

Vertigo and Rotational Movement in Cortical and Subcortical Lesions

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INTRODUCTION

For a number of years the authors have been interested in lesions responsible for dizziness and vertigo. In reviewing clinical material it is apparent that adequate differentiation is not being made between peripheral lesions such as those in the ear or the vestibular nerve and central lesions involving the primary vestibular areas or thalamic or cortical regions related to vestibular functions. Too frequently dizziness or vertigo is attributed to labyrinthitis when in reality it is caused by lesions stimulating the central vestibular centers (BROWN *et al.* 1956; PENFIELD AND FLANIGIN 1950). On the motor side, insufficient emphasis has been placed upon the torsional or rotational movements which are occasionally seen in convulsive disorders in man.

Experimental work (CALHOUN 1966; CALHOUN AND CROSBY, 1965; CROSBY AND CALHOUN 1965) has demonstrated that torsional head and trunk movements and/or somersaulting can be produced in the macaque by the application of a disc of alumina cream upon the inferior frontal or the superior temporal cortex. Several years ago two reports (SCHNEIDER *et al.* 1961; SCHNEIDER *et al.* 1965) from the Neurosurgical Service at the University of Michigan discussed hallucinations which suggested a temporal lobe involvement but which were actually caused by extratemporal lesions. These studies suggested the desirability of a review of the case histories of various patients upon whom temporal lobectomies had been performed for intractable convulsive seizures, in order to discover whether, in cases where symptoms of dizziness, vertigo or perhaps rotational movements had been reported, such symptoms might have aided in the more precise localization of brain lesions. A series of 7 case studies provides the basis for an anatomical, electroencephalographic (EEG) and clinical discussion of central vestibular signs and symptoms.

CASE REPORTS

Case 1

A 17-year-old, left-handed high school boy was admitted to the hospital on 27 December, 1965 for the diagnosis and the treatment of convulsive seizures. At the age of 2 or 3 he had grasped whatever was near him and weaved from side to side with a wide-based stance. These episodes lasted for about

1 year and then disappeared spontaneously. There was an interval of 9 years during which he was perfectly well. At 11 years of age, he began to have seizures during which he was unaware of his surroundings. He was in a trance-like state, in which he carried out inappropriate activity. Usually these seizures were associated with turning of the head and the eyes toward the left side. There were bouts of aphasia. On several occasions he walked up the aisle in his schoolroom to place something in the wastebasket, but he circled to the left and walked around and around the basket and the teacher's desk. During one episode he was carrying some pop bottles in his arms across the gym floor when he suddenly had a spell and dropped all of the bottles; he reached up and grabbed his head and turned around and around in a circle. The patient continued to have seizures three or four times a week in spite of a massive drug therapy program of phenobarbital, Dilantin, phenurone and dexedrine. He had no olfactory, auditory or visual hallucinations. His neurologic examination was completely normal.

His routine skull X-rays demonstrated abnormal calcification in the left temporal lobe above the left petrous ridge, and the left internal carotid arteriogram showed elevation of the middle cerebral artery (Fig. 1A). The fundoscopic and visual field examinations were normal. A radio-active mercury brain scan was equivocal for a lesion in the left temporal area. An EEG was recorded, and the patient demonstrated spiking activity and slow waves in the left midtemporal and anterior temporal regions, with occasional involvement of the premotor and the lateral parietal and posterior temporal areas

(Continued on p. 497)



Fig. 1 A (Case 1).

A: left lateral view of the internal carotid arteriogram demonstrates an elevation of the left middle cerebral artery. Abnormal calcification above the internal auditory meatus (arrows).

B (p. 495): preoperative electroencephalogram. *Column I*: during waking the background is 7/sec. There is serial 3-3.5/sec activity most marked on the left in the mid and anterior temporal regions. Sleep recordings (not shown) bring out the focal nature of the discharge and show the premotor-motor, lateral parietal and posterior temporal areas to be involved. *Column II*: after 50 sec of hyperventilation, the patient had a seizure. The area of onset of spiking during the seizure is ill defined. Both left temporal and parietal leads are involved early with rapid generalization of discharge as shown.

C (p. 495): with hyperventilation the patient became unresponsive and turned his head and eyes toward the left. Shortly thereafter his left arm partially flexed and both legs extended.

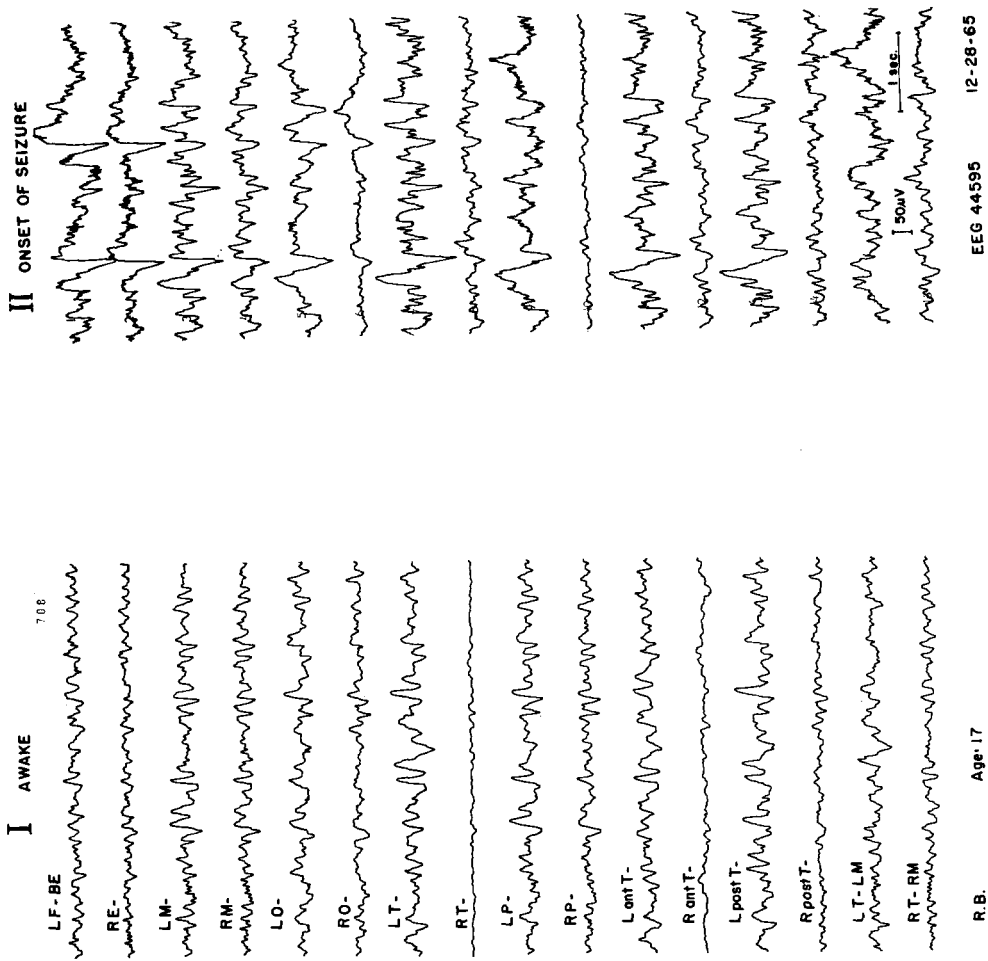


Fig. 1 B.



Fig. 1 C.

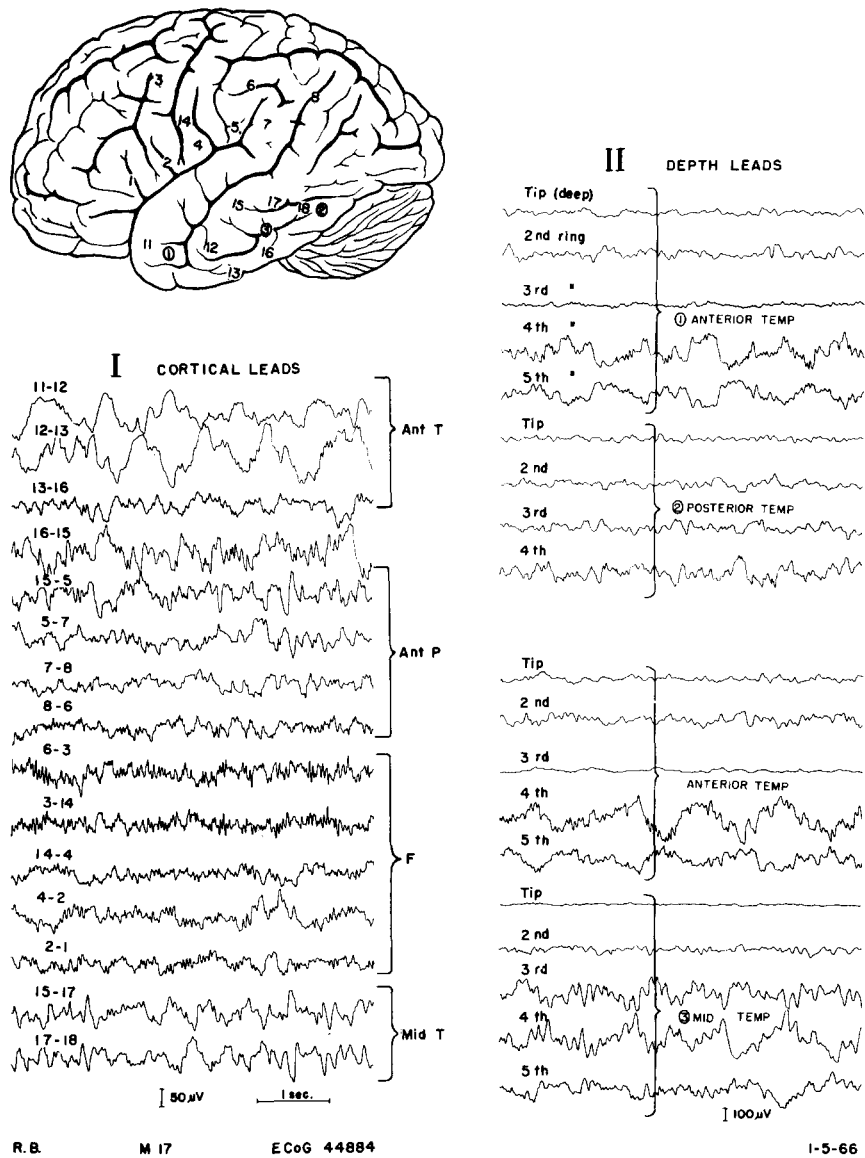


Fig. 1 D. Recordings made at operating room table are shown. *Column I*: electrocorticography revealed 0.5–2/sec waves arising from anterior and midtemporal regions (electrodes 11, 12, and 13, first two lines of recording). The region of maximal EEG abnormality was anterior to the apparent location of calcification in the skull films. There was no prominent spiking, only scattered equivocal spikes and sharp waves. Electrodes over the lateral parietal and midposterior temporal regions (numbers 5, 7, 8, and 15, 16, 17; lines 4 and 5, and 14 and 15) revealed 4–5/sec activity. *Column II*: a depth probe in the same region (2) revealed the same finding. A depth probe inserted at the tip of the temporal lobe (1) also confirmed the presence of irregular long wavelength activity as in the surface leads. A third insertion of a depth probe (3), angled at about 45°, and directed from the middle towards the anterior portion of the temporal lobe revealed irregular slow wave activity at the most superficial contacts.



Fig. 1 E. *Left*: in repose there was a slight flattening of the left nasolabial fold, a drooping of the left corner of the mouth, and a widening of the left palpebral fissure. *Right*: upon being asked to smile, there was no evidence of left facial paresis.

Fig. 1B, Col. 1). Occasionally his seizures were evoked by hyperventilation (Fig. 1B, Col. 2), during which he opened his eyes and became unresponsive to comments, swallowed, extended both legs and hook. Then, gradually, his head and eyes turned to the left and his left arm moved into partial flexion (Fig. 1C). The intracranial calcification and the character of the focal EEG signs suggested the diagnosis of an expanding intracranial neoplasm.

On 5 January, 1966 a left fronto-temporo-parietal craniotomy was performed with electrocorticography and depth electrode studies (Fig. 1D). Following these recordings, a cortical incision was made and, at the depth of 2 cm, a reddish gray, rubbery tumor was found in the left anterior and midtemporal regions. It extended superiorly into the region of the frontotemporal junction and the island and also into the posterior temporal area. The temporal lobe along with the lesion was excised for a distance of approximately 6 cm from its tip.

Postoperatively, the patient developed a mild anosmia and an expressive aphasia, as well as a right central facial palsy, all of which cleared within a week. The pathologic diagnosis was made of neurocytoma; the lesion was regarded as non-responsive to radiation therapy. The boy was discharged from the hospital on 22 January, 1966 on an anticonvulsant regime.

A follow-up examination a year later indicated that the patient had had no further convulsive episodes. He was neurologically negative except for a slight flattening of the *left* side of the face, which was not apparent on smiling on command (Fig. 1E).

Comment. The presence of calcification in the posterior temporal region probably has little or no relationship to the triggering of the temporal lobe symptoms by the tumor. With growth of the neoplasm, there was a definite shifting from the sensory vertiginous symptoms to a motor vestibular response. The hyperventilation caused ipsiversive rotation of the head and eyes (toward the left side) with rigid hyperextension of both lower extremities and clonus of his feet. On other occasions the patient displayed torsional circling movement of the head toward the left side. These motor responses closely resembled those of the macaques (CALHOUN 1966; CALHOUN AND CROSBY 1965) in whom irritative lesions had been made at the frontotemporal junction as described above.

The preoperative EEGs, covering a 4 year period, were successively reported as "mildly abnormal", "abnormal and paroxysmal" and finally in 1965, as "abnormal and epileptogenic in nature", thus sug-

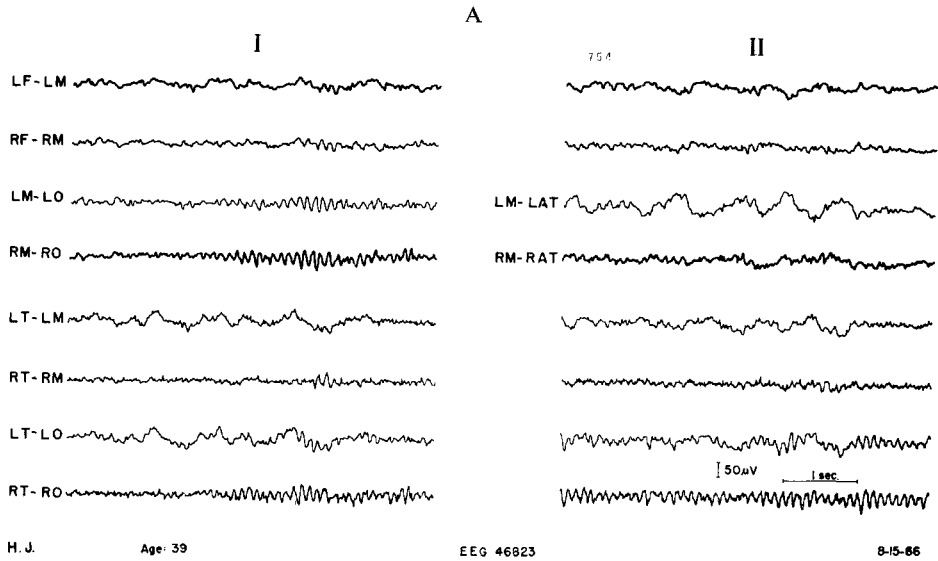


Fig. 2 (Case 2). *A*: preoperative electroencephalogram. *I* and *II* are bipolar recordings showing strong focal continuous 1.5–2/sec activity in the left anterior and midtemporal regions. The background activity is a well developed and organized 9.5/sec rhythm in both occipital areas. *B*: the postexcision photograph of the operative field shows the floor of the left anterior fossa at its lowermost portion with the bank of residual tumor in the upper part of the field immediately underlying the Sylvian veins at the frontal and temporal operculum.

gesting a progressive deterioration in the abnormality with expansion of an intracranial lesion. Electro-corticograms (ECoG) confirmed the slow waves seen in the preoperative studies and the depth electrode recordings showed the greatest abnormality at the most superficial contacts of the probe located in the anterior temporal lobe.

Case 2

H. J., a 39-year-old, right-handed patient, was admitted to University Hospital on 17 August, 1966 with a chief complaint of dizziness and nervousness of 16 months duration. He had a history of peculiar dizzy spells of 1 min duration in which he described "pressure spinning over the top of the head". There was no evidence of lateralization of these symptoms. Initially these attacks had occurred three or four times a month, but, by the time of admission to the hospital, they had increased to six or seven times a day. In March, 1965, early in his illness, he had a peculiar sensation in his head and felt "music was floating by". This music was familiar and yet he was unable to name the title of the piece. After these symptoms subsided, he had tingling of the right side of his face and right upper extremity. There were only two other occasions when he had auditory hallucinations but, in each instance, they were flashbacks of past events which were too indefinite to describe clearly. He had no olfactory aura. Because of confusion and memory impairment (without headache, nausea or vomiting), he was finally committed to a mental institution as a psychiatric patient. At that time his positive neurologic findings were limited to a right central facial paralysis, a partial expressive aphasia, and a slight right-sided hyperreflexia. In June, 1966 he had a skull X-ray which demonstrated the shift of the pineal gland from the left toward the right side.

Although an EEG taken in another community had been described as negative, on 15 August, 1966 he was admitted to the University Hospital where a repeat tracing showed a strong delta focus in the left anterior and midtemporal region (Fig. 2A). His radioactive mercury brain scan was negative. His audiograms and vestibular tests were within normal limits. A left internal carotid arteriogram exhibited a shift of the anterior cerebral artery toward the right side and the "Y" displacement of the left middle cerebral artery, with an elevation of the Sylvian vessels. The available data suggested the presence of a large temporal lobe tumor.

A left frontotemporal craniotomy was performed on 23 August, 1966. When the dura was opened over the frontal lobe, a small cortical abrasion about 4-5 mm in depth was made in the left anterior temporal region and a reddish-gray, friable tumor was exposed. The Sylvian veins were spread apart by the underlying tumor. A subtotal lobectomy was performed for a distance of 6 cm from the tip of the left temporal lobe with partial removal of an astrocytoma, grade 1. Residual tumor could be seen extending upward into the frontal operculum but further excision was limited, since this was the dominant hemisphere (Fig. 2B).

Comment. The gradual shifting of symptoms of vertigo to auditory hallucinations of hearing vaguely familiar music, to a tingling sensation of the right side of the face and right upper extremity, and finally to memory impairment and mental confusion, suggested the extension of a temporal lobe lesion or a spread over the association pathways.

The positive arteriogram, with the EEG finding of well-defined focal waves in the left anterior and midtemporal areas, confirmed the probable diagnosis of a left temporal lobe tumor.

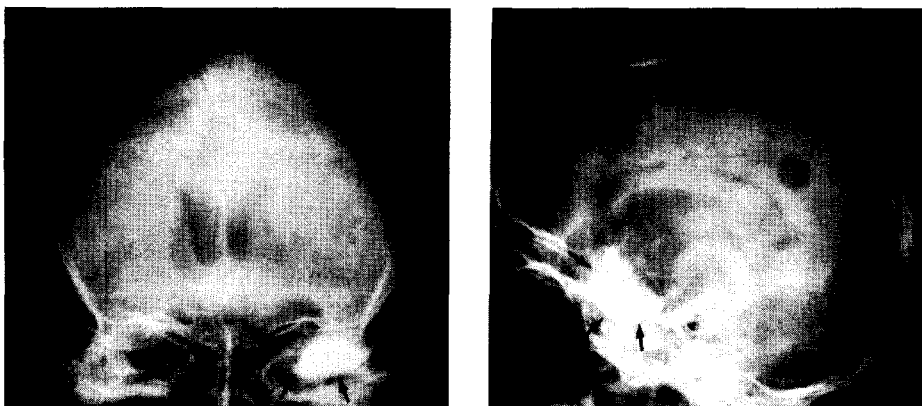


Fig. 3 A and B (Legend see p. 500).

Case 3

An 18-year-old, right-handed milkman, L. K., was first seen in April 1958 for dizzy spells. His birth history revealed that there had been severe molding of the head, but no instruments were used. At 6 years of age he had had episodes of staring, becoming rigid, and nauseated. He never lost consciousness during these spells. The onset of such an attack was an aura of abdominal discomfort. This was followed by a visual hallucination in which he saw 10 men coming toward him; he then clutched some object for support for he became very dizzy. Although he could hear what was going on about him he was unable to speak. He had no olfactory or auditory hallucinations. These episodes occurred about twice monthly and were fairly well controlled on anticonvulsants. At the time of his examination in

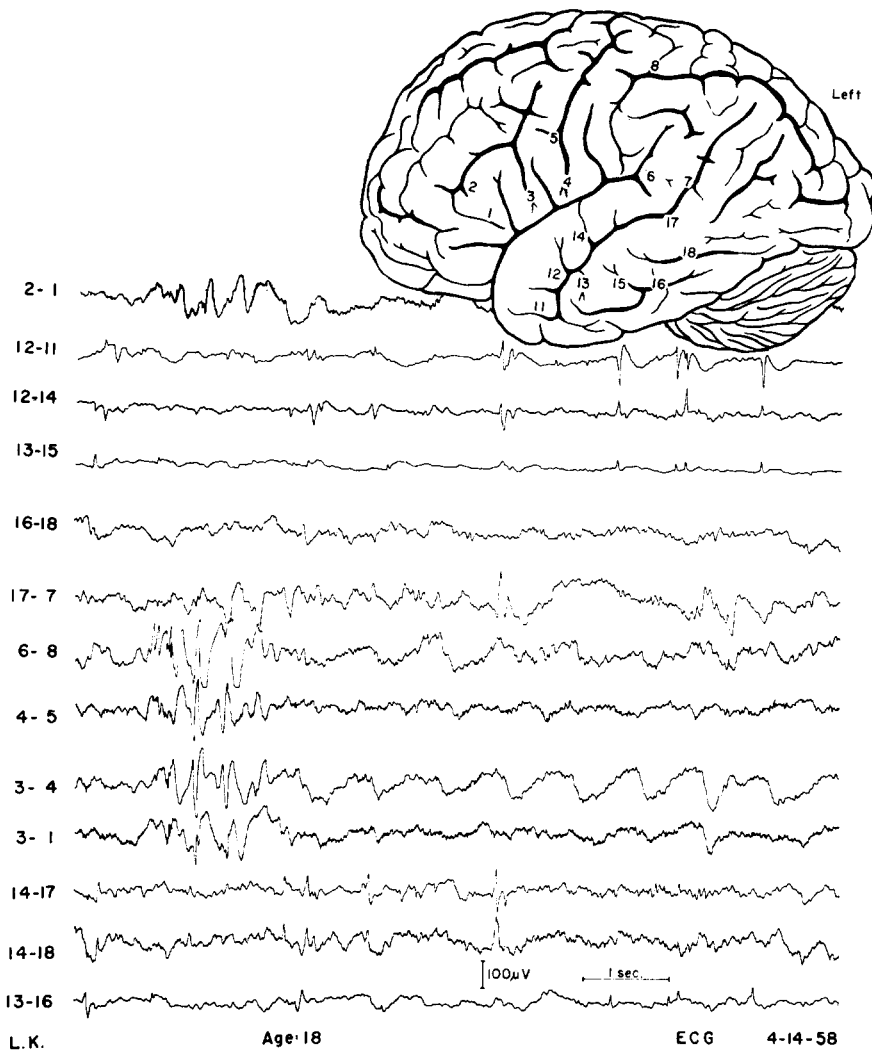


Fig. 3 C.

Fig. 3 (Case 3). *A* (p. 499): the anterior-posterior view of the ventriculogram shows the ventricles lie in the midline and the thorotrast is demonstrated in the dependent part of the tumor cavity. *B* (p. 499): the lateral ventriculographic view shows the relationship of the tumor cavity to the lateral ventricles. *C*: electrocorticography. Ball electrodes were placed directly on the cortex in the areas indicated by numbers. The first line of recording (2-1) was from the frontal cortex, the second line (12-11) from the anterior temporal lobe, following on down the mid-temporal, posterior frontal, premotor and again temporal. Sharp spikes were recorded from the anterior and midtemporal regions, spikes with slow waves from the posterior frontal and lateral premotor areas and these were associated with slow wave forms.

April, 1958, the only positive neurologic findings were slowed mentation, difficulty with pronunciation of words, and symmetrical hyper-reflexia with bilateral upper extremity pyramidal tract signs.

Skull X-rays showed an extensive calcification in the brain and retrosellar areas but there was no erosion of the sella turcica. He had a partial right superior homonymous hemianopsia. The CSF pressure was normal on lumbar puncture, but he had a 3+ Pandy test and the fluid had a protein content of 190 mg/100 ml. Psychological testing revealed a verbal IQ of 71. An EEG demonstrated a bilaterally diffuse pattern with numerous sharp spikes and theta waves even in the posterior quadrants with some focal response in the left temporal area. A left percutaneous arteriogram demonstrated a left temporal space-occupying lesion.

On 14 April, 1958 a left posterior parietal burr hole was made and ventriculography was performed. Next a left temporal burr hole was made and an exploring needle introduced into a cystic cavity at a depth of 1.5 cm. A golden-yellow fluid was removed and about 3 ml of thorotrast was introduced. The aspirated fluid contained 170 mg/100 ml of protein. The ventriculograms with the thorotrast injection revealed a presumably normal ventricular system with a smooth-walled cyst in the left temporal region (Fig. 3A and B). At the time of left frontal and temporal craniotomy, electrocorticograms were recorded and demonstrated a major focal dysrhythmia in the left anterior temporal area with spread to the left mid-temporal and left frontal regions (Fig. 3C). A cystic lesion containing friable brownish-gray tissue and calcified material was subtotally excised because of its adherence to the left middle cerebral artery. Excision was carried to a point 6 cm posterior from the tip of the left temporal lobe. Unfortunately, the tumor specimen was lost so that no pathologic diagnosis could be made. Although the patient had been on large doses of anticonvulsants, he continued to have focal seizures involving the right arm and leg. This was revealed at the time of his re-examination, 8 years after operation. A recheck electroencephalogram at this time exhibited a moderately abnormal paroxysmal pattern with abortive spike waves in the left posterior quadrant. He had sustained no more attacks of dizziness or visual hallucinations.

Comment. The 20-year history of convulsive seizures with the gradually progressive alteration in their pattern suggested the possibility of a slowly-growing and infiltrating tumor. This was supported by the fact that he had extensive calcification in the brain and the parasellar areas in association with the large cyst. It is regrettable that the pathologic specimen was lost so that histologic verification could not be made. Inasmuch as the cyst did not contain machine-oil fluid with glistening cholesterol crystals, the tumor was probably not a craniopharyngioma. Since there was no apparent mural nodule and there was calcification it is unlikely the tumor would be classified as a cystic astrocytoma of the cerebral hemisphere on gross examination. The lesion was more probably an oligodendroglioma with cystic degeneration. The occipital view of the thorotrast study (not shown here) suggested extension to and across the midline.

The preoperative EEG and ECoG showed a diffuse disturbance of pattern. The size of the cystic tumor explains the degree of the abnormality. The tips of the depth probes in the temporal lobe were probably within the cystic area, the spikes recorded from the frontal probes must have been on the periphery of the lesion.

Case 4

A 34-year-old right-handed man, R. H., was first referred to University Hospital on 3 December, 1961 for evaluation and treatment of his temporal lobe convulsive seizures. The patient was a product of a premature gestation being born at 7 months. Shortly after birth he had jaundice which lasted for 6 weeks. His first convulsive seizure occurred at 13 years of age when he was aroused from sleep by a sensation of spinning counter-clockwise with a tendency for his head to draw toward the left side. There was a post-ictal period of confusion. At 16 years of age the patient began to have convulsive seizures during the daytime. These were usually accompanied by a prickling sensation in the fingers of his right hand. He stated he could abort the attacks at times by grasping the right hand firmly with the left one. If the attack progressed the right arm was extended and raised involuntarily above the head. Frequently with the major focal motor type of seizure he pitched to the floor sustaining lacerations of his scalp. This necessitated some protection and a crash helmet was worn in order to avoid serious injury. The patient had been treated with a broad spectrum of anticonvulsant medication without much success.

An EEG performed on 5 December, 1961 revealed some slowing in the left motor, anterior temporal, and midtemporal areas. After hyperventilation, spikes became more prominent in these regions with some spread into the occipital area. Sleep accentuated the spike activity. The routine skull films and bilateral carotid arteriograms obtained from an outside hospital were normal. A pneumoencephalogram on 8 December, 1961 was reported as revealing normal filling of the temporal horns. At this time a spinal fluid protein determination was 55 mg/100 ml and the globulin test was positive. A left fronto-temporal craniotomy was performed and ECoG's were recorded. There were definite spike reversals in

the left temporal area. Depth electrode recordings exhibited slowing from the frontal region and spikes with slow waves from the tip of the anterior and midtemporal areas (Fig. 4). The tumor which was subtotally removed from the left temporal lobe was an astrocytoma, grade 2. The patient recovered satisfactorily for an interval but after 2 years he had recurrent seizures and was committed to an epileptic hospital.

Comment. The original aura on this patient was a spinning sensation followed by an ipsiversive rotation of the head. This movement was eventually replaced by a prickling sensation in the fingers of the right hand with subsequent extension of the right arm upward and over his head.

All electrical studies—preoperative EEG, ECoG and depth electrode recordings—showed the slow wave component but this was overshadowed by the spike activity which was so prominent.

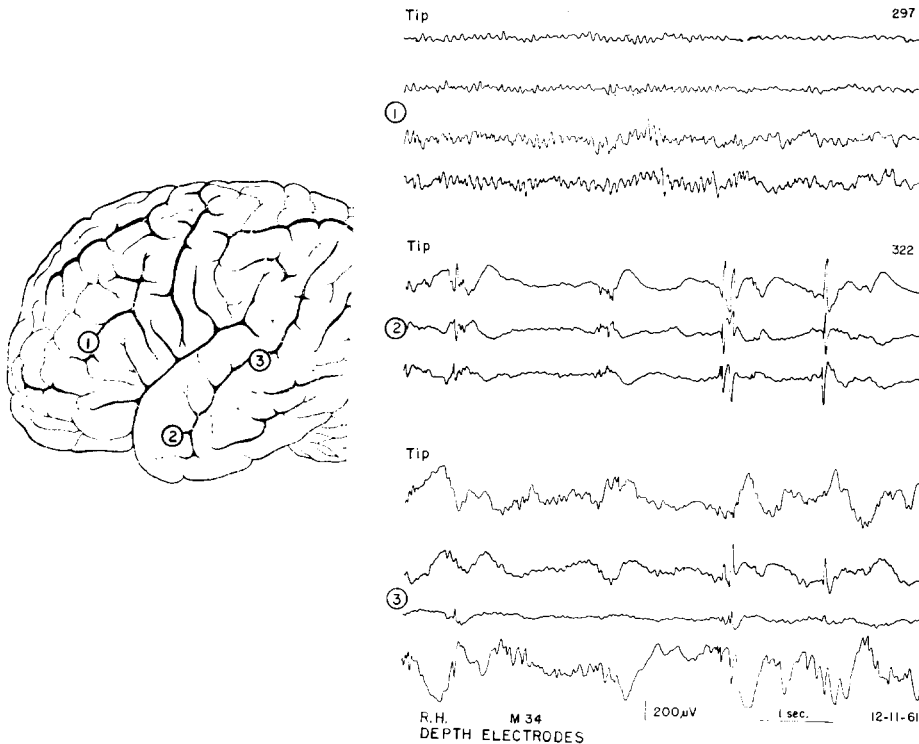


Fig. 4 (Case 4). Tracings from depth electrodes. (1) the frontal lobe: serves as a good control against which comparisons may be made. (2) the tip of temporal lobe: with its high voltage spikes from all contacts. (3) the posterior part of the temporal lobe: with spikes and slow waves. The (2) and (3) electrodes suggest the presence of a deep anterior and midtemporal tumor.

Case 5

On 5 January, 1962, R. P., a 24-year-old, left-handed laborer was admitted to University Hospital, for further diagnostic studies and treatment of a probable brain tumor. For 2 years he had had convulsive episodes with jerking and turning of his head to the left, and rhythmic seizures of the left side of his face with opening and closing of the eye. This was followed by flexion and extension of the left arm. Later there was spread of the seizure to the right extremities with a grand mal type of pattern. Occasionally, the right arm extended directly upward. A year after the onset of his seizures, he noted spells of dizziness followed by a sensation of floating on air which persisted for about 30 sec. The only neurologic abnormality was an equivocal left central facial weakness.

He had a negative radiocative mercury brain scan. The right carotid arteriogram showed a shift of the right anterior cerebral artery toward the left side. Pneumoencephalography also demonstrated a slight shift of the lateral and third ventricles to the left. A diagnosis was made of deep thalamic or temporal lobe tumor and since he was controlled on medication, no operation was performed.

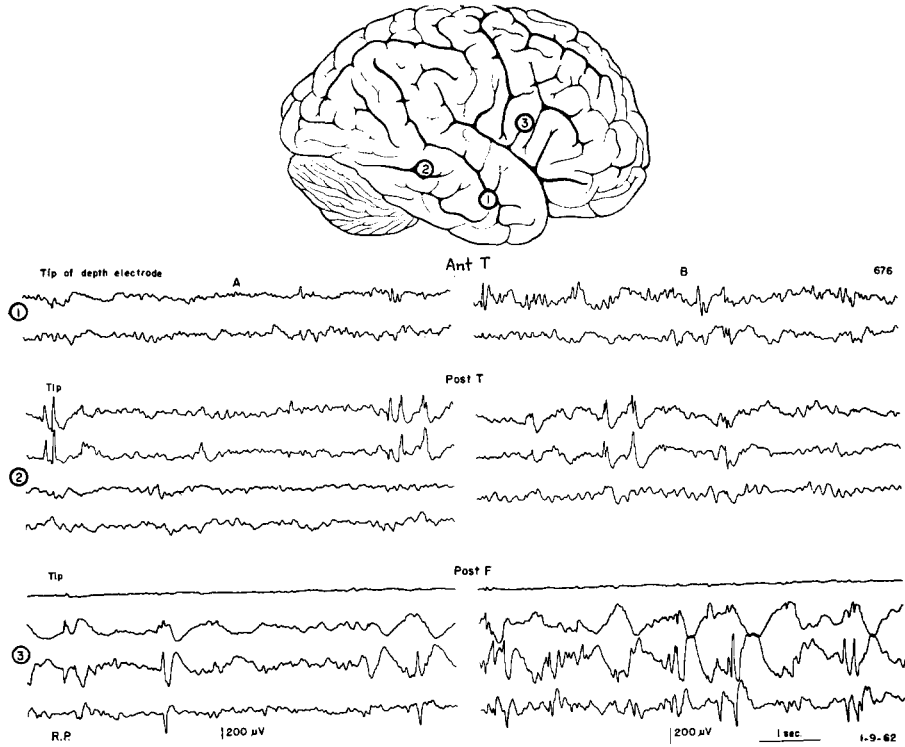


Fig. 5 A.

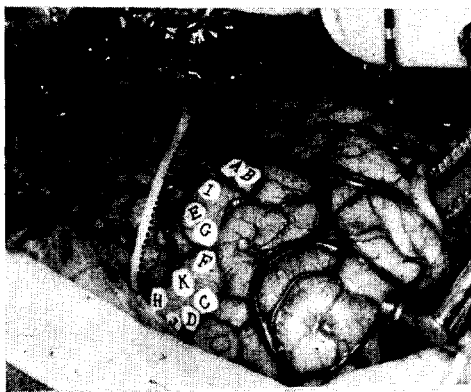


Fig. 5 B.



Fig. 5 C.

Fig. 5 (Case 5). *A*: the depth electrode recordings are presented. A depth electrode (Grass type) was inserted at points (1), (2) and (3). Each electrode is 2 mm in diameter and has ten silver ring contacts electrically insulated from each other. These rings are spaced 10 mm apart. Electrode (1) is recording from the tip and 2 rings above it; (2) the tip plus 4 rings, (3) the tip plus 4 rings. Recording *B* was made 10 sec later than *A* with increased amplification. *Note*: spikes from the tip of (1), from the depth of (2), and in the upper contacts of (3). The frontal electrode (3) was inserted at an angle several times and this so-called quiet or flat recording was obtained from its tip. Frequent, high voltage spikes and slow waves are seen nearer the surface. *B*: the tip of the temporal lobe was stimulated on its lateral inferior surface resulting in ipsilateral movement of the face (because of the inferior location of this point it was impossible to place a marker which could be included in the photograph). Responses obtained from electrical stimulation, 5V 4 msec 40 cy: A, orbicularis muscle; B, tongue; 6V 6 msec 40 cy: C, eye twitching, clonic closure of eye; D, eye moved outward; E, retraction corner of mouth; F, nose and mouth; G, mouth; H, eye out and up, left; I, closure of jaw; J, movement of eyelid; K, forced closure of eye, drawing of mouth to left. *C*: the extent of excision of the frontal and temporal regions is demonstrated.

On 21 August, 1961 he had periods during which he was unaware of his surroundings, talked unintelligibly, and stared off into space. There was no loss of consciousness. On medication he became seizure-free, but on 5 October, 1961 he began to have violent attacks of rage. Two months later he not only had headaches, feelings of unreality, and severe rage states but also his recent and past memory became increasingly impaired. At this time he had an aura of vertigo in which things were spinning around him and his vision became fuzzy. He subsequently developed a left Jacksonian seizure.

Three weeks prior to admission to the hospital he had a markedly abnormal EEG with a deep right temporoparietal focus. On 9 January, 1962 a right frontotemporal parietal osteoplastic craniotomy was performed. ECoG's suggested a spike focus in the right temporal lobe and slow activity in the frontal lobe. Depth electrode studies showed spiking in the right anterior and midtemporal areas and δ -abnormality with spikes in the inferior frontal area (Fig. 5A). Stimulation revealed the location of the motor strip and an ipsilateral motor facial response was obtained from the tip of the temporal lobe (Fig. 5B and C). After excision of the prefrontal portion of the right frontal lobe repeat ECoG's continued to show the paroxysmal right focus. This focus was excised with removal of the anterior 6.5 cm of the temporal lobe. A pathologic diagnosis was made of astrocytoma, grade 1.

A reply to a follow-up letter sent to a mental institution on 4 August, 1964 revealed that the patient had paranoid trends with delusional beliefs about the infidelity of his wife. He had a minimal left central facial paresis. There had been a definite improvement of recent memory but not in past memory. He lacked insight and his judgment was poor. His convulsive episodes had decreased to one seizure a month on an anticonvulsant regime of dilantin, phenobarbital and mysoline.

Comment. This patient exhibited a slightly different seizure pattern from the previous patient since he occasionally had a tonic upward extension of the ipsilateral rather than the contralateral upper extremity. This is the only patient in this series in whom attacks of rage were manifested and these finally were responsible for his commitment to a mental institution.

Case 6

A 43-year-old right-handed woman, H. P., was first admitted to the hospital on 13 November, 1963 stating that she had had orbital pain of 10 years' duration. For the 8 months prior to her hospitalization, she had suffered from sharp left temporal headaches. Six weeks before this evaluation she had struck the left side of her head slightly and noted pain out of proportion to the blow. Eight days before admission to the hospital she stated she tended to spin toward the left side. She had had 3 episodes of dizziness in which the room seemed to spin about her. There was triplopia and twitching of the left eyelid. The remainder of her history and her neurologic examination were normal. A 3 × 4 cm firm raised mass could be palpated at the left frontotemporal junction of the scalp. It was slightly tender to the touch, but the scalp moved readily over this region. When a lumbar puncture was performed the pressure and chemical studies were normal. Skull X-rays revealed a lytic lesion of the skull at the site of the mass (Fig. 6A). Her brain scan suggested a tumor in the left frontotemporal region. A left internal carotid arteriogram showed some displacement of the Sylvian vessels but there was no shift of the anterior cerebral artery across the midline. On 19 November, 1963 a greyish tumor, which proved to be a meningioma, was removed by craniectomy from the left frontotemporal junction. The bone was rongereured away to normal tissue. The dura had a few tumor cells on its surface, but there was no apparent penetration of neoplasm into the brain. The dura was cauterized carefully and thoroughly to prevent recurrence of the tumor. The patient made a good recovery and was discharged from the hospital 11 days later.

When this lady was seen more than 2 years after operation she complained of some recent recurrence of the sensation of spinning toward the left side. However, these symptoms had disappeared only 6–8 weeks prior to this examination. Her neurologic status was normal. Her skull X-rays showed a left frontotemporal defect without any evidence of lytic involvement (Fig. 6B). The brain scan on 6 April, 1966 demonstrated a lesion at the left frontotemporal junction (Fig. 6C and D).

Comment. This patient had the only benign tumor in this series. She had a subtotal excision of a meningioma en plaque which subsequently recurred 2 years postoperatively. Also the tumor in this case was the only one which was superficial and lay directly over the frontotemporal junction similar to the lesion seen in the macaques. The patient's symptoms probably altered as the lesion increased in size.

Case 7

P. J., a 6-year-old, right-handed boy, was the product of a full term forceps delivery which was complicated by the cord being wrapped around the neck. Spontaneous respirations did not occur and the child was revived only after artificial respiration and oxygen administration. For 4 years he had had left-sided focal facial seizures and a sensation of spinning spells. If standing during one of

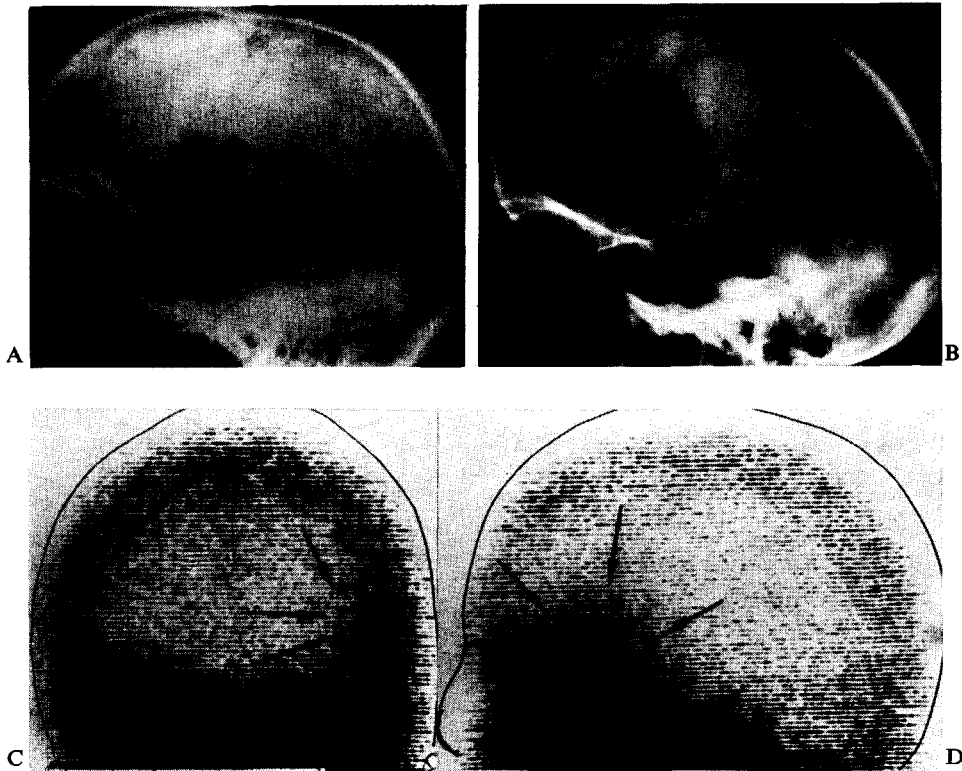


Fig. 6 (Case 6). *A*: left lateral X-ray film showing a frontotemporal destructive lesion of the skull. *B*: a repeat skull X-ray 2½ years later showing no evidence of a lytic lesion in the postoperative defect. *C*: frontal view of brain scan demonstrating a lesion in left frontotemporal junction 2½ years after subtotal excision of the tumor. *D*: the lateral view of the brain scan confirming the location of the lesion underlying the skull defect.

these seizures he would assume a wide stance and weave, but would not fall. If sedentary he might exclaim: "There goes my face again (meaning it disappeared); I will be all right in a couple of minutes". His hands might then drop limply into his plate and when asked to watch where his hands were he would say: "I can't see them". Recovery from such a seizure occurred in 2 min. During more recent months prior to hospitalization he had complained of visual hallucinations of "seeing things upside down". The patient did not lose consciousness, was oriented and was fully aware of his environment during these episodes. He complained of almost daily frontal headaches, and his mother noted he had difficulty with his left foot when skipping. Alternating movements were not well performed with the left hand.

Earlier X-rays had demonstrated a thinning of the inner table and bulging of the outer table of the skull in the posterior parietal area. The current skull X-rays showed further lytic destruction of this parietal area (Fig. 7). His radioactive mercury brain scan was regarded as normal. An EEG was definitely abnormal and paroxysmal with high voltage spikes and serial rhythmic δ -waves over the right posterior quadrant suggesting a structural, or possibly an atrophic, lesion. A right percutaneous arteriogram was deemed unsatisfactory. A pneumoencephalogram was found to be normal.

On 19 January, 1966 a right parieto-occipital craniectomy was performed by Dr. Edgar A. Kahn with total removal of a right parieto-occipital astrocytoma, grade 2. This procedure was followed by an acrylic cranioplasty. Postoperatively, on 9 March, 1966 the patient seemed well and was neurologically negative except for a left homonymous hemianopsia.

Comment. This patient was the only one of the group with an infiltrative lesion of the *parietal* area which produced vertigo. The surgeon believed that he had obtained a complete removal of the tumor.

The EEG was reported as being consistent with a right parieto-occipital abnormality; this was confirmed by operation.



Fig. 7 (Case 7). A skull X-ray demonstrated the degree of destruction of the skull.

CLINICAL DISCUSSION

In our series of 46 patients who had temporal lobectomies for intractable temporal lobe seizures, there were 12 individuals who had brain tumors and 7 others who were found to have cysts. The incidence of brain tumors would, therefore, be 25.9% which is considerably higher than the number of intracranial neoplasms reported in other series (BAILEY AND GIBBS 1951; BROWN *et al.* 1956; FALCONER *et al.* 1955; GREEN *et al.* 1951). Although PENFIELD AND JASPER (1954) reported that intracranial tumors were rarely a cause of epilepsy under 15 years of age, 4 of our 7 cases (Cases 1, 3, 4, and 7) were patients who were under 13 when they had their first seizure.

In the current group of 7 patients who displayed vertiginous symptoms all had brain tumors. The duration of the symptoms varied from $1\frac{1}{2}$ to 21 years prior to the time the patient came to the neurosurgeon for operation. Three of the group had had seizures for less than 5 years; and 4 had had symptoms for over 10 years. Two patients had demonstrated, very clearly, a partial or complete homonymous hemianopsia. None of the patients had olfactory hallucinations suggesting that the lesions arose from a lateral rather than a medial position in the temporal lobe or, perhaps, more likely, a

destruction of the olfactory association fibers. With stimulation of such fibers one would expect olfactory hallucinations, but with destruction these could no longer fire.

In this series there were 4 patients with astrocytomas, 1 patient with a neuroastrocytoma, 1 with a meningioma, and 1 in whom the tumor type was unknown since the biopsy specimen was lost. The best diagnostic procedure in this series was the carotid arteriogram, for 5 studies were positive for an expanding lesion, 1 examination was negative, and 1 arteriogram was deemed unsatisfactory. The brain scan was disappointing in this study for it was negative in 3 cases and equivocal in 1 (in 2 other instances the scan was not performed). This experience has been consistent with the findings in our clinic for astrocytoma, grade 1. These patients have usually had a negative scan, whereas patients with astrocytomas of grade 2 have shown about 50–60% incidence of positive scans. Probably in all but 1 of the patients in this series, namely in 6 individuals, there were infiltrating tumors. In 1 patient with a definitely positive brain scan, there was a benign tumor, a meningioma, which infiltrated the bone.

In general it may be said that vertigo should frequently be regarded as a symptom arising from a lesion of central origin rather than from an irritative process affecting the periphery. Such a situation may occur not only with neoplastic cerebral lesions but also in traumatic ones. With blows to the occipital region frequently there is a contrecoup injury to the frontotemporal junction of the cerebrum as it impinges on the sharp sphenoid ridge. A contusion, or small hemorrhage, in the region of the vestibular projection or association areas may be responsible for the symptoms of vertigo which sometimes occur in the postconcussional syndrome.

Where there is no history of trauma a careful inquiry concerning vertigo may be made and associated symptoms related to the temporal lobe should be sought. When these are present and tend to alter, they suggest the possibility of a neoplastic process which is first causing irritation and subsequently destruction as it increases in size.

ELECTROENCEPHALOGRAPHIC CONSIDERATIONS

In 1938 GIBBS *et al.* analyzed the "paroxysmal dysrhythmias" of epilepsy and considered that the 4–6/sec waves were generalized from all head regions. JASPER AND KERSHMAN (1941) pointed out that the abnormal discharge was localized in the temporal regions and that in the majority of cases it was confined to one temporal lobe. In 1947 GIBBS *et al.* gave their paper on the anterior temporal localization of sleep-induced seizure discharges of psychomotor type.

PENFIELD AND FLANIGIN (1950) reported 68 cases of temporal lobectomy in 1949. BAILEY AND GIBBS (1951) and GREEN *et al.* (1951) reported the removal of the anterior parts of one temporal lobe under EEG monitoring.

Six of the patients in this series had preoperative EEG's. Two were reported as having strong slow wave foci consistent with structural lesions. Slow wave forms, in addition to spike activity, were reported in the remaining 4 tracings. Corticography and depth electrode studies served to delimit the focus.

The most acceptable evidence of a focus is a consistent spontaneous localized spike discharge recorded in the patient's EEG in the waking or sleeping state (Cases 4 and 5). In our experience slow potentials recorded directly from the cortex have been reliable

(Cases 1, 4 and 5). Electrical stimulation to locate the motor cortex and to study its functional organization has been illustrated (Case 5). Tracings are made during the operation, after partial excision and before closure. Follow-up postoperative EEG's from the scalp are made to complete our records.

ANATOMICAL CONSIDERATIONS

Vestibular relations

Impulses set up in the labyrinth are relayed by a series of neuron arcs, with synapses in the vestibular nuclei of the brain stem, to dorsal thalamic levels presumably in the region of the medial geniculate nucleus (CARPENTER *et al.* 1959; MICKLE AND ADES 1954). From this thalamic area, such impulses are projected to the opercular portion and the adjacent lateral surface of the superior temporal gyrus (Fig. 8) rostral to the auditory area (NIELSEN 1946; PENFIELD 1957; PENFIELD AND ERICKSON 1941; PENFIELD AND RASMUSSEN 1950; RAE 1953). It appears quite probable that this cortical vestibular region may have, like the auditory cortex, a projection area on the opercular surface and an association area on the immediately adjacent lateral surface of the superior temporal gyrus but evidence for so specific an arrangement is not yet available. In such an association cortex, impulses relayed from the vestibular projection area would be interrelated with those from other temporal fields and from occipitoparietal, frontal and insular regions over the wealth of association bundles (Fig. 9A and B) interconnecting them.



Fig. 8. The vestibular projection area and the adjacent vestibular association areas are demonstrated on the opercular portion of the superior temporal gyrus. The auditory association and projection areas lie more posteriorly along the operculum. The patterns of the supplementary or second motor areas in the temporal region have been demonstrated in monkey and man. They require slightly increased voltages above those necessary for a response from stimulation of Area 4.

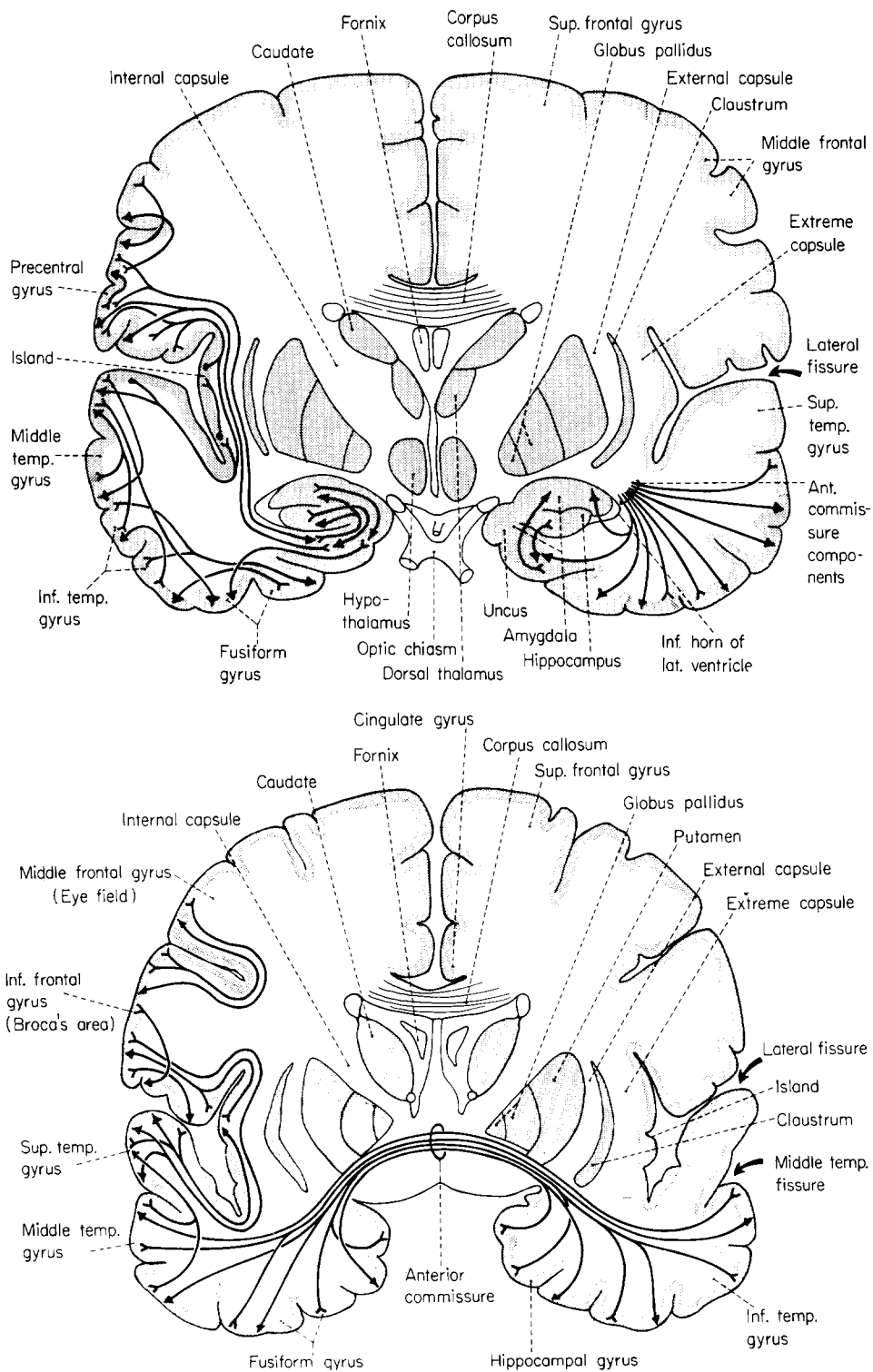


Fig. 9. *A*: the connections of the vestibular association centers by association bundles to other temporal, frontal and insular regions are indicated; *B*: the fiber bundles interconnecting the temporal lobes via the anterior commissure are shown. The short intralobular connections interrelating the portions of the Island of Reil are designated.

Impulses set up by vestibular stimulation probably come into consciousness at thalamic levels as a feeling of satisfactory equilibration when the area is functioning normally and as a sense of dizziness and of unstable equilibrium under clinical conditions. The impulses relayed from the thalamus to the vestibular receptive cortical area will have a similarly normal and clinical character. The full meaningfulness of these impulses for orienting the individual to his environment is probably built up in the vestibular association cortex, where there is a rich interrelation of association fascicles from various cortical areas.

Irritation of the vestibular cortical area may be due to lesions directly involving it (as was probably the situation in most of the cases here reported) or to a firing of the area from some other cortical region over association paths (SCHNEIDER *et al.* 1961; CROSBY *et al.* 1966) or possibly to a releasing of the vestibular cortex from the inhibitory effects of some cortical region which has been destroyed. It seems quite possible that the circling toward the side of the lesion exhibited by a monkey following a one-sided removal of the frontal cortex (CROSBY *et al.* 1966) may be affected by, and is possibly due, at least in part, to a removal of frontal lobe inhibition over the homolateral vestibular cortical area. Such circling may be demonstrated when an animal with one frontal lobe removed is excited, long after his ability to look to both right and left has returned. Consequently the circling is probably not primarily dependent on difficulties in viewing the visual field.

The feeling of instability with reference to the surroundings and the dizziness in patients with lesions of the vestibular cortex subsequently demonstrated are illustrated in Case 1 (at the age of 2 years), Case 2 (during the patient's peculiar "spells"), Case 3 (when he had seizures), Case 4 (when he was aroused from sleep), Case 5 (when dizzy, accompanied by a sensation of "floating on air") and Case 6 (during some of the lady's attacks). In several of these individuals (Cases 1, 4, 6 and 7), the vestibular impulses were built up (presumably in the vestibular association cortex) into a subjective feeling of whirling such as PENFIELD AND RASMUSSEN (1950) described for some of their patients.

The local application of alumina cream to produce seizures has been used by many experimenters (CALHOUN AND CROSBY 1965; CURE AND RASMUSSEN 1950; KOPELOFF *et al.* 1942; YOUMANS 1956). In monkeys (CALHOUN 1966) implantation of this cortical irritant on to inferior frontal cortex or the superior temporal area in or around the vestibular cortical region resulted in movements comparable to those described by patients with irritative lesions in the vestibular area. The monkeys either turned around or somersaulted.

Extrapyramidal or supplementary motor areas

A supplementary (or extrapyramidal) motor pattern has not as yet been demonstrated on the vestibular cortical area. This vestibular area, however, in primates (and probably in man) is interconnected with the supplementary motor areas in the insular region and in the middle and the inferior temporal gyri and the tip of the temporal lobe. In both the insular (FRONTERA 1955, 1956; SHOWERS AND LAUER 1961) and the temporal (DEJONGE 1968; DEJONGE AND CROSBY 1960; SCHNEIDER AND CROSBY 1954) supplementary motor areas a pattern of movements of the homolateral side of the face (Case 5), the contralateral side of the face and the homolateral and the contra-

lateral upper and lower extremities has been demonstrated in monkeys and there are indications of its existence in man, particularly in the temporal region.

Since the temporal lobe and the insular cortex are supplementary or extrapyramidal cortices, it is not surprising that seizures form an important part of the clinical picture in all the cases discussed. It is interesting that in Case 1, when the homolateral face area—a region concerned with cortical automatic associated movements—was, of necessity, partly destroyed during the operation, the patient showed some loss of automatic smiling on the side of the lesion (that is had less expression on his face on that side) but smiled normally on both sides of his face on command (Fig. 1E).

Hallucinations

The further symptoms exhibited by each of the patients with primary or secondary involvement of the vestibular cortex depended upon either the increase in the lesion itself, its shift from an irritative to a destructive lesion, or the irritation of association bundles which fired (or inhibited the activity of) other cortical regions. Thus one of the patients (Case 3) saw objects not present at the time but which he had seen before, and another (Case 2) heard music with which he was familiar although no music was being played. NIELSEN (1958) has stated that “the only portion of the cerebral cortex from which formed visual hallucinations can be produced by stimulation is area 19”. Some of the cases described by CRITCHLEY (1953) as showing visual perseveration may illustrate such a type of hallucination. The existence of visual “dreamy states” and the evocation of visual memories, such as NIELSEN (1946, 1958) accepted and such as those demonstrated by PENFIELD AND RASMUSSEN (1950) and others, may be associated with temporal lobe seizures. From our observations of this series of temporal lobectomies carried out on either the dominant or the non-dominant hemisphere it seems that the midtemporal lobe area (largely in the inferior temporal area for visual associations) is a region to which impulses are relayed not only from primary and secondary visual association areas but also from frontal cortex, amygdaloid-pyriform areas and various temporal regions. In this midtemporal region visual or auditory recall is apparently elaborated. Memories of past events may be evoked in the absence of direct visual stimulation by stimulation or irritation of association paths from cortical areas of different significance. Visual or auditory hallucinations will result.

It is evident that these highly complex midtemporal auditory and visual association areas can be activated by stimulation of, or irritation of, the temporal vestibular cortex with discharge over the short association paths which can be demonstrated to interconnect them.

If the tumor increases in size and destroys considerable portions of the vestibular area, the dizziness and the whirling will disappear. If it extends into the regions concerned with formed visual and auditory hallucinations (supposed to be usually in the non-dominant hemisphere) these too will vanish. The patient will have, instead, a loss of, or deficit in, past memory (Cases 2 and 5), perhaps because man has very well-developed auditory-visual association patterns on which many of his memories depend.

Involvements of island

Extension of the tumor directly dorsalward from the vestibular region of the tempo-

ral lobe into the insula (or its appearance there early) may add another feature to the clinical picture in the form of gustatory auras. Quite certainly nausea and stomach pain, and perhaps other visceral signs, may appear if there is irritation directly, or through association fibers, of the posterior island region. If the tumor invades and irritates the island first, as appears to have been the situation in Case 3, the nausea and the vomiting may be the first signs of the lesion. Gustatory cortical areas (PENFIELD AND RASMUSSEN 1950; CROSBY *et al.* 1962) probably extend from the base of the central fissure (FOERSTER 1936b) through the upper rostral part of the island region toward the hippocampal gyrus. In the dog (GORSCHKOW 1901a and b; VON BECHTEREW 1908) a gustatory area was demonstrated in the region comparable to the primate frontal operculum and upper rostral island. Studies of such clinical cases led PENFIELD AND RASMUSSEN (1950) to the conclusion that an irritative lesion of the caudal end of the insula gives sensations of pain in the stomach.

Broca's area, motor and visual loss

Extension around the island of the dominant hemisphere into Broca's area (CROSBY *et al.* 1962) and into the fibers underlying it (Fig. 9B), or perhaps, in some cases, inhibition of this area by irritative lesions of the superior temporal gyrus, may produce the more or less transient expressive aphasia seen in certain patients (Cases 1 and 3). Involvement of part of the motor cortex (or fibers of its discharge path, the pyramidal tract) may produce a partial paresis, such as the typical contralateral central facial paresis and incipient loss of voluntary motor activity of both upper extremities seen in Case 2. These effects may be either temporary or permanent depending upon whether they are due to postoperative edema versus actual destruction of the region. Moreover, a large tumor in the temporal lobe may press upon the pyramidal tract in the cerebral peduncle producing an incomplete contralateral hemiparesis. Compression of the optic tract as it overlies the cerebral peduncle may produce blindness in the contralateral visual field up to a homonymous hemianopsia. Extension of the lesion deep into the substance of the temporal lobe may interfere with the fibers in Meyers' loop with a resultant visual loss contralaterally in the superior temporal quadrant of the visual field.

Hippocampus and amygdala

If the hippocampal gyrus and the amygdala—areas regarded by many clinicians as parts of the temporal lobe but by most anatomists as portions of the limbic lobe—are the sites of irritative lesions or are fired over association bundles from other irritated cortical areas (SCHNEIDER *et al.* 1961, 1965), as the impulses spread out through the anterior temporal area (SCHNEIDER *et al.* 1963), typical psychomotor epilepsy may develop. This may be characterized by olfactory (and sometimes also, or only, gustatory) auras, déjà vu phenomena and temporal lobe seizures. The involvement of the various areas was evident in the trance-like state and déjà vu phenomena seen in Cases 1 and 2 as the seizures developed.

If the hippocampus is involved in the anterior temporal lobe lesion, there may be a lessening of cortical arousal; destruction of its rostroventral portion may affect recent memory, most markedly when the lesion is bilateral. Lesions in the frontotemporal

region may destroy paths between the frontal cortex, amygdala and hippocampus on the one hand and the hypothalamus on the other. Loss of cortical regulations, then, of the posterior tuberal region of the hypothalamus, by way of such paths as the medial forebrain bundle, stria terminalis, and possibly the fornix, may underlie the rage responses (Case 5) (MACLEAN AND DELGADO 1953; SAWA *et al.* 1954; URSIN 1960) or other changes in behavior manifested by some of these patients. Destruction of frontal cortex from a lesion may also lead to personality changes and may affect the patient's insight and judgment (Case 5). There are commissural connections between the frontal cortex and the superior temporal gyrus of one hemisphere and like cortical areas in the other hemisphere through the corpus callosum. The anterior portions of the other temporal gyri, the hippocampal gyri and the amygdalae of the two sides of the brain are interconnected through the anterior commissure (Fig. 9B) (FOX *et al.* 1948). It is not possible, then, to have a major lesion in any of these cortical regions without affecting to some degree the functioning of the contralateral cortical areas also.

Parietal lobe involvements

In addition to the cortical area in the temporal lobe related to the reception of impulses from brain stem vestibular areas, another region responding to peripheral vestibular stimulation has been demonstrated in experimental animals, in the parietal cortex (FREDERICKSON AND KORNHUBER 1965; SPIEGEL *et al.* 1965). Such a region is probably to be expected in man, since positional sense, coming into consciousness in the somesthetic cortex, will combine with impulses related to the position of the body in space (vestibular) in the interest of recognition and orientation of body parts. FREDERICKSON AND KORNHUBER (1965) demonstrated such a vestibular center at the ventral tip of the fissure separating area 5 and area 7 on the lateral surface of the monkey's cerebral hemisphere. Clinically, irritative lesions involving the inferior lip of the intraparietal fissure have been reported to produce sensations of the world whirling around the individual. Since the inferior frontal and the inferior parietal cortices are interconnected with each other over fascicles in such association bundles as the inferior occipitofrontal fasciculus (CROSBY *et al.* 1962; SCHNEIDER *et al.* 1965), it would not be surprising that irritative lesions in one area might produce both types of sensation of whirling at different times (Case 6). A parietal lobe ataxia, indicated in the stance of the little boy of Case 7, is due to the destruction of the parietopontine paths (CROSBY *et al.* 1962) from the superior parietal area.

The parietal association cortex is also a region of correlation of visual, auditory and somesthetic impulses through which the individual orients the objects around him with reference to himself. Irritative lesions invading the inferior parietal lobule may produce distortions in the object seen, suggesting an interference with a somesthetic visual correlative area. To patients with such an involvement, objects may appear to be crooked or inverted (HAYMAKER 1956). This is illustrated by the little boy (Case 7) who saw objects around him as if they were upside down. In this same general area, *i.e.*, in the superior parietal lobule, but probably not in precisely the same field, are associative proprioceptive-visual fields through the functioning of which the individual recognizes a part of his body as belonging to himself (CRITCHLEY 1953). A lesion in the

non-dominant hemisphere is said to affect this function more (perhaps only more usually) than one in the dominant half of the brain. Temporary loss of such an association (which has a visual as well as a somesthetic component) explains the child's statement (Case 7) that his face was disappearing. The transient inability of this child to see both hands draws attention to the role of visual association paths. The lesion was on one side. This transient episode was, then, not due to visual field loss since if it were, with slight movement of his head he would be able to bring both hands into focus. It does suggest a temporary loss or exhaustion of visual association components to parietal lobe areas and perhaps, through the corpus callosum, to contralateral visual cortex.

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SUMMARY

The case histories of 7 patients with brain tumors, who experienced vertigo or rotational movements, have been presented as a basis for a discussion of the central origin of these symptoms. Six of the neoplasms were in the frontotemporal region and 1 in the parietal area. The value of activation of the EEG focus, and of electrocorticography and depth electrode studies in determining the location and extent of the lesions has been demonstrated. A discussion of the anatomical basis for the development of these symptoms and their relationship to neoplasms and traumatic lesions is included.

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