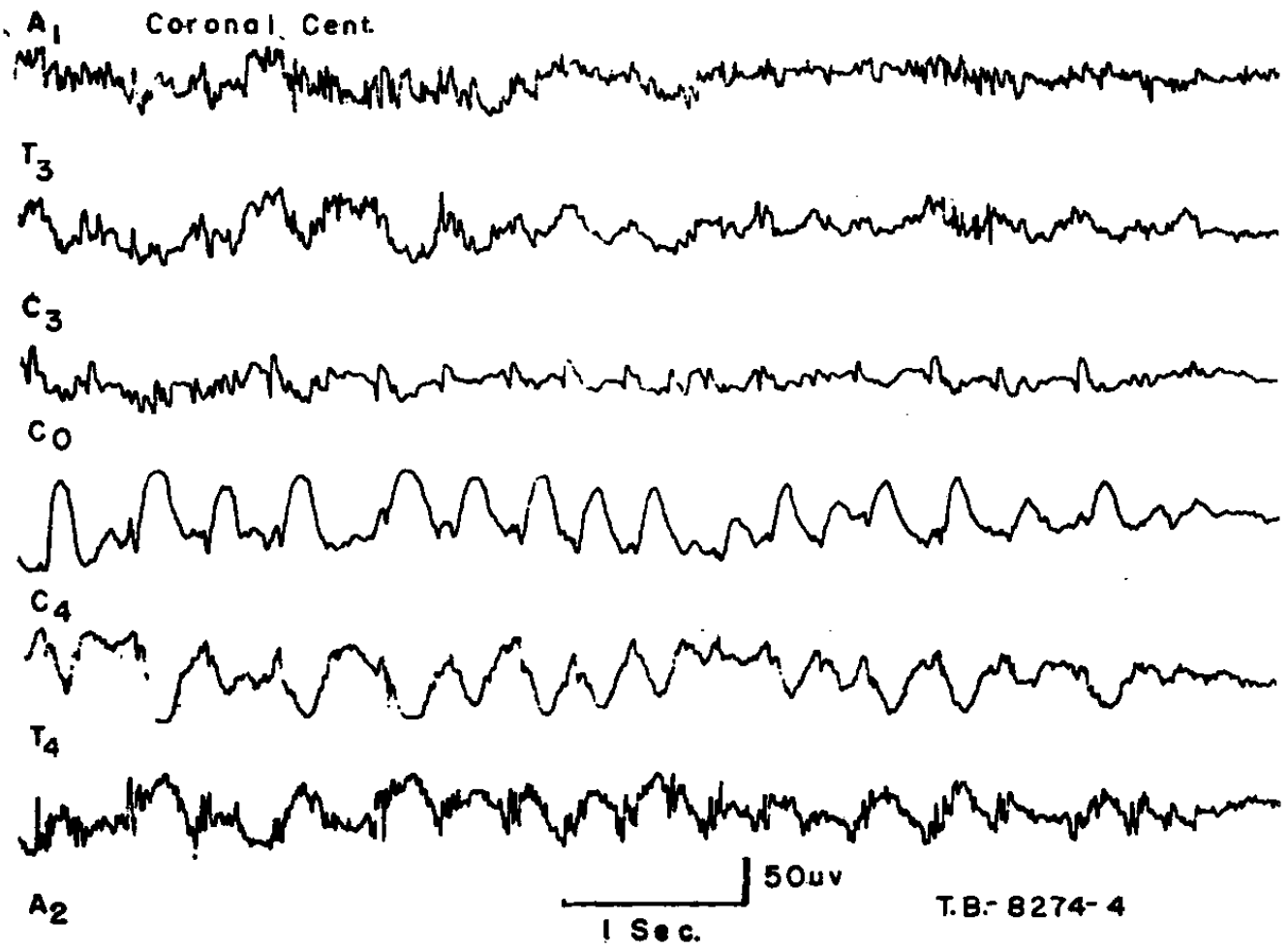

Fig. 7. Case T.B. EEG during an induced seizure in a coronal central derivation. The spike component is more marked over C3 where the tumour lies, whereas the slow wave component is of higher voltage over the hemisphere contralateral to the lesion.

the fact that it is of lower voltage over the site of the lesion may also be observed during the recording of a seizure. This is shown in the following example of a patient with an unclassified parasagittal glioma over the left precentral region extending to the intermediate frontal area.

Case B.T.- 43 years. Had major and minor seizures since 5 years. The major seizures were controlled by medication, while the minor seizures continued almost every day. The seizures began with a tingling sensation and pain in the right foot gradually rising to the face, arm and body. This would last about 2 min. and be followed by a feeling of thightness over the right side. The patient felt weak over the attacks.

Preoperative EEG showed some low voltage sharp waves over the left central region. A seizure was reproduced with hyperventilation during a central coronal record. This was characterized by 2.5/sec. spike and slow wave activity with phase reversals over the central vertex. The spike component was more prominent over the left central region followed by a low voltage slow wave, whereas the slow waves were of higher voltage over the right side (Fig. 7). These results were interpreted as being consistent with an epileptogenic focus over the left central parasagittal region.

Neurological examination revealed a marked weakness of the right leg, active knee and ankle jerks on the right side with planter extension. There was also a bilateral papilloedema.

Pneumographic studies showed evidence of an expanding lesion over the left fronto-parietal area.

An osteoplastic craniotomy was done and the tumor excised. Electrocorticography was carried out only after the excision of the tumor. No abnormality was observed from the electrodes placed deep within the area of excision. There were some sharp waves inferior and anterior to the excision in frontal region.

The patient did not experience any seizures during the post-operative course. There was however, an aphasia and a hemiplegia both of which began clearing by the 10th post-operative day and had markedly improved by the time the patient was discharged.

The above case illustrates a frequently observed EEG finding in patients with parasagittal epileptogenic lesions, namely that the higher voltage discharge may appear on the side opposite the lesion itself.

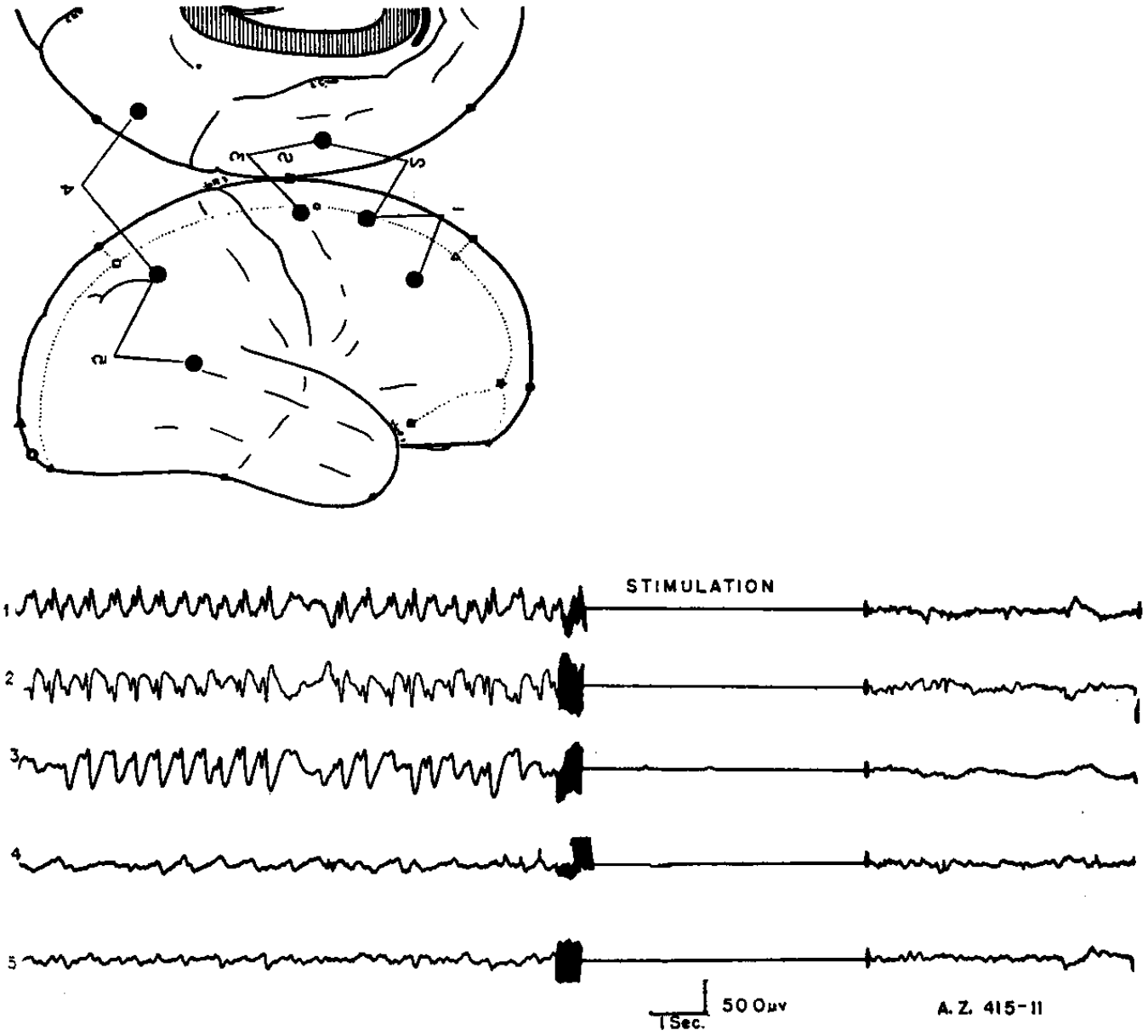

Fig. 8. A.Z. Suppression of the spike and wave activity observed over the anterior head regions by stimulation of the supplementary motor region at point S.

5. Suppression at Onset of Attacks

An important electrographic feature of the lesions originating in the supplementary motor region is the suppression 1* of the electrical activity preceding and/or during an induced seizure. In cases where the resting record is characterized by definite abnormal activity, this abnormality is suppressed; whereas in cases where the resting record does not give any clear abnormality (electrically silent seizures H.J.) the seizure begins with a suppression of the background activity. The suppression can also be observed in the corticogram with electrical stimulation, particularly in the supplementary motor region.

1* The word suppression is used here to indicate a sudden decrease in voltage of the electrical activity of the cortex. It should be clearly distinguished from other processes, such as spreading depression. Which have often been confused with it (Sloan and Jasper 1950).

An example of suppression in the corticogram produced by electrical stimulation is shown in a case with a left mesial parietal astrocytoma and atrophy over the left supplementary motor region.

Case Z. A., a young lady of 21 years, had major and minor seizures since the age of 11 years. Her seizures began with a stiffness in the right arm and leg. The patient then stared and the right arm raised to the level of the chest. This was followed by the extension of both arms. There were then adversive movements of the head and eyes to the right and vocalization. The patient remained conscious throughout the seizure. She heard herself vocalize but could not speak.

The preoperative EEG showed sharp waves and sharp and slow wave complexes over the left frontal region. With hyperventilation bursts of bilaterally synchronous slow waves and sharp-slow forms were observed. In a coronal

frontal recording this bilaterally synchronous activity showed phase reversals over the standard electrode position F0 and F3, with a low voltage equipotential zone between F1 and F0. These results were interpreted as evidence of a parasagittal epileptogenic focus over the left intermediate frontal region.

There were no positive neurological findings except a questionable smallness of the right hand.

Pneumograms revealed a calcified area over the left parietal region with dilatation of the left lateral ventricle in the parietal region.

At operation a tumor was found over the left parietal parasagittal area. The corticogram showed a continuous slow spike and wave activity of about 2.5-3/sec. over the mesial aspect and convexity of the intermediate frontal region. This abnormality was not observed from the parietal region. The patient's aura and her habitual seizures were reproduced by stimulation of the mesial intermediate frontal region. With stimulation over the supplementary motor region (5 in Fig. 8) a suppression of this spike and wave activity for about 15 seconds was observed.

The parietal tumor was then excised. Post-excision records showed no change in the spike and wave activity recorded over the intermediate frontal region. This region then was excised and the second post-excision record seemed to be entirely normal.

The patient did not experience any seizures during the post-operative course. There was however, a transitory motor aphasia and a right hemiparesis as well as a grasp reflex elicited in the right hand.

The post-operative EEG showed no spontaneous abnormality. With hyperventilation, bilaterally synchronous 3/sec. waves were observed, but these were though to be insignificant for this early post-operative period (19 days).

In our experience suppression at the onset of an attack has been observed only when the parasagittal focus was located in or near the supplementary motor region, or in the intermediate frontal region. It has not been observed with foci in other portions of the parasagittal cortex, though it is even more frequently found in epileptogenic focii of the anterior temporal region. More complete studies of the phenomenon of suppression in epileptic patients are being prepared for a later report. It has been observed sufficiently frequently in this series of patients to present another important complication in the interpretation of the electroencephalograms of patients with parasagittal lesions in the intermediate frontal region. Such foci do not seem to involve the anterior cingulate gyrus as might have been expected from experimental studies of suppressor areas in primates (Bailey et al 1944).

6. Intracranial Recording

In some cases with epileptiform activity of a secondary bilateral synchronous form, the above mentioned EEG features may be absent and the lateralization consequently, very difficult. The clinical pattern also may not be of

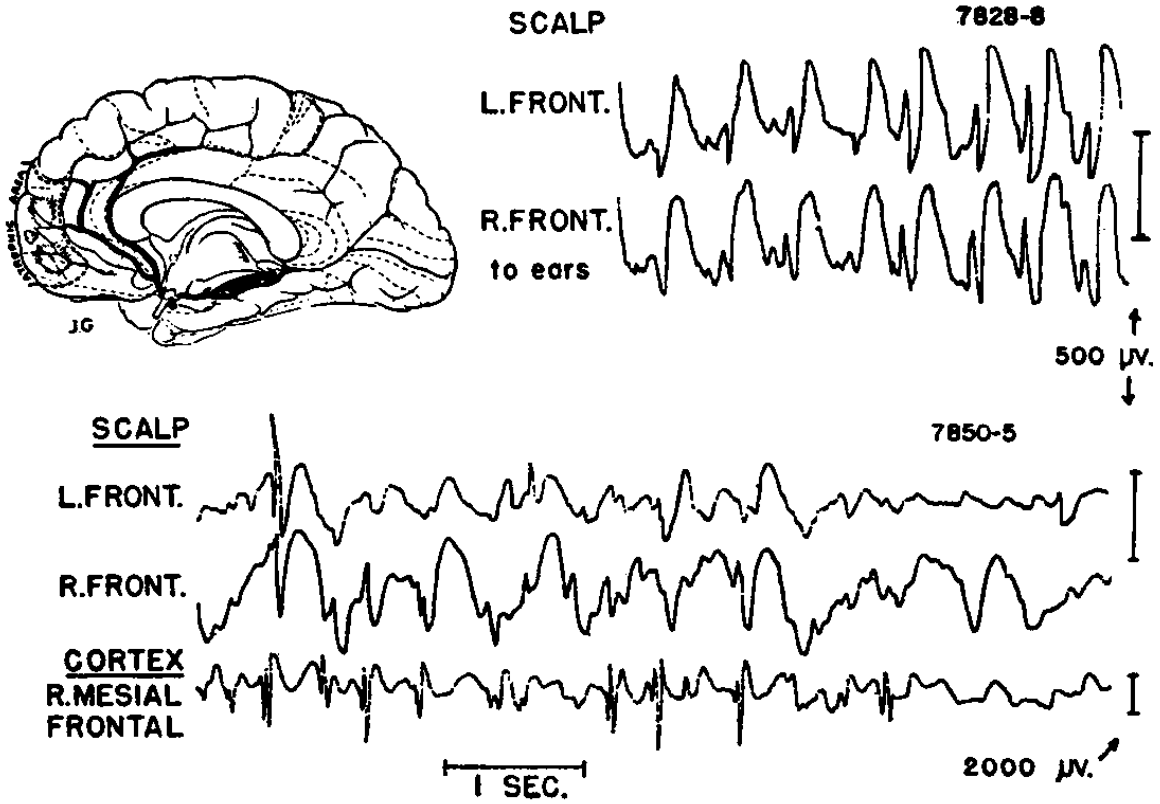

Fig. 9. Case J.G. The area of atrophic gyrus is shown above. The heavy broken lines indicate the area of excision. The first two lines represent the findings in the first EEG. The last 3 lines are representating the simultaneous recording from the scalp electrodes over the left and right frontal regions; and the intracranial recording from the mesial aspect of the right frontal region.

assistence as the lateralization to one hemisphere. Then the only means of lateralizing the focus is to record with intracranial electrodes. An example of this is given below in a patient with a large atrophic lesion over the right mesial anterior frontal region, adjacent to the falx.

Case G. J., a girl of 14 years, had seizures begining at the age of 10. She had suffered a head injury. Her minor seizures were characterized by lapses of consciousness, during which she stared straight ahead and then took a deep breath and move about in a confused manner. She then usually slept for about one and a half to two hours. The minor seizures were observed to be followed by very marked sweating. The major seizures also came without warning. There were adversive movements of the head to the left. It was reported that at times the head would initially turn to right for a short while and then swing back to the left. Eyes turned upward. This was followed by twitching of the face and shivering of the body.

Electrographic studies elsewhere, and our own initial observations, showed bilaterally synchronous, irregular 2.5-3/sec. waves and wave and spike complexes. Phase reversal studies over coronal frontal derivations showed reversals over both hemispheres. Re-examination was made with direct cortical leads leading from both hemispheres. This showed spike and sharp wave activity maximal from the electrode on the mesial surface of the right frontal lobe (Fig. 9), where a large area of atrophy was found at operation. This spike activity was followed by bilateral synchronous discharges from the scalp electrodes.

X-ray of the skull showed calcification of the falx. Pneumograms did not reveal any significant abnormality.

Neurological examination did not reveal any sign as to localization or lateralization.

Exploration of both frontal lobes was carried out. There was a calcification of the falx adjacent to the atrophic area on the inferior mesial frontal convolution as shown in figure 9. Corticograms revealed sharp waves and spike and wave activity over both hemispheres. The atrophic convolution on the right side was removed and the calcifications over the falx were excised. Post-excision electrograms did not show any abnormal discharge.

During the post-operative course the patient experienced five minor seizures during the 12th and 13th post-operative day. EEG on the 20th, post-operative day did not reveal any epileptiform activity. The patient did not experience further seizures. Another EEG six months after operation was entirely normal and she was free of seizures.

Re-examination five years later showed again a normal EEG and the patient had no further attacks.

POST-OPERATIVE STUDIES

Of these 31 cases, 20 patients (64 percent) did not have any seizures after operation. Fifteen of these 20 patients (75 percent) had a normal post-operative EEG. The remaining 4 cases showed paroxysmal activity only during hyperventilation. This, however, is thought to be insignificant for this early post-operative period since two of these cases re-examined five and six months later showed an entirely normal EEG.

Four cases (12 per cent) showed only a small number of seizures confined to the immediate post-operative period and no epileptiform activity was observed in their post-operative EEG.

Six cases (19 per cent) showed the same seizure pattern and EEG abnormalities as preoperatively. One of these cases was operated a year and a half later, a second excision carried out at the anterior border of the first removal. The seizures then stopped and postoperative electrograms showed no abnormality.

One case had attacks of a petit mal form and a calcified lesion over the right hemisphere most marked on the mesial parietooccipital region. The attacks can now be controlled and there has been a steady improvement in his mental capacity since operation when the calcified area over the parieto-occipital region was excised.

From a clinical standpoint most of the cases showed sensory or motor disturbances in the immediate postoperative period attributable to cortical excision or side effects of exploration and excision. However, most of these effects were transitory.

Fourteen cases (45 per cent) showed transitory motor weakness. Eight cases (26 per cent) showed a postoperative aphasia for about a week. Two cases (6 per cent) showed drowsiness with confusion for the same duration.

The only persisting postoperative disturbance was the cortical sensory impairment observed in two cases.

DISCUSSION

A review of cases with verified epileptogenic lesions involving chiefly a local area on the mesial surface of one hemisphere has served to clarify their electroencephalographic and clinical characteristics.

Clinically these patients present difficult problems of localization particularly if the focus of onset of epileptic discharge lies in the anterior frontal region. From this region the pattern of attacks may resemble closely that of petit mal or generalized grand mal epilepsy, with initial loss of consciousness. In addition there may be autonomic phenomena, sensory aura, or motor onset referrable to both sides of the body, if the focus lies outside the Rolandic sensory motor area.

The problem of localization may be even greater in the electroencephalogram if the electrographic characteristics of such lesions are not fully understood. It is important to recognize the apparently non-focal bilaterally synchronous rhythmic discharges, often of an irregular spike and wave form, may result from local epileptogenic lesion on the mesial surface of one hemisphere. Special studies with coronal lines of electrodes have revealed certain characteristics of such lesions which aid in their differentiation from the primary bilateral synchrony of petit mal epilepsy. The fact that bilaterally synchronous discharges and clinical seizures have disappeared in the majority of these cases following unilateral cortical excision gives strong evidence in support of the hypothesis that these seizures are truly of focal cortical type, and that epileptogenic foci in the cortex surrounding the corpus callosum is capable of setting up widespread electrical disturbances over the convexities of both hemispheres.

In some of the cases described above the question has been raised of the involvement of both cortical and subcortical structures in the pathological process, while in others one might raise the question of bilateral cortical abnormality. It is true that a few of these patients had rather large areas of atrophy rather than small discrete focii. In all, however, the principal lesion and the focus of epileptic discharge was in parasagittal cortex as verified at operation. In many (circumscribed tumors or scars) both the lesion and the focus were clearly restricted to parasagittal cortex. In these latter cases the EEG findings were indistinguishable from those in which additional subcortical abnormality may well have been present (such as in the case of post-encephalitic seizures). In all cases a careful analysis of the clinical seizure pattern in relation to the EEG findings is most important in the differential diagnosis between cortical and subcortical epileptogenic processes.

CONCLUSIONS

Electroencephalographic, clinical, and pathological findings have been summarized in 31 cases with epileptogenic lesions of the parasagittal cortex of one hemisphere verified at operation. The principal electroencephalographic characteristics of these patients are as follows:

1. Bilaterally synchronous paroxysmal rhythmic discharge, often of an irregular spike and wave form, with frequencies of 2 to 4 per sec.
2. Phase reversal studies in a coronal line of electrodes may reveal a peak voltage at the mid-line or near the mid-line on the side of the lesion.
3. There may be a low voltage area over the lesion itself. This may be due either to an equipotential zone within the area of the focus, or it may represent a low voltage area over a destructive lesion.
4. Higher voltage discharge may appear over the homologous area of the contralateral hemisphere, during high voltage paroxysmal discharge, or during clinical attacks.
5. The onset of a clinical seizure may be associated with a suppression of electrical activity if the focus lies in or near the supplementary motor region.

REFERENCES

1. Bailey P, et al: The functional organization of the medial aspect of the primate cortex. J Neurophysiol 1944; 2:51-56
2. Dell MB, et Hecaen H: Complexes pointe-ondes à début unilatéral et épilepsies giratoires. Rev Neurol 1951; 84:656-659
3. Jasper H: Etude anatomo-physiologique des épilepsies. 2e Cong. Inter, d'EEG Proc Paris 1949 (EEG Clin Neurophysiol 1951 Suppl 2:99-111
4. Lennox MA and Robinson F: Cingulate-cerebellar mechanisms in the physiological pathogenesis of epilepsy. EEG Clin Neurophysiol 1951; 3:197-205
5. Penfield W and Jasper H: Highest level seizures. Res Publ Ass Nerv Ment Dis 1946; 26:252-271
6. Penfield W and Ramussen T: The cerebral cortex of man. New York, The MacMillan Company 1950; 248 pp
7. Penfield W and Welch K: The supplementary motor area of the cerebral cortex. Arch Neurol Psychiat Chicagı 1951; 2:59-78
8. Woolsey CN and Erickson TC: Observations on the supplementary motor area of man. Amer Neurol Ass Trans 1951; 50-56

TÜKEL, K and JASPER, H. The electroencephalogram in parasagittal lesions. **EEG Clinical Neurophysilogy, 1952, 4:481-494.**
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