FEAR IN EPILEPSY: a consideration of its nature and significance.

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### INTRODUCTION

When a patient, who complained of a "horrible sense of fear" (fear for no accountable reason) followed by an epileptic fit, was seen several questions arose; but only when it was determined that the fear was not anxiety regarding the imminence of an attack or the unpleasantness of any somatic sensations. Was this fear an aura? If so, did it signify that the causative epileptic activity was focal or generalized? If focal, did all patients with a similar symptom have a discharging lesion in the same situation? Where was this situation, left, right or bilateral? But this aura appeared to be an emotion. Could an emotion - a state of feeling - have a physical basis? Was the site of the discharging lesion that for the mechanism underlying the emotions, and in particular the emotion fear?

When this first case was seen, in 1949, a study of the literature failed to reveal the answers to these questions. Yet, in 1937, Papez (60) had posed his classical question, "Is emotion a magic product, or is it a physiologic process which depends on an anatomic mechanism?" He proposed, for theoretical consideration that it had an anatomic basis, and further stated, "Emotion is such an important function that its mechanism, whatever it is, should be placed on a structural basis". The importance of the emotions in medical practice and human behaviour has been amply asserted by scientific writer and novelist alike.

It did appear possible that, should fear occur as an aura in epilepsy and the localization of the epileptic focus could be ascertained, not only could a fear aura be an important clue to the localization of the lesion in other similar cases, but also the fear itself would be, of necessity, the product of physical (epileptic) activity in the cerebrum; and the localization of the focus would be, within limits, the structure elaborating this specific emotion.

Cobb (14), among others, had given consideration to the cerebral localizing value of the symptom fear; nevertheless, in his well known dissertation on the emotions, in 1950, he stated that there was little evidence that this symptom had any cerebral localizing value.

The earliest account of fear in epilepsy was by Hughlings Jackson (42) in 1880. He (43) published its

occurrence in a male, aged 51 years, whom he suspected had a temporal lobe tumour. He wrote; "the patient's face according to a competent eye witness of several seizures, one of which was carefully observed throughout, was pale and his expression was that of one facing an expected horror, but he was apparently not unconscious. The patient, himself, had a sense of fear and of impending death." In further amplification he wrote that he did not mean fear of the fit, "but fear which comes by itself" - 'isolated fear'. He expressed the view that it occurred "during slight discharges of very complex nervous arrangements representing parts of the body, especially organic parts, concerned in the manifestations of fear".

Gowers, 1881, (30) stated that "emotional aurae in all took the form of fear, vague alarm or intense terror".

In his description of psychical aurae Penfield (62) reported a case who had "as an invariable aura of an attack, a feeling of fear". The same author in his studies on cortical localization reported briefly three further cases. Gibbs (25) tabulated the aurae in psychomotor seizure discharges. Fear occurred in four of his

cases but no description of the character of the fear or details of the cases were presented. A case of epilepsy, reported by Earle, et al, (20) for a reason other than the study of fear, had "an initial feeling of fear and a perceptual illusion that things were far away" as an aura. A Russian publication (57) entitled "Localized Sense of Fear in Epileptics" was not available for study, but the use of the adjective 'localized' suggests their acquaintance with the writings of Hughlings Jackson (hh), who used the descriptive term 'isolated fear'.

The material in the literature is meagre. Penfield (63) found himself unable to "make any conclusions about a possible cortical representation of the complex phenomena that fear pepresents".

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### THESIS

The thesis proposed is: that the emotion fear, of the type to be defined, may occur as an aura in epilepsy; that this aura has a physical basis which can be localized within certain limits. Observations on the nature of this fear may be made.

### PRESENT STUDY, MATERIAL AND METHODS

A study of fear in epilepsy - fear occurring in an organic nervous state - was of interest: to ascertain whether premonitory to a fit it was an aura; an aura with focalizing significance, and so with many implications in the practice of neurology. That it might further an understanding of the nature of fear was possible.

Over the past four years, 40 cases have been selected and studied along clinical, electroencephalographic and pathological lines.

# CLINICAL STUDY

Only cases who, on clinical grounds, were suffering from epilepsy were included in the study. A minimum of three episodes of loss of consciousness, of a characteristic type, was the criterion for the clinical diagnosis

of epilepsy. The occurrence of aurae, other than fear, was accepted as an aid to diagnosis. In two cases no aura other than fear occurred, but in these the clinical diagnosis of epilepsy was well established.

The fear in each of the cases selected occurred immediately preceding a fit; not fear of a fit, but fear which, according to the patients, arose for no apparent reason - isolated.

Most sufferers from epilepsy experience some form of anxiety or fear during their aurae. This, at times, is an anxiety concerning the imminence of dissolution of consciousness or that they may be injured or behave inappropriately during the attack. In some, the crude sensations which constitute many aurae are unpleasant and fear producing.

There is another type of fear - fear such as was experienced by Jackson's case. This fear wells up unaccountably and apparently spontaneously, at times swamping consciousness. It may occur in any situation and irrespective of the prevailing mood or thought.

Its onset and departure are abrupt and its duration brief.

It may occur several times a day or only a few times in

twelve months. Thus it resembles epilepsy itself - and, it is suggested, is part of it. This latter fear is the subject under discussion.

In practice little difficulty was experienced in recognizing these cases, though it must be accepted that, in investigations concerning subjective experiences, the investigator is, to a point, at the mercy of the patient. These paroxysmal intense 'invasions' of the patients' affects by fear are, however, so alike from patient to patient, so removed from common experience, so closely related to the fits and so readily differentiated by the patients from ordinary anxiety concerning the fits, that they can be considered with the same objectivity as cortical paraesthesiae or other well-recognized sensory aurae.

A brief summary of all case histories, including clinical findings and results of investigations, is given in the Appendix. Two illustrative cases will now be presented:

Case F8: A female, aged by years, presented her complaint. "I am suddenly seized by a horrible feeling of terror" - an unaccountable terror of something un-

known. "I cannot describe it. After about one minute the terror passes off and is replaced by a horrible smell - not a real smell. It is unpleasant but I seem to recognize it. It is somewhat like the smell of burning hedges. After a further few seconds I have a dreadful feeling as though being choked by the smell and I suddenly sink to the ground. After a minute or so I am again perfectly well." These attacks had occurred frequently during the preceding twelve months. Although she sank to the ground, she only rarely lost consciousness and was at no time convulsed. She was anxious that her bizarre symptoms might be diagnosed as 'nerves'. Neurological examination revealed a right anosmia and atrophy of the right optic nerve. Further investigations revealed the presence of a space occupying lesion in the posterior portion of the floor of the right anterior fossa. A meningioma weighing 59 grams was successfully removed. She was subsequently free from these attacks.

Case Fh: A 23 year old medical student stated that, for  $1\frac{1}{2}$  years, he had been subject to frequent attacks: "several things happen suddenly and at about

epigastrium. I feel an intense fear, worse than and different to any fear ever; but yet the fear is peculiarly familiar as though previously experienced, even though at the time I know it has not been. Voices or the radio may sound different and I may have noises in my ears. These sensations lasted 15-20 seconds. Loss of consciousness usually, though not always, followed. The fear was commonly followed, whether or not loss of consciousness occurred, by a cold sweat, at times pallor of the face, and at times an urge to defaecate.

These two patients were of good intelligence, but they failed, as all did, to find words to describe, to their own satisfaction, their vivid experiences.

Even Dostoievsky (19) required an experiential parallel for description of his own aura (ecstacy tinged with fear): "clear as the edge of a precipice, strangely threatening".

The fear experienced was recognized as a precursor of an attack. The term 'familiar' was used, not to describe its lack of novelty, but to convey the subtly intense, though elusive, significance.

## Accompanying Aurae

In the two cases just summarized other aurae occurred. In 38 of the 40 cases, in this series, the fear was followed, accompanied or preceded, by another aura or aurae: olfactory, gustatory, auditory, vertiginous, epigastric, psychical, aphasic or motor. The aura in each individual case is recorded in the Appendix and in Table 1. The time relationship of these aurae to the fear is noted in Table 2, which shows the aura which immediately followed fear, when fear was the initial phenomenon, and also the aura which immediately preceded fear, when fear was not the initial phenomenon.

While the fear was commonly followed by a seizure, it at times occurred in brief paroxysms, either alone or accompanied by other aurae. One such case (F12) rarely lost consciousness, but suffered frequent attacks of paroxysmal fear, each attack lasting one-half to two minutes.

Two of her attacks of fear were personally observed.

During a consultation she abruptly interrupted herself

to say "it is here. I have it now." Outwardly, apart

from a mild agitation, she remained unchanged except

ļ	AURAE	EEG Focal - 25 cases	EEG Generalized 8 cases	EEG Normal 5 Cases	EEG Not done-2 cases
A.	SEN 1.	F6, F8, F18, F25	G2		NE2
	2. Auditory (Vestiginous)	F3, F7, F17, F20	G2, G5, G7	ZN 'SN 'TN	NEJ
m	PSYGHICAL  1. Aphasia	F5, F10, F15, F17	G2		
	<ol> <li>Perceptive (Visual, auditory</li> </ol>	F1, F2, F3, F4, F5, F6, F7, F8, F10, F11, F12, F15, F19, F18, F29, F20,F25	63, G4, G6	N2,N2,N4,N5	
	3. Automatism	F10, F12, F23, F24, F25	G1, G8		
ပ်	AUTONOMIC 1. Epigastric	Fl. F5, F9, F15, F18, F19, F24	G <b>11,</b> G8	NI, N2	LEN
	2. Palpitation, sweating, urge to defaccate or urinate	F1, F2, F4, F14, F15, F16	G <b>J</b>		NEL, NE2
å	MOTCR 1. Masticatory	F18, F20	G2 <b>,</b> G8	N2	NE2
		Cases with aurae: 23	Cases with aurae: 8	Cases with aurae: 5	Cases with aurae: 2

The aurae and EEG findings in 40 cases, with fear as an aura See Appendix A for fuller details. Two cases had no additional aurae.

TABLE 1.

AURA	(1) Fear Initial	(2) Other Aura Initial
Epigastric	1	9
Psychical	5	6
Vertiginous	1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1. 1	3
Auditory	1 180 000	<b>2</b>
Automati sm	3****	
Autonomic	3 €	
Olfactory	1 (1 N)	ere i jaron 🚅
Aphasic	•	1
		entired (me
TOTAL	15	23

Table 2 - Column (1) shows aura immediately
following fear when fear was initial.

Column (2) shows the aura preceeding
fear when the fear was not the initial
phenomenon.

for her withdrawal from conversation, and showed no signs of motor or autonomic activity. During the 'fear' she would answer a question with only yes or no. These monosyllabic responses were always appropriate. Recovery was abrupt. She suddenly appeared her usual bright self and behaved as though nothing untoward had happened. On both occasions she described the attacks as: "a feeling of terrible fear, the same as always. It makes me want to run away." She denied being afraid of anything known, "it just comes".

'isolated' fear, recognizing it as part of epilepsy and being frightened by the unpleasantness of the fear and its significance for her. This rather mixed emotional situation, as far as could be analyzed, consisted of a crude subjective feeling of unaccountable fear partially swamping consciousness, some anxiety evoked by conscious thought concerned with the unpleasantness of the feeling and its possible import; and an ability to view the fear rather objectively, almost as though it

were a severe pain in the leg.

A desire for flight, as seen in the latter case, either into solitude under the bedclothes or to a person, was usually present in most cases, but in only two cases was it compulsive.

When isolated fear occurred frequently, as in this case, a psychiatric label was the usual one. This case had been diagnosed thus, despite a history of three to four attacks of loss of consciousness immediately following upon the fear. Epanutin, grain 12, t.d.s. resulted in a recovery from the attacks of fear. Phenobarbitone being sedative rather than anticonvulsant was for this reason withheld. Case (N1) was labelled 'anxiety state', even by two neurologists, until a sub-arachnoid hemorrhage revealed the organic nature of the underlying condition.

Some patients presented the fear as the main complaint, even when they were anxious lest they be considered as "bordering on insanity", while others preferred to discuss the other auras for the same reason, so that the fear was elicited only

on direct questioning.

An attack which, in some ways, differed from the last mentioned was personally observed. She (Case G2) suddenly complained of a horrible smell. appeared agitated and frightened. She exclaimed "oh, I am afraid". Asked what she was afraid of. she replied "I don't know. I don't know." Asked whether she was frightened by the smell, she replied, "no, I am just afraid. I am so afraid." She then smacked her lips and stated that she heard voices. To further questions she replied in a jargon aphasia. After about 12 minutes she lost consciousness and had a generalized convulsive seizure with pallor, sweating, increased pulse rate and noisy passage of flatus. On regaining consciousness she was completely amnesic for the whole event.

# Duration of Fear

The duration of the fear in these cases ranged from a few seconds to a maximum of two minutes.

Therapy

As the clinical diagnosis in all cases was focal epilepsy, they were for this reason treated

with anti-convulsant drugs. Thirty-four cases
(tumour cases excluded) were studied for a sufficient period of time to note the response.

Phenobarbitone, Epanutin, Dilantin, Mesantoin
and occasionally Phenurone were used. The results
of drug therapy are shown in Table 3, below.

Response To Anticonvulsants	CASES
Complete Control	6
Good Control	10
Moderate Control	5
Slight Control	4
No Improvement	$\frac{4}{34}$ - TOTAL

Table 3 - Response to anti-convulsant therapy, phenobarbitone, hydantoinates and occasionally phenurone, of 34 cases of epilepsy with fear as an aura.

Four cases (12%) showed no response to anti-convulsant therapy. The others showed an improvement
which varied from slight control to complete recovery from the attacks. In a few cases, when the
seizures became infrequent, the fear and the other
aurae, though also less frequent, tended now to
occur without subsequent fits.

## The Incidence Of Fear In Epilepsy

In comparison with the small number of cases in the literature this study is large. It does not, however, represent the number which one individual, in the practice of neurology, may see naturally over a period of four years. This study represents the material available in the large practice of the National Hospital, Queens Square, London (35 cases) and the Department of Neurology, University of California Hospital, San Francisco (5 cases). The cases being selected in this way, the incidence cannot be reasonably assessed.

Many patients hesitate to mention the fear, giving as a reason: "the doctor might think I was going mental;" not recognizing its significance some fail to mention it, "because I was not asked". Other aurae are not revealed for similar reasons. The loss of consciousness is usually the main complaint, and aurae often are elicited only by direct questioning, unless they occur frequently without being followed by a 'fit'. That fear in epilepsy is not so rare as might be expected from the literature is probable.

# ELECTROENCEPHALOGRAPHIC STUDY

Twelve-eighteen electrode electroencephalographic studies were obtained in 38 cases. The EEG studies were of a routine kind, with hyperventilation as the routine activation method. Sleep, as an activation technique, was employed only when the resting and hyper-ventilation recordings were normal. Using sleep, four cases with previously normal records showed abnormal EEG changes. Details of the EEG findings in individual cases are shown in the Appendix. Criteria for abnormality were those accepted by the British EEG Society. Records, with only mild slowing, irregularity or poor synchronism, which could only be classified as borderline, were considered, for the purpose of this study, as normal.

Of the 38 EEG studies (Table 4), 33 (87%) were definitely abnormal. In 25 (76%) of these abnormal cases the changes were focal. In 8 cases (24%) the changes were generalized, but in one of these more marked on one side. In 5 cases (13%) the EEG was normal.

EEG FINDINGS	CASES
Focal Abnormality	
Unilateral	22
Bilateral but	
predominantly unilateral	<u>3</u> 25
Total Focal	25
Generalized Abnormality	
Symmetrical	7
With greater abnormality	•
on one side	1
Total Generalized	8
Normal	5
	===
Total Number EEG Studies	38

Table 4: EEG findings in 38 cases of epilepsy with fear as an aura.

The situation of the EEG focus, in the 25 cases with well marked focal changes, is shown in Table 5 below.

EEG LOCALIZATION	CASES
Anterior-Temporal - Low-Frontal	12
Temporal	8
Posterior Temporal	2
Anterior Temporal	1
Frontal	2
TOTAL	25

Table 5: Focal localization of EEG abnormality in 25 cases of epilepsy with fear as an aura.

The EEG changes varied from case to case and no specific pattern was observed. Five to seven per second irregular slowing or even slow delta waves were seen, and less frequently spiking potentials occurred. Fig. 1,2, and 3 are examples of these abnormal changes. In three cases, there was EEG evidence of bilateral focal changes, though, in each, the abnormality was greater on one side. These bilateral foci were in the frontal lobe (one case) and temporal lobe (two cases).

### Correlation of EEG and Aurae

Table 1 shows the aurae for cases in the different EEG groups. There was no essential difference between the aurae in cases with a focal electroencephalographic change and in cases with a generalized abnormal or normal electroencephalogram.

#### PATHOLOGICAL STUDY

The cerebral pathology was verified at operation or post-mortem in 6 cases. In 5 cases a tumour was removed or verified at operation. Two tumours removed were meningiomas (cases F5, F8), one filling the posterior two-thirds of the floor of the right anterior fossa, the other situated over the left parietal convexity. Three tumours (cases F20, NE2, F6) were astrocytomas. These were situated within the temporal lobe. Two were small, situated deep within the lobe. The third was large, more malignant, and invaded the whole lobe. An x-ray of the skull (case F6) showed intracranial calcification within the temporal lobe (Fig. 4). One case (N1) showed, post-mortem, an aneurysm at the junction of the left anterior cerebral and anterior communicating arteries (Fig. 5).

A further well documented unpublished case, a female aged 35 years, may be included, though she was not personally seen. A report from the Mayo Clinic, Rochester, Minnesota gave the information that, when admitted, she showed, on examination, bi-lateral papilloedema, aphasia and a right homonymous

hemianopia. She rapidly deteriorated and died during operation. The operation was continued and a large malignant glioma in the left temporal lobe was discovered. Her mother in a personal communication wrote that her daughter's first complaint, two years previously, was "I have such terrible spells of fear". To the doctor's question, "what is the cause of your fear?", she replied "I do not know myself - just an awful fear". She later had several brief spells of aphasia. The addition of this case increases the total of gliomas, within the temporal lobe, to four.

The pathology, as far as could be reasonably ascertained in all hi cases is shown in table 6. In il cases a history of severe head injury was obtained, suggesting that a traumatic cerebral lesion was the basis for the epilepsy. In five cases the underlying pathology was considered to be vascular. In one case (F17) a localized dilatation of the left temporal horn was revealed by pneumomencephalographic examination. A history of severe head injury was available in this case. A generalized

PATHOLOGY	9	CASES	
Trauma		11	
Neoplasm			
Meningioma Glioma	2 4	6	
Vascular			
Aneurysm Arteriosclerosis Thrombophlebitis Haemorrhage	1 3 1	6	
Cerebral Atrophy (s	ir e	ncephal	logram)
Generalized Unilateral Focal (Temporal) Suspected	1 1 1	4	
Unknown		14	
		41 TO	TAL

Table 6: Pathological diagnosis, verified and unverified, in a series of hl cases with an aura of Fear (Mayo Clinic case included).

dilatation suggestive of cortical atrophy was seen in one case (F2) and a left-sided cortical dilatation in another (F9). Each of these latter three cases showed a fronto-temporal EEG focus.

In 14 cases there was insufficient evidence on which to base a pathological diagnosis. In the nine cases with verified pathological lesions, the lesion was on the left side in six and on the right in three.

Though the EEG did not correctly localize in all cases, it correctly lateralized in all the cases with a verified pathology.

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### DISCUSSION

### Fear As An Aura

Is the fear described in these cases an aura? Galen (72) first used the term aura. An epileptic boy, describing the sensation which he experienced prior to a fit, likened it to a cold breeze (aura) blowing upon a part of his body. Galen recognizing this sensation as a part of the epileptic process and as a warning of the imminence of a fit, henceforth used the term aura for all such sensations. In this sense the word, aura, is still used.

The fear, as it occurs in the cases in this study and in those cited from the literature, preceds the epileptic fit, is recognized by the patient as a warning of the imminence of the fit and even as part of it. When the epilepsy is treated, either by removal of the underlying cause or by drugs, the fear also responds. When the fear occurs frequently without a fit ensuing, it still has the same epileptic quality: paroxysmal, unexpected and completely unrelated to prevailing mood, thoughts or somatic sensations.

On the basis of the customary definition,
fear as it occurs in the cases in this study and
in those cited from the literature, must be termed
an aura.

### Definition

The subject under discussion may be defined as the fear which occurs as an aura in epilepsy, usually but not always accompanied by other aurae; not anxiety or apprehension induced by other aurae either by virtue of their unpleasantness or strangeness, or by virtue of their warning of the imminence of a fit; but that fear, or terror, which wells up unaccountably (for the patient), and not consciously relative to thoughts or experience. By description it is strange, different from any 'normal' accountable experience of fear; yet, with some having a strange quality of unnatural familiarity as though an experience in a distant past, vividly known and yet unrecognized, significant though meaningless; all despite a conscious knowledge that the experience is within the present and merely signifying that the attack has already begun. It appears to be of

abrupt onset, of brief duration seldom lasting two minutes, severe in intensity, and invariably without content.

# The Physical Basis For This Fear

Galen recognized that an aura was neither divine guidance nor visitation by evil spirits, but an integral part - the initial part - of the attack.

Aretaeus of Cappodocia (77) described in particular visual, auditory, and olfactory aurae. Richard Bright (8) in 1831 reported focal motor attacks occurring in a horseman who had suffered, in a fall, a depressed parietal fracture. These attacks ceased following upon elevation of the bone. The significance of these events, in terms of cerebral physiology, escaped recognition.

The significance of aurae in terms of cerebral function was first appreciated by Hughlings Jackson (44). Starting with focal motor attacks, his brilliant inductive reasoning led to the hypothesis that during a fit a local discharge occurred in an area of the cerebral gray matter; and that the march of motor events in the seizure reflected the spread

of this discharging state to adjacent areas. He concluded that the irritative discharge was in certain convolutions of the opposite hemisphere - that motor function had a cerebral localization.

Fritsch and Hitzig (23) and Ferrier (21)
demonstrated the validity of this hypothesis; the
former by experimental electrical stimulation of
the cortex; the latter by ablation experiments.

By recognizing that the aura was the initial part
of the attack - that the aura was the manifestation
of the cortical discharge - Hughlings Jackson led
the discovery of the functional anatomy of the
cerebral cortex; and was able to present a physical
basis for a rational explanation of all types of
epileptic seizures. That the aura is the initial
part of the epileptic seizure now forms, in neurological practice, the basis for clinical localization
of cerebral pathology in symptomatic epilepsy.

Reproduction of aurae by cortical stimulation
has been extensively used, by Penfield (63), Green
(31), Bailey (2) and others, to discover the situation
of the discharge responsible for the aura. The

area 'firing off' may then be removed surgically, for the relief, in some cases, of the epileptic disorder. In Penfield's (63) series of 125 cases, in whom the localization was verified by the reproduction of the aura by cortical stimulation, the discharging site coincided in 97% with gross evidence of electrical abnormality, as shown by cortical recording.

Thus an aura is part - the initial part - of a focal seizure and is the result of physical activity in an anatomical situation which may be localized.

Fear as an aura should, on the same basis, represent focal cerebral activity.

. වැටුව කතා දෙවැනි සහම් ලේ වුනුවේ වුනුවේ විනුකුරු දිහිණ පුරුවානු නොවැනි වෙනුවේ.

## THE CEREBRAL LOCALIZATION OF THE ACTIVITY UNDER-LYING THE, AURA, FEAR

cerebral activity, it is to be expected: that the site from which it arises will be in close anatomic relationship to the foci of origin of the accompanying aurae: that the electroencephalogram should lateralize and, in a significant proportion of the cases, localize, within limits, the site of origin of the causative abnormal electrical activity: that in the cases with verified pathology, the lesions will be either in identical situations, or will involve structures with a common neuronal integration.

The evidence adduced from a study of the accompanying aurae, electroencephalograms and path-ology will be examined.

# Accompanying Aurae

Jackson (44) recognized, from his pathological and clinical studies, that the site of discharge of olfactory and gustatory aurae was in the region of the uncus. Penfield (62), by stimulation of the uncus, has reproduced an olfactory aura in a patient

who had been liable to uncinate seizures. In the literature, Jackson (44), Gowers (30), Penfield (62) (63) (64), Gibbs (25) (27), Green (31) (32), Bailey (2), and others have shown by clinical, pathological and stimulation studies that aurae such as those which, in the present series, accompanied fear have a temporal lobe localization.

The crude sensory aurae (olfactory, gustatory, auditory, vertiginous) have a well mapped localization. The more complicated psychical aurae, though recognized as being temporal lobe in origin, are anatomically less well defined. Penfield (62, 63,64,65), in the years since 1933, has, by utilizing electrocorticography and cortical stimulation methods during surgery, carefully mapped out the situations from which aurae can be reproduced. Fig. 6, modified from Penfield (63), shows the site of auditory, epigastric, perceptual and psychical aurae, as reproduced by stimulation of the lateral aspect of the temporal lobe. The olfactory and gustatory aurae are the only ones recognized as being exclusive to the medial aspect of the temporal lobe.

The difficulty of access to the medial aspect of the temporal lobe has, in man, allowed little study of this area by direct stimulation and electrocorticographic methods.

Magnus et al. (54) reported 34 cases with masticatory aurae. Fear and thoughts related to fear, at times associated with an epigastric sensation, occurred in eight of these. In some, an abdominal or thoracic sensation, often associated with fear, was obtained on stimulation of the insula and base of the temporal lobe. In all, the evidence was of temporal lobe epilepsy.

In one (G.P.) of the three cases (with fear as an aura) reported by Penfield (63), on electrical stimulation during operation, a feeling of fear was reproduced from the fusiform gyrus on the left.

Resection of the anterior end of the temporal lobe resulted in freedom from attacks during the five years of observation.

If one can accept that aurae accompanying
fear arise from a situation closely related to that
producing fear, the evidence is that the localization

for fear is in close anatomic relationship or neuronal integration with the temporal lobe.

That stimulation of the intermediate frontal area (Penfield's case) resulted in a sensation of fear is not against this viewpoint. In all cases in which the aura, fear, was reproduced by electrical stimulation there was already a diseased site present.

Stimulation of a diseased site sets in train
the march of events which occurs in the particular
seizure that the patient is liable to. The resulting
arrae merely reflect the neuronal pathways which
the march of events follows, and merely indicate
that these areas are in neuronal connection with
the situation stimulated. The significant point
is that on no occasion has stimulation of the lateral
surface of the brain, in a patient without epilepsy
of this sort, resulted in the production of the
arra, fear, while it has resulted in the crude sensory
arrae, such as auditory, visual, vertiginous, aphasic,
etc. The same arguments can be used against the
hypothesis that the localization of fear is in

close anatomic relationship with the localization of the other accompanying aurae. It is, however, significant that, in 36 out of the 38 cases with an aura of fear, the accompanying aura has a known temporal lobe localization. In 15 (65%) of the cases in which fear was preceded by another aura, the aura was either epigastric or psychical. A psychical aura occurred in 29 cases (72%) and an epigastric aura in 13 cases (32%). An olfactory aura occurred in 5 cases (12.5%).

It is suggested from these findings: that
the cortical mechanism which is set in activity to
result in the aura, fear, is in the temporal lobe:
That in view of the non-production of fear by
cortical stimulation of non-diseased temporal lobes
the situation of the neuronal mechanism is on the
medial aspect.

# Electroencephalography

The value of the electroencephalogram in epilepsy is now well established, not only in demonstrating the presence but also the localization of abnormal activity.

In the early days of the work on electroencephalography in epilepsy, Gibbs and Lennox (26) found abnormal activity in almost 100% of patients with clinical epilepsy. In time, it was found that to produce such a high percentage activation techniques had to be used. Schwab (72) presenting the present situation stated that, in 75% to 85% of patients with clinical epilepsy, the inter-seizure record was abnormal when only over-breathing was used for activation, but in almost 100% using additional techniques: sleep, metrazole, photic stimulation, etc. In the present series (using over-breathing and sleep activation techniques) an abnormal rate of 89.5% compares not unfavorably.

In this study, however, the EEG was considered not as a method of diagnosing epilepsy - a diagnosis already clinically established - but in aiding localization of the underlying lesion. Culbreth (16) found that the EEG correctly localized in 85% of tumour cases. In the present series the EEG localization was correct for three temporal lobe gliomas. The EEG localization for the sub-frontal meningioma and the parietal meningioma was however

also in the anterior temporal area. In these latter two cases, the EEG correlated more closely with the clinical localization than with the verified anatomical situation of the lesion. The EEG localizes the site of physiologically abnormal activity, and so it is likely that though the tumour was not correctly localized the 'discharging' focus was.

The sub-frontal meningioma, in all likelihood (though this was not verified at operation) was in immediate relationship with the uncus, thus 'firing off' the uncal discharge and possibly the fear.

It is more difficult to explain the failure of the EEG to localize accurately the parietal meningioma. An explanation is, however, possible. When aged 4 years this patient suffered a severe head injury. As so often happens in head injuries, a lesion in the temporal pole may have been produced. In the presence of raised intra-cramial pressure, this previously inactive lesion became a discharging focus. This view is compatible with Grey Walter's (79) findings that the EEG focus may be at a distance

'Uncinate' epilepsy, which occurred in each of these cases, is so well localized to the uncus that it is difficult not to accept that the discharging site was in fact that shown by the EEG and was in the region of the uncus.

Jasper and Kershman (45), and Jasper (46)
described the temporal origin of the electroencephalographic disturbances in most patients with
psycho-motor seizures. Gibbs et al. (25), (27)
confirmed the temporal and fronto-temporal origin
of electrical disturbances in patients with psychomotor seizures. Hill (40), however, found that
such seizures are associated with EEG disturbances
in the temporal region only twice as frequently as
they are with EEG foci in other areas of the brain.
Nevertheless, the evidence in favour of the former
view is abundant.

Jasper et al. (47), in an analysis of 500 cases, who had been operated on by Penfield, concluded that the psychomotor form of epilepsy was not a specific form of epilepsy but merely one form

of focal cortical seizure arising within the temporal lobe; though many seizures, arising within the temporal lobe, with the same form of EEG, could not be described as psychomotor. In their series, 75% of 91 cases showed clear focal evidence in one temporal region in the pre-operative EEG.

A decided improvement in 66% of those operated on confirmed the accuracy of the EEG localization and the situation of the cortical discharge.

Bailey and Gibbs (2) found anterior temporal foci in 24 of 25 cases with psychomotor epilepsy. The focus was unilateral in 18 and bilateral in 7. Ten per cent of these cases had uncinate fits with 'deja vu'. The temporal lobe origin was confirmed by electrocorticograms, demonstration of pathological changes in portions removed at surgery, and improvement in the epilepsy following temporal lobe gyrectomy or lobectomy. The failure of anterior lobectomy in some cases, despite the presence of cortical spikes, was interpreted by Green et al. (32) as indicating that psychomotor seizures may be fired from an area of neuronal injury within the

subcortical connections of the anterior temporal lobe.

While most of the cases in the present series show some features of psychomotor epilepsy (temporal lobe epilepsy variant) as described by Gibbs, the significant EEG finding is the localization - in the temporal or anterior temporal-low frontal area - in 75% of those with abnormal EEG studies; a localization which coincides with that of psychomotor epilepsy.

The EEG thus strongly confirms a temporal lobe localization for these cases. If the conclusion arrived at by Jasper, et al. (47) "that the regions primarily involved in psychomotor attacks may be within or subjacent to the temporal lobe, probably in the archipallium or in the sub-cortical structures related to the temporal lobe" is correct, the conclusion that the localization of the activity producing fear in epilepsy is on the medial aspect of the temporal lobe, is thereby supported.

No conclusion can be drawn, regarding the focus of discharge, from the cases with a generalized

abnormal or normal EEG's; but, as seen in Table 1, in 38 cases, irrespective of the findings, the aurae suggest a temporal lobe discharge.

### Pathology

While Jackson suspected that the case he described, with fear as part of the aura, had a temporal lobe tumour, the diagnosis remained unverified. There are, however, three cases in the literature with verified lesions. Penfield (62) found a glioma deep in the temporal lobe in a patient, who had fear as an invariable aura. In a similar case, Earle et al. (20) found macroscopic and microscopic abnormality in the first temporal convolution anteriorly, extending into the uncus and the hippocampal gyrus. Pampiglione (59) reported a case with uncinate epilepsy resulting from an aneurysm of the internal carotid artery. Fig. 7 shows the aneurysm nestling against the medial aspect of the left uncus. In his report, he did not describe fear but, in a personal communication, stated that fear accompanied an olfactory aura.

An analysis, of these three cases and the 8

cases with verified pathology in the present series, shows that the lesion was within the substance of one temporal lobe in 6 cases and on the medial surface of the temporal lobe in 2 cases (Fig. 9).

The evidence for the temporal lobe origin of the aura in the remaining two tumours has been pre-viously discussed.

Some difficulty is presented by Case Nl. aneurysm (Fig. 5) was small in its deflated postmortem state. The size it attained during possible periodic dilatation is unknown, but it probably did not enlarge sufficiently to stimulate the medial aspect of the temporal lobe. Its immediate anatomical relationship was with the anterior portion of the cingulate gyrus. Its effects, however, were paromysmal and were probably the result of periodic anoxaemia of the area of distribution of the anterior cerebral artery. Critchley (15) described a case (Case 2) in which there was softening of the left para-central lobule and the gyrus cinguli following thrombosis of the anterior cerebral artery. initial symptoms were fright, followed by a hysterical

fit, semicoma and unconsciousness. Though in the present case there was no histological evidence of cortical damage, it is likely that a similar situation was affected. In the present case. however, the epigastric, vertiginous and perceptual aurae suggest that the temporal lobe was in some way involved. The cingulate gyrus (Fig. 8) according to Papez (60), Cobb (14), Fulton (24), Maclean (52). Brodal (9), and Weiner (81), sends fibres to the hippocampus and receives fibres from the fornix. mamillary body and the anterior thalamic nuclei. Its connections with the hippocampus may explain the temporal lobe components of the aura - LeGros Clark (12) speculated whether activity in this area may play a part in activation or inhibition of the neopallium. Its connections with the diencephalic mechanism may explain the post-ictal sense of dying - a state which was subjectively very different from the feeling of fear preceding the attack. The initial fear was a feeling state, the post-ictal a physical state.

In the other cases, in this series, the local-

ization of the pathology was not verified. The high incidence of trauma may, however, be significant. A focal abnormality was found, by Greenblatt (33) and Gibbs, et al. (28), to correlate well with the site of injury and the occurrence of focal fits. The relationship of temporal lobe features, EEG focalization and trauma in these cases suggests that trauma to the temporal lobe did occur.

Of the three cases with air encephalographic evidence of cerebral atrophy, in only one was this confined to the temporal horn. On the basis of the history, this was probably the result of trauma.

Earle et al. (20), in their study of 157 cases in whom temporal lobe seizures originated in the temporal lobe, found that, in 100 (63%), pathological study suggested compression or anoxaemia at birth as the cause. The uncus, hippocampal gyrus and post temporal convolution were the areas most frequently involved. The possibility that some of the 1½ cases in this series, with unknown pathology, had a similar congenital lesion is likely.

#### Conclusion

The pathological, electroencephalographic and clinical evidences present a congruent account and suggest that the lesions resulting in the aura, fear, are in the temporal lobe, most probably on the medial surface, either on the right or left side.

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# THE ANATOMY OF THE MECHANISM UNDERLYING THE AURA, FEAR

While it can be concluded that the aura, fear, is a clue to the site of the focus of epileptic discharge, no safe deduction can be made, on the basis of this study, regarding the precise anatomy of this site. This fear, however, though it is unusual and the product of abnormal physiology, is a pure emotion. While it has received scant attention, the anatomy of the emotions has been extensively studied.

### The Anatomy Of The Emotions

A tacit recognition that the emotions had their source within the cranial vault has existed since the dawn of psycho-surgery - the attempts in early civilization to release the evil spirits through holes trepanned through the cranial vault.

The term emotion implies two conditions: The physical state, and the feeling state. Thus when we say a person is frightened we imply that there are external manifestations of fear: pallor, tacherycardia, dryness of the mouth, dilated pupils,

tremor of hands, and bowel movements; and that
there is, as there must be, an experience of the
sensations resulting from these physical activities.
We also imply a feeling of fear - a psychical state
we define as being neither conscious thought nor
organic sensation, based on the physical manifestations
of the fear state.

The famous James-Lange Theory of the Emotions has been extensively criticised on the basis, as Raw (67) stated, that in effect it meant no more than that an emotion is actually the sensation of the visceral changes which accompany it.

Cannon (10), (11), Head (35), Ransom (68),

Dana (17), Bard (3), (4), (5), (6), Masserman (55),

Penfield (61), Papez (60), Maclean (52), (53), Cobb

(14), Fulton (24), Spiegel (74), and others have by

their researches and speculations founded the

knowledge there is of the possible mechanism under
lying the emotions.

The anatomy of the emotions, as yet but insecurely and imperfectly established, will be considered briefly; and only those parts which may have significance with relation to the study of fear.

Goltz (29) in 1891 and Sherrington (73) in 1904 observed 'angry' reactions in partially decorticated dogs and cats. In his careful and extensive experiments Bard (3), (5), (6) demonstrated that the principal centre for the motor expression of 'sham rage' was in the posterior hypothalamus. He called it sham rage because coordinated motor behaviour indicative of rage was emphatically expressed, but there was no evidence that the animal was feeling any emotion. Masserman (55), Rioch and Brenner (69), and Hess (39) have confirmed, by stimulation experiments, the hypothalamic localization of the rage reactions. Fulton (24) described the case of sham rage occurring in a man after Zenkers fluid had been accidently applied to the hypothalamus during an operation. In the experiments of Bard, emotional expression occurred in the absence of the cerebral hemispheres and the dorsal thalamus. For subjective emotional experience he concluded the cortex to be essential.

It can then be said that, for the expression

of fear, the sensori-motor mechanism is the hypothalamus acting through the sympathetic and parasympathetic nervous systems.

The autonomic functions of the hypothalamus was first demonstrated by Karpus and Kreidl (48).

Their work has been amply confirmed and extended.

The cortex, as shown by Bard, is essential for emotional experience. On the basis of comparative anatomy, the cortex connected closely with the hypothalamus is that of the medial wall of the hemisphere, while the lateral wall is more closely integrated with the dorsal thalamus (Herrick (38)).

Architecturally the medial wall is composed of the palaeo-cortex (transitional) which includes pyrifirm, uncus, anterior hypocampus, cingulum, and subcallosal regions. Part of it three layered, and part of it six layered; and the archipallium, the primitive rhinencephalon, i.e., olfactory bulb, tubercle, septum, dentate gyrus, and main part of hippocampus, all definitely three layered. The lateral wall, the neopallium is entirely a six layered structure.

It might be expected that the primitive medial wall of the hemisphere is closely integrated with the hypothalamus, not only anatomically but also functionally.

In 1937 Papez (60) synthesized the available evidence and proposed that the hypothalamus, the anterior thalamic nuclei, the gyrus cinguli, the hippocampus, and their inter-connections, constitute a harmonious mechanism which may elaborate the functions of central emotion, as well as participate in emotional expression. Most of the experimental support for this view has accrued since. His paper was referred to by Cobb (lh) as a farsighted synthesis supported by much recent research. Fulton (2h) stated, "this shrewd deduction has been richly vindicated by all recent experimental work".

Fulton further stated that strychnine and other stimulation studies indicate that "the hippocampus and the structures with which it is immediately connected do in fact constitute a harmonious mechanism, and through the higher areas, such as the hippocampal gyrus, cingulate, orbital surface, and

temporal tip, the visceral brain influences processes taking place in the neopallium and the neopallium in turn influences the visceral brain. The term visceral brain was used by Maclean for the phylogenetically old and architecturally primitive complex: fornix, hippocampus and stria terminalis, which takes origin in the amygdala; visceral because of its functions, and having close anatomic relationship with the autonomic nervous system, and the sensation of smell, and direct expression through the effector system of the hypothalamus.

Pribram and Maclean (66) have shown, by their neuronographic methods, the close neuronal relationship which does exist between the medial aspect of the temporal lobe and the neopallium. The close integration between the archipallium and hypothalamus has long been established, while it still remains to be shown whether the neopallium and hypothalamus are directly connected. Le Gros Clark and Meyer (13), and Ingram (41) have discussed this at length without arriving at a final conclusion.

Brodal (9) concluded that the hippocampus,
the largest part of the archipallium was an integrating mechanism coordinating olfactory and other

sensory impulses to make a cortical control mechanism for autonomic reflexes.

Head (35) believed that sensations received emotional content when nervous impulses reached the thalamus. Brain (7) agreed with Head's view without giving reasons. All sensations with the exception of the olfactory probably pass through the thalamus, and yet smells do have marked emotive effects.

spiegel (75) failed to produce emotional reactions from circumscribed lesions in the thalamic region. Lashley (49), in his critical analysis of the role played by the thalamus in emotions, concluded that "the affective changes resulting from thalamic lesions are restricted to a small group of somesthetic sensations and cannot be interpreted as a general change in affectivity".

He concluded that the only part of the thalamic theory of emotions which had factual support is the localization of the motor centres for emotional expression within the hypothalamus. While Head's view was a stimulus to a physical approach to the

emotions the weight of evidence, as expressed by Lashley, is against it.

The emotion experienced by the patients in this series was subjective. If the well defined anatomical system proposed by Papez is the mechanism which forms the basis of emotional elaboration, it is likely that the activity resulting in the aura, fear. occurs in its cortical portion. The case described by Earle and Case Nl in this series lend strong support to his proposition. Van der Horst (78). reporting a case of affective epilepsy (anger being the emotion expressed), with a gliomatous lesion affecting the hippocampus, described it as an experiment designed by nature to emphasize the validity of Papez's conclusion. All the evidence presented in the present study either supports or is consistent with this conclusion.

7

### ON THE NATURE OF FEAR IN EPILEPSY

This fear, experienced as an aura in epilepsy, is unusual; not merely qualitatively, for this is unobservable except to the patient, but for its isolation from thought, sensation, or memory.

Normal accountable fears have a basis, while many psychiatric fears are slenderly based, though reasonable, others apparently completely irrational (Ryle) (70)).

What relationship is there between the epileptic fear and these fears?

# Fear and Autonomic Activity

The question might be raised, whether activity in the hypothalamus may result in unpleasant visceral sensations which, in turn, are interpreted as fear.

Large doses of Adrenaline, intense vertigo (Gowers)

(30) or other shock producing mechanism (Ryle) (71)

do result in a state of agitation akin to fright.

In these, however, the autonomic manifestations are primary and are described as either resulting in a feeling "as though frightened" or else by their unpleasantness "frighten".

Ryle (71) studied these mechanisms with particular regard to 'angor animi'. He wrote "what strange force is it that can stir and with such alarming intensity a consciousness of the imminence of death - a state of which there can never have been any actual experience; and yet when death is imminent this sense is absent. He concluded that these manifestations were autonomic in mechanism. He noted that, while the symptoms were unpleasant and during early attacks fear provoking, in later attacks, though the symptoms were still equally unpleasant, this objective emotional state fear ceased to be present. He carefully differentiated between this sense of dying and a subjective emotional state.

In the cases of this series it can be said,
even as Jackson (44) said of his cases, that the
fear was a "pure feeling" and "isolated", and there
was no evidence of preceding autonomic activity.
In the 3 cases in which autonomic manifestations did
occur as in Case F4, they followed the fear, probably
indicating spread of epileptic activity. If the

autonomic sensations had been secondary to a mental process, they should have been more frequent.

Fear And Sensations

Somatic sensations or emotions are frequently referred to as feelings. The distinction between them must be made. Feelings of the former type, e.g., of pain, cold, hunger, etc. are localized by the subject to a part and are the product of stimulation of receptor end organs in these parts, or of the cortical areas representing these parts. The feeling state, emotion, on the other hand is not localized to any particular part, but is a general state of feeling.

Sensations may produce a general state of feeling. Thus a painful stimulation or loud noise may be fear provoking. A loud noise may result in a startle reaction which is partly an experience of fear, and partly a physical state with autonomic accompaniments. A pain may result in a fear experience in addition to the localized pain feeling. The pain, or other nociceptor stimulus, has connotations of threat. To what extent these produce

fear via vegetative mechanisms and at the same time through thought processes must vary.

No sensory stimulus by itself is perceived as fear in a manner similar to the perception of visual objects, smells, etc. Physiologically the visual stimuli from a stick or a snake are the same (Hebb) (36). The feeling state fear following on the stimuli from the snake, is a product of the whole visual pattern. Field experiments on recognition of predatory birds by small birds on which they prey, have shown that two visual characters. outline and movement, are required to produce a fear reaction. When a silhouette, which when moved in one direction causes fear (apparently because it is mistaken for a predatory bird) is moved in the opposite direction, it does little more than attract attention (Hartley) (34). The sensory impulses are unchanged. To produce fear, the sensory data has to be analyzed in terms of the characters, form and movement, and the synthesis determines the subsequent events. Neither the form by itself, nor the movement by itself sets into activity the sensory motor mechanism. The combination appears to be the key and this combination is an abstraction from the visual data. Such an abstraction is the product of neuronal activity, and is in the nature of simple thought.

Fear And Mental Processes

While sensations resulting from stimulation of receptor end organs may set in action a train of thoughts which have an affective component, anticipation of events, real or fancied, may be equally fear provoking. The soldier preparing for battle anticipates danger and exhibits 'normal' fear. This, too, must be on a physical basis, many neuronal connections being required to elaborate the assocatations.

That there is EEG evidence of local neuronal activity during the process of thinking was concluded by Grey Walter (80). The alpha rhythm, which is regarded as the indication that the visual cortex is in a state of rest 'blocks' with attention, suggesting that "these cerebral regions which were previously responding regularly to external stimuli are mobilized for internal service".

In the enduring anxieties, phobias, and panics, as described by Henderson, while it cannot be said that they are the product of any specific area, there must be an elaboration by some neuronal activity. Leucotomies have shown in man how mood and affectivity are altered on severence of some frontal pathways. Since Moniz (58) in 1935 introduced the operation as a therapeutic measure in affective and psychotic disorders, much confirmation has accrued in the voluminous literature on the subject. Among the writers the contributions of Meyer and McLardy (56), Freeman and Watts (22), Dax and Radley-Smith (18) are important.

# The Fear In Epilepsy

Fear of any kind is a product of physical activity in the brain, and for its motor expression or subjective experience many parts of the brain appear to play some role. But is fear, the feeling of fear, a function of the whole or part?

Speech, in a manner similar to fear may, in its elaboration, be contributed to by cerebral activity in diverse areas; either its content, reception association or expression, yet subserving

it is a neuronal mechanism which has a well recogmized anatomical situation (Symonds) (76).

Is it possible that the feeling state fear is a product of activity in a similar mechanism, which subserves all cerebral activity elaborating fear as the appropriate feeling state for the situation, whether internal or external? Speech is not localized by the patient to any particular part; that the feeling state fear is a general state of feeling and not localized is no reason to assume that it is not elaborated by a localized mechanism.

The evidence, adduced from consideration of
the cases in this series and the cases in the literature, supports the view that at least one form of
fear (the aura fear) is the product of localized
cerebral activity. The isolated nature of the fear
is possibly important, suggesting that the mechanism
whose activity produces it has no recognizable
function other than the production of the pure feeling;
and suggesting that fears with content are also its
product, though the content is a function of other
structures in neuronal connection, either directly

or indirectly, with this mechanism. Moreover, some of the irrational fears without basis may be a product of its activity.

The evidence is that all parts of the brain are continuously active and may be self maintaining or even self initiating (Adrian (1), Lorente De No (50) (51). Thus either excitation or alteration of the mechanism from a previous level of activity may result in the feeling state, fear.

Ablation of part of this mechanism (anterior cingulectomy) does appear from the literature to affect fear states. Fulton (24), in his review, concluded provisionally that this operation was one of choice for anxiety states (and for obsessional psychotics), and that no intellectual impairment resulted from the procedure.

If there is a localized neuronal circuit which elaborates the emotion fear, as a response to certain patterns of stimuli which reach it via a wide intemprative system, it can be readily understood how epileptic activity in this site will result in the experience of fear without content, in the same manner

as epileptic activity in the post-central area is mis perceived as a sensation in, say, the right hand. If the activity is confined to this mechanism alone, fear alone will be perceived. If the activity spreads, the area to which it spreads will be revealed by the character of the resulting aurae. Confusion or loss of consciousness is an index of even greater activity and greater spread.

Thus, at least one form of subjective fear occurs during activity in a particular mechanism; a mechanism which is situated in an architecturally simple and phylogenetically old protion of the brain. In animals, we recognize emotional expression and there is ample evidence that this is mediated by the hypothalamus. In man alone can we understand, even though poorly, the feeling of fear. Is the production of a feeling state by activity in this old portion of the brain some indication that feeling states occurred in early stages of development, and still occur in lowly forms of life? The amoeba behaves in direct accord with the sensorial situation, but scarcely experiences emotion. The hasty flight of

the mouse from the cat on the very first occasion it sees it, or never (Brain (7)), is probably not the result of thought, but behavior in accord with a genetically endowed pattern. That it may feel fear is possible.

With regard to the differentiation between fear in epilepsy and psychiatric fears, there is little difficulty. The fear in epilepsy is usually followed by a seizure, is commonly accompanied by another aura, is always paroxysmal and occurs, as most epileptic discharges do, abruptly, and out of context. Clinically, the distinction is important, if only because fear in epilepsy is more amenable to therapy, and at times is a symptom of a more serious underlying disorder.

state: a peculiar familiarity; the feelings of reliving; an alteration of relationship of person to
environment - all of absorbing interest to the
patient at the time. It is a strange objectivity
in which consciousness, or at least remaining consciousness is concerned with a subjective unaccountable
state, yet contact with environment may be maintained

and behaviour may be appropriate. When behaviour is inappropriate, as in automatism, presumably during more complex and widespread activity (Jackson) (144), the patient is behaving in accord with the mental situation. Whether this indicates a widespread or bilateral activity or merely intensity of activity in one situation, is a matter for speculation. If the former, it signifies a physical state blocking consciousness. If the latter, it may signify intense activity focussing attention - that is, inattention for other processes and sensory stimuli.

The explanation of this ability of some patients to experience abnormal psychical states and, at the same time, still recognize that which is abnormal from the normal, is according to Jackson, that the abnormal is the product of one temporal lobe while the other unaffected temporal lobe is still able to function normally. This state he referred to as "mental diplopia".

The flight or desire for flight, shown by patients during the experience of epileptic fear, while appropriate with regard to the internal situation

is inappropriate with regard to the total situation.

Appropriate behaviour was usual and anxiety concerning the implications of the fear state was commonly expressed. That is, ability to abstract from the total situation was commonly retained; possibly a reflection of the bilateral situation of the cortical representation of the fear mechanism — a further example of Jackson's mental diplopia.

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# SUMMARY AND CONCLUSIONS

Fear, without content and unrelated to thought processes or somatic sensations has been observed in 40 cases of epilepsy.

The paroxysmal nature of the fear and the relationship to the seizures have led to the conclusion that this fear is an aura. Other cases with fear as an aura have been cited from the literature.

Clinical, electroencephalographic and pathological studies strongly suggest that this aura is a product of epileptic activity on either side; and that the localization of the focal activity producing the fear is situated in the medial aspect of the temporal lobe.

Pertinent literature concerning the emotions, their anatomy and their nature, has been discussed.

The aura when not commonly followed by a seizure may not be recognized as epileptic activity, and yet as epileptic activity it is not only amenable to treatment, but also may be symptomatic of a

serious underlying pathology, neoplastic or vascular.

Observations on the nature of fear in epilepsy have been made.

The importance of consideration of the aura, fear, rests, not only in that the aura may not be recognized as epileptic activity, but also in that it affords an approach, along physical lines, to the study of at least one form of emotional activity in man. The emotions play such an important role in the realm of human behaviour and endeavour, yet knowldge concerning their physical nature is still very limited.

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#### BIBLIOGRAPHY

- 1. Adrian, E. D.: Electrical activity of the nervous system, Arch. Neurol. & Psychiat. 32:1125-1136, 1934.
- 2. Bailey, P. and Gibbs, F. A.: The surgical treatment of psychomotor epilepsy, J.A.M.A. 145:365-370, 1951.
- 3. Bard, P.: A diencephalic mechanism for expression of rage with special reference to sympathetic nervous system, Am. J. Physiol. 84:490-515, 1928.
- 4. Bard, P.: Central nervous mechanisms for emotional behavior patterns in animals, Research Publ., A. Nerv. & Ment. Dis. 19:190-218, 1939.
- 5. Bard, P. and Rioch, D. M.: Study of four cats deprived of neocortex and additional portions of the forebrain, Bull. John Hopkins Hosp. 60:73-147, 1937.
- 6. Bard, P. and Mountcastle, V. B.: Some forebrain mechanisms involved in expression of rage with special reference to suppression of angry behavior, Research Publ., A. Nerv. & Ment. Dis. Proc. 27:362-404, 1948.
- 7. Brain, W. R.: Mind, Perception and Science, Springfield, Illinois, C. C. Thomas, 1951.
- 8. Bright, R.: Reports of Medical Cases, London, Vol. 2, pt. 2, 1831.
- 9. Brodal, A.: The hippocampus and the sense of smell. A review, Brain 70:179-222, 1947.
- 10. Cannon, W. B.: The James-Lange Theory of Emotion, a critical examination and an alternative theory, Am. J. Psychol. 39:10-124, 1927.
- 11. Cannon, W. B.: The James-Lange Theories of Emotion, Psychol. Rev. 38:281-295, 1931.

- 12. Clark, W. E. Le Gros: The connections of the frontal lobes of the brain, Lancet 254:353-356, 1948.
- 13. Clark, W. E. Le Gros and Meyer, M.: Anatomical relationships between the cerebral cortex and the hypothalamus, Brit. Med. Bull. 6:341-345, 1950.
- 14. Cobb, S.: Emotions and Clinical Medicine, New York, W. W. Norton & Co. Inc., 1950.
- 15. Critchley, Macdonald: The anterior cerebral artery and its syndromes, Brain 53:120-165, 1930.
- 16. Culbreth, G. C.: The electroencephalogram in brain tumour suspects, EEG Clin. Neurophysiol. 2:115, 1950.
- 17. Dana, C. L.: The anatomic seat of the emotions: a discussion of the James-Lange Theory, Arch. Neurol. & Psychiat. 6:634-639, 1921.
- 18a. Dax, E. C. and Radley-Smith, E. J.: Discussion: Prefrontal leucotomy with reference to indications and results, Proc. Roy. Soc. Med. 39:448-449, 1945-46.
- 18b. Dax, E. C., Reitman, F., and Radley-Smith, E. J.: Prefrontal leucotomy. Summary of three papers presented at the International Congress of Psychosurgery, Lisbon, Digest Neurol. & Psychiat. 16-533, 1918.
- 19. Dostoievsky, Fyodor: The character Kirillov in The Possessed.
- 20. Earle, K. M., Baldwin, M., and Penfield, W.:
  Incisural sclerosis and temporal lobe seizures
  produced by hippocampal herniation at birth, Arch.
  Neurol. & Psychiat. 69:27-62, 1953.

- 21. Ferrier, D.: The Croonian Lecture. Experiments on the brain of monkeys, Philos. Trans. 165:433-488, 1875.
- 22. Freeman, W. and Watts, J. W.: Psychosurgery, intelligence, emotion and social behavior following prefrontal lobotomy for mental disorders, 2d ed. Springfield, Illinois, Charles C. Thomas, 1951.
- 23. Fritsch, G. and Hitzig, E.: Ueber die elektrische Erregbarkeit des Grosshirns, Arch. f. Anat., Physiol. u. wissensch Med. 37:300-332, 1870.
- 24. Fulton, J.: Frontal Lobotomy and Affective Behavior, New York, W. W. Norton & Co. Inc., 1951.
- 25. Gibbs, E. L., Gibbs, F. A. and Fuster, B.: Psychomotor epilepsy, Arch. Neurol. & Psychiat. 60:331-339, 1948.
- 26. Gibbs, F. A., Gibbs, E. L., and Lennox, W. G.: Epilepsy: a paroxysmal cerebral dysrhythmia, Brain 60:377-388, 1937.
- 27. Gibbs, E. L., Fuster, B., and Gibbs, F. A.:
  Peculiar low temporal localization of sleep induced
  seizure discharges of psychomotor type, Arch.
  Neurol. & Psychiat. 60:95-97, 1948.
- 28. Gibbs, F. A., Wegner, W. R., and Gibbs, E. L.:
  The electroencephalogram in post-traumatic epilepsy,
  Am. J. Psychiat. 100:738-749, 1944.
- 29. Goltz, F.: Ueber die Functionen des Lendenmarks des Hundes Pflüg, Arch. ges Physiol. 8:460-498, 1874.
- 30. Gowers, W. R.: Epilepsy and other Chronic Convulsive Diseases, London, J. A. Churchill, Ed. 2, 1901.
- 31. Green, J. R., Duisberg, R. E. H., and McGrath, W. B.: Focal epilepsy of psychomotor type, J. Neurosurg. 8:157-172, 1951.

- 32. Green, J. R., Duisberg, R. E. H., and McGrath, W. B.: Electrocorticography in psychomotor epilepsy, EEG Clin. Neurophysiol. 3:293-299, 1951.
- 33. Greenblatt, M.: The E.E.G. in late post-traumatic cases, Am. J. Psychiat. 100:378-386, 1943.
- 34. Hartley, P.: Physiological mechanisms in animal behaviour, Cambridge, 1950. Cited by Brain, W. R. in Mind, Perception and Science, Springfield, Illinois, C. C. Thomas, 1951.
- 35. Head, H. and Holmes, G.: Studies in Neurology, Oxford Med. Publ., 1920, vol. 2, p. 605.
- 36. Hebb, D. O.: On the nature of fear, Psychol. Rev. 53:259-276, 1946.
- 37. Henderson, J. A.: Significance of fear, Edinb. Med. J. 48:649-661, 1941.
- 38. Herrick, C. J.: Morphogenesis of the brain, J. Morphol. 54:233-258, 1933.
- 39. Hess, W. R.: Das Zwischenhirn Syndrome, Lakalisationen, Funktionen, Basel: Benno Schwabe, 1949.
- 40. Hill, D.: A Summary IVe Congres International de Neurologie, 1:27-33, 1949.
- 41. Ingram, W. R.: Nuclear organization and chief connections of the primate hypothalamus, Research Publ., A. Nerv. & Ment. Dis. 20:195-244, 1940.
- 42. Jackson, J. H.: On right or left-sided spasm at the onset of epileptic paroxysms, and on crude sensation warnings, and elaborate mental states, Brain 3:192-206, 1880-81.
- 43. Jackson, J. H. and Stewart, P.: Epileptic attacks with a warning of a crude sensation of smell and with the intellectual aura (dreamy state) in a patient who had symptoms pointing to

- gross organic disease of the right temporosphenoidal lobe, Brain 22:534-549, 1899.
- 44. Jackson, J. H.: In selected writings of John Hughlings Jackson, Ed. James Taylor, London, Hodder and Stoughton, 1931.
- 45. Jasper, H. and Kershman, S.: Electroencephalographic classification of the epilepsies,
  Arch. Neurol. & Psychiat. 45:903-943, 1941.
- 46. Jasper, H.: Electroencephalography in Penfield, W. and Erickson, T. C., Epilepsy and Cerebral Localization, Springfield, Illinois, C. C. Thomas, 1941.
- 47. Jasper, H., Pertuissit, B., and Flannigin, H.: E.E.G. in temporal lobe seizures, Arch. Neurol. & Psychiat. 65:272-290, 1951.
- 48. Karpus, J. P. and Kreidl, A.:
  Gehirn und Sympathicus. 1. Zwischenhirn
  Pflig Arch. ges Physiol, 129:138-144, 1910.
- 49. Lashley, K. S.: The thalamus and emotion, Psychol. Rev. 45:42-61, 1938.
- 50. Lorente De No' R.: Transmission of impulses through cranial motor nuclei, J. Neurophysiol. 2:402-404, 1939.
- 51. Lorente De No<sup>\*</sup> R.: Cerebral Cortex: architecture, in Physiology of Nervous System by Fulton, J. F., New York, Oxford Univ. Press, 1943, 274-301.
- 52. MacLean, P. D.: Discussion, Am. J. Psychiat. 108:431-432, 1951.
- 53. MacLean, P. D.: Psychosomatic disease and the visceral brain, Psychosom. Med. 11:338-353, 1949.
- 54. Magnus, O., Penfield, W., and Jasper, H.:
  Mastication and consciousness, Acta. Psychiat. et

- Neurol. Scandinavica 27; fasc. 1-2, 91-115, 1952.
- 55. Masserman, J. H.: Is the hypothalamus a center of emotion? Psychosom. Med. 3:3-25, 1941.
- 56. Meyer, A. and McLardy, T.: Clinico-anatomical studies of frontal lobe function based on leucotomy material, J. Ment. Sc. 95:403-417, 1949.
- 57. Minor, L. S.: Localised sense of fear in epileptics, Sovrem Psikhoneurol 9:813-825, 1929.
- 58. Moniz, E.: Tentatives operatoines dans le traitement de certaines psychoses, Paris, Masson et Cie, 1936.
- 59. Pampiglione, G.: Aneurismi intracranici multipli, Riv. di. Neurologia, 19:1-16, 1949.
- 60. Papez, J. W.: A proposed mechanism of emotion, Arch. Neurol. & Psychiat. 38:725-743, 1937.
- 61. Penfield, W. Influence of the diencephalon and hypophysis upon general autonomic function, Bull. New York Acad. Med. 9:613-637, 1933.
- 62. Penfield, W. and Erickson, T. C.: Epilepsy and Cerebral Localization, Springfield, Illinois, C. C. Thomas, 1941.
- 63. Penfield, W. and Kristiansen, K.: Epileptic Seizure Patterns, Springfield, Illinois, C. Thomas, 1957.
- 64. Penfield, W. and Rasmussen, T.: The Cerebral Cortex of Man, New York, Macmillan, 1950.
- 65. Penfield, W. and Flannigin, H.: Surgical therapy of temporal lobe seizures, Arch. Neurol. & Psychiat. 64:491-500, 1950.

- 66. Pribram, K. M. and MacLean, P.D.: A comparative neuronographic study of the connections between the phylogenetically old and new cortex, cited by Fulton, J. F. in Frontal Lobotomy and Affective Behaviour, New York, W. W. Norton & Co. Inc., 1951.
- 67. Raw, N.: Fear and worry, J. Ment. Sc. 75:573-583, 1929.
- 68. Ransom, S. W.: The Hypothalamus: Its Significance for Visceral Innervation and Emotional Expression, Tr. Coll. Physicians, Philadelphia, 2:222-242, 1932.
- 69. Rioch, D. M. and Brenner, C.: Experiments on corpus striatum and Rhinencephalon, J. Comp. Neurol. 68:491-507, 1938.
- 70. Ryle, J. A.: Nosophobia, J. Ment. Sc. 94:1-17, 1948.
- 71. Ryle, J. A.: Angor Animi or sense of dying, Guy's Hospital Reports 99:230-235, 1950.
- 72. Schwab, R. S.: Electroencephalography, Philadelphia, W. B. Saunders, 1951.
- 73. Sherrington, Sir Charles: The Integrative Action of the Nervous System, 2n. ed., Cambridge University Press, 1951, p. 564.
- 74. Spiegel, E. A., Miller, H. R., and Oppenheimer, M. J.: Forebrain and rage reactions, J. Neuro-physiol. 3:538, 1940.
- 75. Spiegel, E. A., Wycis, H. T., Freed, H., and Orchinik, C.: The central mechanism of the emotions, Am. J. Psychiat. 108:426-432, 1951.
- 76. Symonds, Sir Charles P.: Aphasia, J. Neurol. Neurosurg. Psychiat. 16:1-6, 1953.
- 77. Temkin, O.: A history of epilepsy from the Greeks to the beginnings of modern neurology, John Hopkins Press, 1945

- 78. Van der Horst, L.: Affective epilepsy, J. Neurol. Neurosurg. Psychiat. 16:25-29, 1953.
- 79. Walter, W. Grey: The electroencephalogram in cases of cerebral tumour, Proc. Roy. Soc. Med. 30:579-598, 1937.
- 80. Walter, W. Grey: The functions of electrical rhythms of the brain, J. Ment. Sc. 96:1-31, 1950.
- 81. Weiner, N.: Cybernetics, New York, Wiley, 1948.

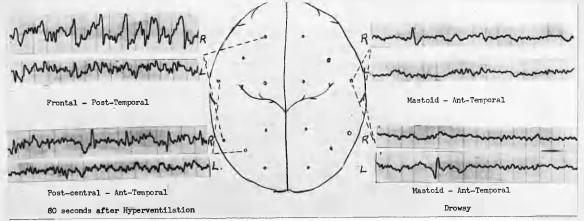


Fig. 1 - EEG in case Fh: focal changes in right frontotemporal area during and after hyperventilation; independant spikes in both anterior-temporal areas during drowsiness.

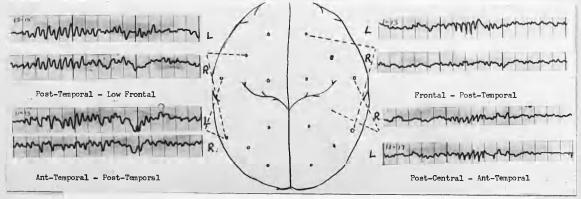


Fig. 2 - EEG in case F3: well marked lh and 6 per second positive spikes in the left posterior-temporal ar

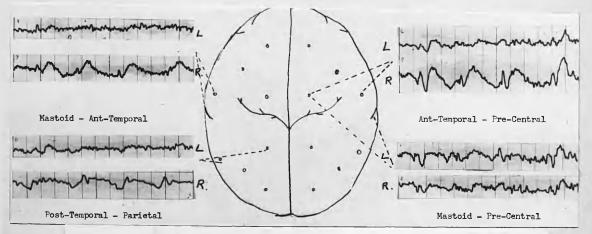


Fig. 3 - EEG in case Fl: Right anterior temporal delta wave focus, with occasional spikes; the focus appeared during a drowsy phase.

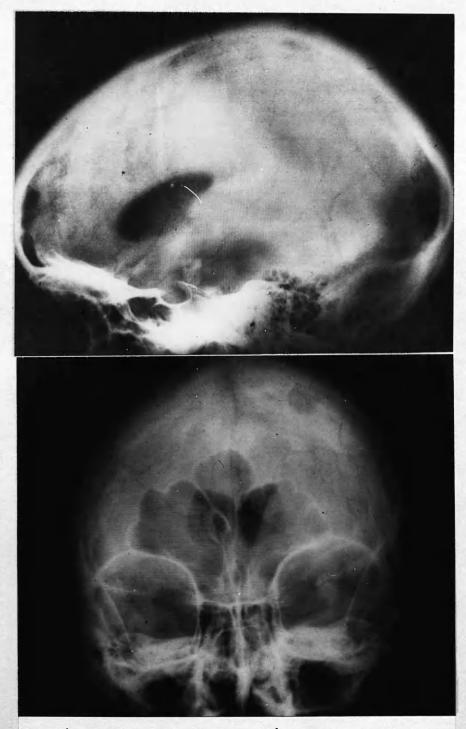


Fig. 4 - Ventriculogram, case F6. Calcified astrocytoma in the right temporal lobe.

The tip of the right temporal horn is displaced backwards, slightly.



Fig. 5 - Coronal section of brain, case Nl, at site of aneurysm. Aneurysm at junction of anterior cerebral and anterior communicating arteries.

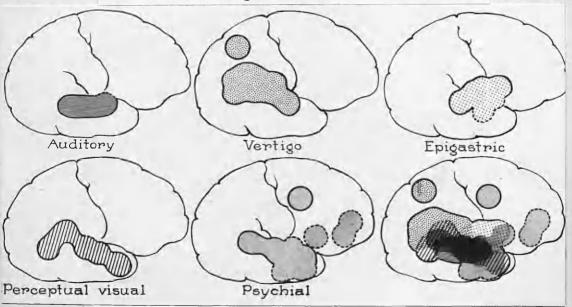


Fig. 6 - Localization of epileptogenic lesions in his patients with the aurae shown (after Penfield 1951, modified)



Fig. 7 - Aneurysm of the right internal carotid (after Pampiglione 1949, case 2).

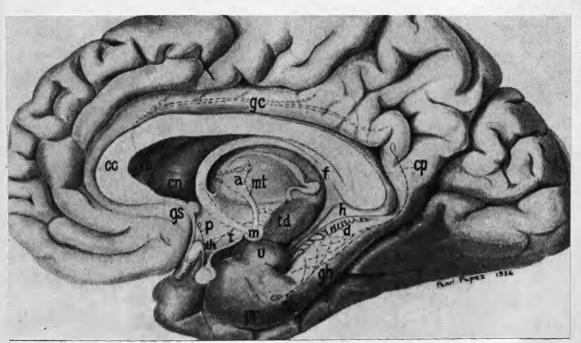


Fig. 8 - Medial view of right cerebral hemisphere (after Papez, 1937) showing the hippocampus, hypothalamus, cingulate gyrus, anterior thalamic nuclei and their connections.

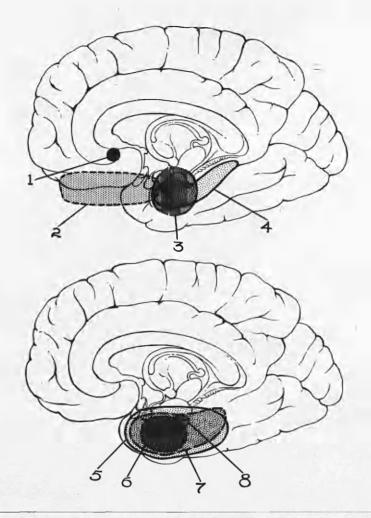


Fig. 2 - Situation of the lesions in 8 cases (Penfield's case and cases F8 and F17 are not shown).

1 - aneurysm in case N1, Fig. 5

2 - meningioma in case F5

3 - aneurysm, Fig. 7

4 - area of sclerosis, Earle's case, pg. 40

5 - astrocytoma in case F206 - astrocytoma in case NE2

7 - glioma in Mayo clinic case, pg. 21

8 - calcified astrocytoma in case F6, Fig. 4

### APPENDIX

Cases	· · · · · · · · · · · · · · · · · · ·	Page
F1 - F25		1
G1 - G10		40
NI - N5		52
NEL - NE2		59

F - Focal EEG change
G - Generalized EEG change
N - Normal EEG

NE - No EEG

#### APPENDIX

CASE: (F1) Age: 25 years Sex: Female

HISTORY: She complained of attacks of loss of
consciousness with generalized convulsions, since
aged 3 years. An attack is preceded by "a sudden
change in life, everything looks strange and
different; it is awful. I feel full of fear
and my heart beats fast. I am so frightened that
I wish I would lose consciousness." The fear
lasts a few seconds. Loss of consciousness was
seldom complete. She denies being afraid because
of the imminence of a fit, rather welcoming the
change in consciousness which brings the experience
to an end. She is right-handed.

FAMILY AND PREVIOUS MEDICAL HISTORY: Nothing significant.

EXAMINATION: Mentally rather retarded. No neurological abnormality discovered.

# INVESTIGATIONS:

Electroencephalogram: Showed frequent isolated spikes in the left-anterior temporal area, several independent spikes in the right-anterior temporal area and occasionally in the low frontal areas on both sides, during drowsiness and light sleep. During

hyperventilation high potential spikes and  $2\frac{1}{3}$  per sec. waves occurred in the right low frontal anterior temporal area.

X-ray Skull: - Normal

Air Encephalogram: Normal

<u>Wasserman Reaction</u>: Negative in blood and C.S.F.

<u>TREATMENT</u>: Dilantin, mesantoin, phenobarbitone
and phenurone were all given in various combinations. The best improvement was with Mesantoin
combined with phenobarbital.

DIAGNOSIS: Temporal lobe epilepsy, cause unknown.

CASE: (F2) Age: 33 years Sex: Female

HISTORY: She complained of attacks since aged
8 years. They begin with "an odd undescribable
sensation, which becomes an intense feeling of
fear." She feels, "I am going to die," and runs
when possible to her husband. An urge to urinate
occurs, usually preceding the fear, so that now
she is afraid to appear in public places lest
"I take my pants down right there." Occasionally

during these attacks she has the hallucinatory experience that she is in the family's old buck-board riding "lickety-split" for the wood shed at their old home. This reminisence is very vivid. Consciousness is infrequently lost though there is clouding.

She is right-handed.

PREVIOUS MEDICAL AND FAMILY HISTORY: Bevealed no significant facts.

EXAMINATION: No neurological or other abnormality was noted.

### INVESTIGATIONS:

X-ray Skull: Normal

Air Encephalogram: Showed dilatation of cerebral sulci suggestive of mild cortical atrophy.

Electroencephalogram: Showed synchronous 4-5/sec. activity in both fronto-temporal areas with numerous medium voltage spikes in the low-frontal and anterior-temporal areas on the right.

Cerebro-Spinal Fluid: Normal constituents.

Wasserman Reaction: Negative in blood and C.S.F.

DRUGS: Phenurone, mesantoin and dilantin were used but with little improvement.

<u>DIAGNOSIS</u>: Temporal lobe epilepsy, cause unknown.

CASE (F3) Age: 17 years Sex: Male

HISTORY: He complained of attacks which began
shortly after a head injury, when aged 3 years.

The attacks begin with vertigo—a sensation that
the head is whirling clockwise—and an intense
feeling of fear. He denies that he is afraid
because the head is whirling and states that the
fear rises "out of the blue." The attacks last
a few seconds and are infrequently followed by
loss of consciousness. Apart from the attacks he
may have vivid 'deja vu' experiences.

He is right—handed.

PREVIOUS MEDICAL HISTORY: When aged 8 years he was injured in a motor car accident and was unconscious for several hours. The post-traumatic amnesia is of several hours duration, the retrograde amnesia being brief but well-defined, probably a few seconds.

FAMILY HISTORY: Revealed nothing significant.

EXAMINATION: Revealed average intelligence and no neurological abnormality, in particular no evidence of vestibular disturbance.

### INVESTIGATIONS:

X-ray Skull: Normal

Wasserman Reaction: Negative in blood.

Electroencephalogram: At several points in the recording well marked 14 and 6 per second positive spikes were seen predominantly in the left posterior temporal area.

<u>DRUGS</u>: Dilantin 0.1 grams b.i.d. and mebaral 0.1 grams b.i.d. were given with moderate improvement in the frequency of the attacks, but the patient ceased to attend after two months and the effect of higher doses of drugs was not observed.

<u>DIAGNOSIS</u>: Temporal lobe epilepsy, post-traumatic.

CASE (F4) Age: 23 years Sex: Male - a

medical student

HISTORY: He complained of attacks of loss of consciousness of 1½ years duration. "Several things happen suddenly and about the same time. I have an unpleasant sensation in my epigastrium. I feel an intense fear, worse and different to any fear ever; but yet the fear is peculiarly familiar as though previously experienced, even

though at the time I know it has not been. Voices or the radio may sound different and I have noises in my ears." The fear is followed by a cold sweat, and at time pallor, and "I may have an urge to defaecate." There is no desire to flight and he sits still. In one attack loss of consciousness was preceded by an involuntary movement of eyes and head to the right. On several occasions his situation and events "took on a different meaning" which at the time appeared very real and of significance, though he was later unable to recall what the significance was. These aurae usually last 15-30 seconds and loss of consciousness usually, though not always, follows, lasting a few minutes. He is right handed.

PREVIOUS MEDICAL AND FAMILY HISTORY: Revealed nothing significant.

# INVESTIGATIONS:

X-ray Skull: Normal

Air Encephalogram: - Normal

Bilateral carotid arteriograms: Normal

Electroencephalogram: - In the drowsy phase delta

waves with occasional spikes appeared in the right anterior temporal area.

<u>Cerebro-Spinal Fluid</u>: Normal constituents

<u>Wasserman Reaction</u>: Negative in blood and C.S.F.

DRUGS: Phenobarbital 0.15 grams q.i.d. reduced the attacks from several to one per week, and the remaining ones were usually aurae seldom followed by loss of consciousness.

<u>DIAGNOSIS</u>: Temporal lobe epilepsy, cause unknown.

CASE (F5) Age: 31 years Sex: Male

HISTORY: He complained that for  $2\frac{1}{2}$  years he

was liable to attacks of loss of consciousness

preceded by flashes of light in the lower outer

corner of right eye, often followed by an in
describably terrible fear, "out of this world" 
"I wish I could describe it." "I may feel as

though transferred to another world or as though

a million years ago one had lived." "An un
pleasant epigastric sensation follows and the

heart pounds." After about 30 seconds loss of

consciousness occurs followed by a generalized

convulsion. The fear, he states, is not anxiety lest he have an attack, but a fear which comes by itself. He used to experience anxiety at the onset and at the same time as the other fear, but "this fear or terror is not like anything else." In a few attacks he had, during the aura, a difficulty with speech. He knew what he wanted to say but could not say it; however, he could understand conversation. The condition had, in the beginning, been diagnosed as 'psychiatric.'

He is right-handed.

PAST MEDICAL HISTORY: He was injured in an automobile accident when aged 4-5 years, and his skull was fractured.

FAMILY HISTORY: Revealed nothing significant.

EXAMINATION: Showed early bilateral papilloedema and no physical signs.

### **INVESTIGATIONS:**

<u>Ventriculography</u>: Showed a downward and right lateral displacement of the ventricular system on the left.

Electroencephalogram - Showed a high potential 4-7 per second activity focal to the left anterior temporal and low frontal on the left. At operation, a mengioma was removed by Dr. Edwin Boldrey, University of California Hospital, from the left upper parieto-occipital In the three months following operation no further attacks have occurred. DIAGNOSIS: Left parieto-occipital meningioma

with uncinate epilepsy.

CASE (F6) Age: 41 years Sex: Female HISTORY: Since aged 10 years she was liable to attacks: a peculiar smell, not quite like a hard-boiled egg, lasting a second, followed by a frightened feeling with a vague hallucination as though someone was "chasing me upstairs in the dark," lasting a very few seconds. The fear always appeared to come from behind and was not related to any past experience. These were very infrequently followed by loss of consciousness till aged 24 years. Since then loss of consciousness has been more frequent. She is right-handed.

PREVIOUS MEDICAL AND FAMILY HISTORY: Nothing significant was revealed.

EXAMINATION: No abnormality was noted on neurological and general examination.

### **INVESTIGATIONS:**

X-ray Skull: Showed a small area of calcification in the right temporal lobe.

<u>Ventriculogram</u>: Showed the tip of the right temporal horn displaced backwards very slightly.

Electroencephalogram: Showed a low voltage irregular 5-7/sec. wave focus in the right temporal area.

OPERATION: Note by Mr. Wylie McKissock, The National Hospital. The mid-temporal convolution was incised and opened to the temporal horn. Firm resistance was met 3 cms. deep and extended back and forward. The firm tissue was excised.

PATHOLOGICAL EXAMINATION: A mass of laminated calcified concretions with adjoining gliomatous tissue, which on histological examination, had the appearance of astrocytoma, grade 1.

DIAGNOSIS: Right temporal lobe astrocytoma.

CASE (F7) Age: 70 years Sex: Female HISTORY: She complained that since aged 40 years she was liable to attacks in which a "substance not a living thing" appears from over the right shoulder; the vision to the right becomes obscured: the right hand appears large, twice its normal size; a funny feeling develops under the chest, and there is a feeling of unaccountable fear. The sensations used to frighten her but do so no longer. The fear is strange and "different to any fear ever." Occasionally she may have a peculiar 'sharp' taste and unusual 'indescribable' smell. At times she may hear a voice, her own, talking to her. Consciousness is usually lost. Three separate attacks were observed in the hospital. In each she shouted, "come, oh, come," and "oh, dear, this is dreadful," but would not respond to questioning. On each occasion she was described by a nurse who observed the attacks as "appearing frightened." The attacks lasted 1-2 minutes and no movements were observed.

She is right-handed.

PREVIOUS MEDICAL HISTORY: Immediately after her last pregnancy, when aged 40 years, she was unconscious for a day, and afterwards confused and unmanageable. She was in bed for 6 weeks and diagnosed as having cerebral thrombophelebitis.

FAMILY HISTORY: One daughter suffered from schizophrenia.

<u>EXAMINATION</u>: Revealed no neurological abnormality.

<u>INVESTIGATIONS</u>:

X-ray Skull - Normal

X-ray Chest - Normal

Cerebro-spinal Fluid - normal constituents.

<u>Electroencephalogram</u> - On over-breathing low voltage 5-7 per second and occasional sharp waves appeared in the left-frontal area.

DRUGS: Phenobarbital, grain 12, t.d.s. Epanutin,

grain  $1\frac{1}{2}$ , t.d.s. with moderate control.

<u>DIAGNOSIS</u>: Left temporal lobe epilepsy, secondary to cortical thrombo-phlebitis.

CASE (F8) Age: 44 years Sex: Female

HISTORY: For one year liable to frequent

attacks: "I am suddenly seized by a horrible feeling of terror" - terrified of something unknown. "I cannot describe it. After about one minute the terror passes off and is replaced by a horrible smell, not a real smell. It is somewhat like the smell of burning hedges. After a few seconds I get a horrible feeling as though being choked by the smell and suddenly sink to the ground. Consciousness is rarely lost and after 1-10 minutes she is able to resume as though nothing had happened. When consciousness is not lost she can understand conversation but is not able to reply. For several years she frequently had an overpowering feeling of 'familiarity' as though reliving the past.

She is right-handed.

PAST MEDICAL HISTORY AND FAMILY HISTORY: Revealed nothing significant.

EXAMINATION: She showed marked euphoria. She was unable to distinguish smells on the right.

The right eye was blind and the right optic

disc showed the pallor of atrophy.

### INVESTIGATIONS:

Right carotid arteriogram - Showed a spherical 'blush' above the floor of the right anterior fossa from 1 cm. posterior to the gabella to the coronal plane.

Electroencephalogram - Bursts of 4 per second and almost continuous 1-2 per second waves appeared in the right low-frontal anterior-temporal area, appearing to centre in the temporal tip.

OPERATION: Note by Mr. McKissock, The National Hospital. A meningioma weighing 59 grams, which occupied the whole of the right anterior fossa except the anterior two cms, was removed.

COURSE: Six months post-operative she was free from fits and the fear.

CASE (F9) Age: 17 years Sex: Female

HISTORY: Since aged 3 years she was subject

to generalized convulsions, preceded by an

aura. In recent years she recognized this aura

as an indescribably funny feeling which affected her stomach. At times, but not always, she also felt very frightened.

Left-handed in a right-handed family.

<u>PAST MEDICAL HISTORY</u>: When aged 1-2 years it was observed that she could not use her right arm or leg as well as the left, and this has persisted.

<u>FAMILY HISTORY</u>: Revealed nothing significant.

<u>EXAMINATION</u>: Showed mental retardation; mild weakness of right side of face, arm, and leg, with exaggerated reflexes and extensor plantar response on the right.

# **INVESTIGATIONS:**

<u>Air Encephalogram</u> - Showed dilatation of left lateral ventricle with pooling of air over the left cortex, compatible with a left cortical atrophy.

Electroencephalogram - Slow 5-7 per second medium voltage waves appeared to centre in the left low fronto-anterior temporal area.

DRUGS: Bromide had been given in early years.

The addition of Phenobarbitone, grain 3/4, t.d.s., and Dilantin, grain  $1\frac{1}{2}$ , t.d.s., decreased the frequency of the attacks slightly. DIAGNOSIS: Epilepsy secondary to infantile hemiplegia.

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CASE (F10) Age: 37 years Sex: Female HISTORY: For 10 years she had infrequent attacks of loss of consciousness of a few seconds duration preceded, usually, by a feeling that something was going to happen - "an uncanny feeling difficult to describe," and a feeling of odd fear of brief duration. Her husband described a "very startled look." prior to some attacks. The loss of consciousness was of 2-3 seconds duration. She might then talk nonsense or behave in an apparently automatic inappropriate fashion. On one occasion she filled the bath with water and stepped into it fully clothed. In a few attacks involuntary jerking movements of the right arm were observed. She is right-handed.

FAMILY HISTORY AND PAST MEDICAL HISTORY: Revealed no significant facts.

EXAMINATION: Showed no neurological abnormality.
INVESTIGATIONS:

Cerebro-spinal fluid: Normal constituents.

Electroencephalogram: Showed a 5-7 per second activity, focal to the left low frontal-anterior-temporal area.

Air Encephalogram: Showed a slight dilatation and distortion of the tip of the left temporal horn.

<u>DIAGNOSIS:</u> Psychomotor epilepsy? Left temporal lobe focus, cause unknown.

<u>DRUGS</u>: Phenobarbitone and Epanutin
were used with slight decrease in the frequency
of attacks.

CASE (F11) Age: 43 years Sex: Male

HISTORY: Frequent fits with generalized convulsions occurred during the past 12 years.

These were almost always preceded by some aura:
either a horrible feeling that something cold
was happening to his left ear, "not quite a touch
and yet not auditory;" or a feeling of fear,

"an indescribable horrible fear, with a feeling that something was going to happen; like a night-mare, a 'terror'." These feelings were familiar; as though they had a meaning, yet the significance eluded him. He feels that he wants to hide himself. During the fear there is a peculiar feeling in the epigastrium. The fear may occur "out of the blue, even when I am making a joke." Nightmarish dreams occur frequently.

He is right-handed.

PAST MEDICAL HISTORY: He suffered a severe head injury in 1931, resulting in a fractured skull and a post-traumatic amnesia of one month's duration.

FAMILY HISTORY: Nothing significant.

EXAMINATION: Showed an intelligent but rather obsessive man rather disturbed by his attacks.

No neurological abnormality was noted.

X-ray skull - Normal

Electroencephalogram: Showed a 5-7 per second medium voltage waves focal to the left temporal area.

Air Encephalogram: No definite abnormality.

<u>DIAGNOSIS:</u> Post-traumatic epilepsy - temporal lobe focus.

<u>DRUGS</u>: Phenobarbitone gr. 1,.t.i.d. and Epanutin gr.  $1\frac{1}{2}$ , q.i.d., controlled the attacks except for a very occasional aura.

CASE (F12) Age: 17 years Sex: Female HISTORY: She complained of attacks of intense fear, since aged 12 years, preceded by a sudden flashing momentary pain in the head. The fear is intense and accompanied by a strong desire to run away without knowing where to - a desire for flight. This may last up to 2 minutes and then she is unable to utter a word. Though she knows what she wants to say, she cannot find words. Towards the end of this period she may behave in an automatic fashion doing unnecessary things "in a daze." In childhood these attacks were followed by loss of consciousness on several occasions. The frequency, over the few months prior to being seen, varied from 2 per day to l per week.

She is right-handed.

PAST MEDICAL HISTORY: Whooping cough at 12 years.

The first attack occurred at this time.

FAMILY HISTORY: Revealed nothing significant.

EXAMINATION: Showed no neurological abnormality.

She appeared a very intelligent garl, well adjusted to her condition, but afraid that these attacks might indicate that she was "going mad."

INVESTIGATIONS:

X-ray skull: Normal

Cerebro-spinal fluid: Normal constituents.

Air Encephalogram: Normal

Electroencephalogram: Almost continuous 4-8
per second, with occasional sharp waves, occurred
in the left-temporal area; on over-breathing a
high voltage 2½ per second appeared in the same area.

DIAGNOSIS: Epilepsy with temporal lobe focus,

<u>DIAGNOSIS</u>: Epilepsy with temporal lobe focus, probably resulting from a small vascular lesion which occurred during whooping cough.

<u>DRUGS</u>: Caps. Phenytoin Co. one t.i.d. improved the attacks; on increasing to caps one, q.i.d., the attacks stopped entirely and remained so during the subsequent nine months! observation.

CASE (F13) Age: 54 years Sex: Female

HISTORY: She complained of sudden attacks of
fear, "coming out of the blue," occurring as often
as three times per day over the past year and
unrelated to spells. During these attacks she
would feel strange as though "somewhere else."

These were all of a few seconds duration. On
three occasions the attacks resulted in complete
loss of consciousness of a few minutes duration.
Since the onset she felt worried and rather
depressed.

She is right-handed.

FAMILY HISTORY AND PAST MEDICAL HISTORY: Revealed nothing significant.

EXAMINATION: She was mildly depressed and anxious regarding the attacks. No neurological abnormality was noted. There was evidence of mild arteriosclerosis. BP 140/95.

## **INVESTIGATIONS:**

X-ray Skull - Normal

Blood Serology - Normal

ELECTROENCEPHALOGRAM: Showed medium voltage 5-7

per second activity, occurring in paroxysms and appearing to centre in the left-temporal area.

DIAGNOSIS: Epilepsy of late onset - temporal lobe focus, cause unknown, probably vascular.

DRUGS: Phenobarbitone, gr. 1, t.i.d. and Epanutin gr. 1½, t.i.d. resulted in an almost complete control of the attacks.

CASE (F14) Age: 14 years Sex: HISTORY: When aged 9 years he had several generalized convulsions with loss of consciousness lasting up to 10 minutes. He remained free from attacks till 5 years ago, since when he has brief attacks lasting a few seconds with only partial loss of consciousness. During these spells he suddenly appears frightened, becomes pale and clasps the person nearest to him. unable to do this, he hides, usually under the bed-clothes. He says that he is frightened but for no reason. He then suddenly recovers and says, "I am alright now," and then appears flushed. On several occasions he was unduly aggressive but not during his "spells."

He is left-handed.

FAMILY HISTORY AND PREVIOUS MEDICAL HISTORY: Revealed nothing significant.

<u>EXAMINATION</u>: Showed no neurological abnormality.

<u>INVESTIGATIONS</u>:

X-ray Skull: Normal

Electroencephalogram: Showed widespread medium voltage 3-7 per second activity, but with higher potentials, greater asynchromy and irregularity in the right low fronto-temporal area.

<u>DIAGNOSIS</u>: Epilepsy with temporal lobe features, cause unknown.

<u>DRUGS</u>: Mesantoin, gr.  $1\frac{1}{2}$ , b.i.d. and phenobarbital gr.  $\frac{1}{2}$ , t.i.d. resulted in only slight improvement in the frequency of the attacks.

CASE (F15) Age: 40 years Sex: Female

HISTORY: She complained of several attacks of
loss of consciousness, with no recollection of a
preceding aura, during the preceding two months.

Over the same period she also had almost daily
attacks starting with a vague epigastric sensation; "something queer going across," lasting a
few moments. They were followed by a feeling as

though "very frightened," but unlike any feeling of fear previously experienced, "very strange."
Usually during this period she can converse but at other times speech is jumbled, so that though she knows what she wants to say, a jargon ensues.
During some 'turns' she appears pale.

She is left-handed.

FAMILY HISTORY: Revealed nothing significant.

PREVIOUS MEDICAL HISTORY: A head injury at 10 years

of age resulted in a brief loss of consciousness.

EXAMINATION: Showed no neurological abnormalities.

INVESTIGATIONS:

X-ray Skull: Showed an area of sclerosed bone in the right frontal region which appeared consistent with an old head injury or infection.

Air Encephalogram: Normal

Electroencephalogram: Showed medium voltage
3 per second waves interspersed with random sharp
waves in the left low fronto-anterior temporal
area.

<u>DIAGNOSIS</u>: Epilepsy of late onset with temporal lobe focus, possibly post-traumatic.

<u>DRUGS</u>: Phenobarbitone, gr.  $\frac{1}{2}$ , t.i.d. and Epanutin, gr.  $1\frac{1}{2}$ , b.i.d. resulted in complete freedom from attacks during the succeeding six months.

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(F16) Age: 15 years CASE Sex: Male HISTORY: Since aged 5 years and intermittently since, he has been liable to sudden attacks when he appears frightened and pale, and turns to seize hold of the person or object nearest to him. He then usually, but not always, loses consciousness for up to 10 minutes. When this does not occur, he, after a few seconds, says, "I am alright." He was unable to describe his feelings except to say, "I was scared," but does not know of what, saying "of nothing." That he experienced fear without objective appeared, on close questioning, clear.

He is right-handed.

FAMILY HISTORY AND PAST MEDICAL HISTORY: Revealed no significant facts.

EXAMINATION: Showed no neurological abnormality.

INVESTIGATIONS:

X-ray Skull - Normal

Electroencephalogram: Showed medium to high

voltage 3-6 per second waves focal to the right low fronto anterior temporal area.

<u>DIAGNOSIS</u>: Epilepsy with temporal lobe focus.

<u>DRUGS</u>: Phenobarbitone, gr.  $\frac{1}{2}$ , t.i.d., Mesantoin, gr.  $1\frac{1}{2}$ , b.i.d. resulted in a markedly diminished frequency of attacks.

CASE (F17) Age: 29 years Sex: Male (doctor) HISTORY: For 11 years he was liable to brief attacks during which, for a few seconds, he would suddenly be unable to utter words, understand conversation or write though he could manipulate a pen. During this time events from the past would flash in rapid succession through his mind in an unusually clear and vivid manner but the same pattern was not invariably repeated nor could he later recall the actual events. Occasionally he would hear a musical note like "the plucking of a violin string." At times a feeling "of dread," "of unexplainable fear" would be experienced but not fear of loss of consciousness which in any case only rarely occurred. He is right-handed.

FAMILY HISTORY: Revealed no significant features.

PREVIOUS MEDICAL HISTORY: When aged 8 years he sustained a fractured skull from the left temporal bone to the vertex, and was unconscious for several hours.

EXAMINATION: Showed no neurological abnormality.
INVESTIGATIONS:

X-ray Skull: Normal

Electroencephalogram: Showed a medium voltage 5-7 per second focus in the left temporal area.

Air Encephalogram: Showed slight dilatation of the left temporal horn.

<u>DIAGNOSIS</u>: Temporal lobe epilepsy, probably post-traumatic.

DRUGS: The response to Phenobarbitone, gr. 3/4 t.i.d. and Epanutin, gr. 1½ t.i.d., during a six-weeks' period of observation, was poor.

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CASE (F18) Age: 27 years Sex: Male

HISTORY: For 5-6 years he was liable to fits

which were always preceded by some aura. The

usual aura was one of familiarity. Suddenly

whatever he looked at, even a drainpipe, would

appear unusual, as though he had looked at it

"just like this" before, intensely aware of it as though it had a new meaning for him. During this time he could neither remove his gaze from the object nor stop thinking about it. This would continue for about 30 seconds and was followed by a generalized convulsion. On several occasions the aura consisted of a gripping sensation in the epigastrium which appeared to rise to the upper chest and was accompanied by a queer feeling of fear. Usually he had no fear though he knew he was about to lose consciousness. During one observed fit, the nurse reported, "he was smacking his lips and on being questioned, stated that he could taste something sour and dirty. He then appeared very frightened, would not answer, and suddenly he had a generalized convulsion." He afterwards had no recollection of these reported events.

He is right-handed.

FAMILY HISTORY AND PAST MEDICAL HISTORY: Revealed no significant features.

EXAMINATION: Of low average intelligence and morbidly interested in his symptoms, but more so with sex and masturbation. No neurological abnormality was noted.

#### INVESTIGATIONS:

X-ray Skull: Showed a large, but not pathologically enlarged, sella turcica.

<u>Cerebro-spinal fluid</u>: Showed slight elevation of pressure but he was tense and it was not considered significant.

Electroencephalogram: Showed slow irregular 4-5 per second activity, focal to the right post-temporal area.

<u>DRUGS</u>: The response to various combinations of anti-convulsants was slight.

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CASE (F19) Age: 42 years Sex: Female

HISTORY: For 17 years she has been subject to

frequent brief attacks in which she gets a

"quivering niggling" feeling in the epigastrium

and a sensation as though a "cloud was spreading

from the back over the head," and a "something

over the right shoulder." She then has an intense

fear, "as though I was being drawn away into

another world." "I feel that it is this thing

over my shoulder which is doing this and yet it

is not of this I feel afraid." "The world looks

strange and unreal, and yet I know at the same time that I am in the real world." Loss of consciousness almost always occurs with, occasionally, a generalized convulsion. While she was anxious concerning the attacks, she denied that the "fear" was of this nature. One fit was observed. At the onset she smacked her lips, rubbed her upper lip, and moved her legs in a quasi-purposeful manner. For a few minutes she picked aimlessly with her fingers. She was sitting and did not fall. She did not lose consciousness though she neither responded to questions nor commands. This lasted several minutes and, on being questioned immediately on recovering full consciousness, stated that her right leg felt "furry and scratchy" and that voices sounded a "long way away."

She is right-handed.

PAST MEDICAL HISTORY: She suffered a head injury in childhood but could give us no further details.

EXAMINATION: Revealed an intelligent patient with complete insight, upset because the attacks made her life uncertain, but well adjusted.

There was no neurological abnormality.

# INVESTIGATIONS:

X-ray Skull: Normal

Electroencephalogram: Showed a mild generalized dysrhythmia, and a medium voltage 5-7 per second irregular activity focal to the left temporal area.

DIAGNOSIS: Temporal lobe epilepsy, probably posttraumatic.

DRUGS: Slight improvement was obtained with Phenobarbitone gr. 1 t.i.d. and Epanutin, gr. 1 t.i.d.

CASE (F20) Age: 55 years Sex: Male

HISTORY: A good history could not be obtained
in his state when seen. He had been well until
3 years previously when his mood changed from
placid to being irritable, in spells lasting
about two weeks at a time. During these spells
he was "suspicious," especially during the last
few months. He would appear frightened and at
times aggressive. He at no time lost consciousness. He on one occasion suddenly and without
reason attacked his wife with a look of extreme
anger on his face. He, himself, could add little

except that he was frightened all over, but was unable to describe his feelings. The fear, though poorly described, appeared spontaneous and without apparent cause.

He was right-handed.

EXAMINATION: He was disorientated and lacked insight, and showed evidence of recent intellectual deterioration. He had a left homonomous hemianopia and no other neurological signs.

### INVESTIGATIONS:

<u>Ventriculogram</u>: Showed a shift of the ventricular system to the right suggestive of a left temporal tumour.

Electroencephalogram: Showed some generalized slowing, but a high voltage slow wave focus appeared in the right temporal area.

POST MORTEM: A tumour measuring 80 mm. x 60 mm. x 50 mm. was found with its anterior end at a level with the right temporal pole and occupying nearly all the white matter of the temporal lobe. The temporal horn of the lateral ventricle was invaded from the lateral aspect and its lumen was obliterated by apposition of its walls.

Histological examination was reported by Dr. Blackwood as: Astrocytoma, grade 3.

CASE (F21) Age: 21 years Sex: Male HISTORY: He complained that for 2 years he was liable to episodes, of 30 second's duration. ushered in by " a rhythmic noise or tune, a few bars repeated, going through the mind." and almost immediately followed by a feeling of fear, very real and unpleasant and yet, "I did not know what I was afraid of." At this stage he felt unable to speak or understand what was said to him though he could hear conversation. While the tune appeared very familiar, at the time. he was at no time able to recall the music or name the piece. These episodes were almost always followed by a loss of consciousness. He worked in a deaf-aid factory and high frequency noises appeared to precipitate attacks, very few occurring otherwise.

He is right-handed.

FAMILY HISTORY: Revealed no significant facts.

PAST MEDICAL HISTORY: When aged 3 years he fell

lacerating his head badly, but does not know whether or not he was unconscious.

EXAMINATION: An intelligent young man, at first anxious lest his story would not be believed, as his own doctor had told him there was nothing wrong with him. No neurological abnormality was noted.

## **INVESTIGATIONS:**

X-ray Skull: Normal

Electroencephalogram: Showed much widespread moderate voltage 6 per second activity and rather frequent episodic runs of 2-4 per second, particularly in the left temporal region.

<u>DIAGNOSIS</u>: Temporal lobe epilepsy probably post-traumatic.

<u>DRUGS</u>: Epanutin, gr.  $l_{\frac{1}{2}}$ , b.i.d. and phenobarbital, gr. 1, b.i.d. was given and resulted in complete freedom, except for one attack, over 2 years.

CASE (F22) Age: 57 years Sex: Male

HISTORY: He stated that for 4-5 years he was

liable to attacks, abrupt in onset, when for no
apparent reason and unrelated to any particular
circumstance or mood, he would have a feeling
of terror, unlike and more severe than any fear

ever previously experienced. This was occasionally followed by a generalized convulsion, but usually the fear was isolated and lasted 1-2 minutes.

When it does occur, he has anxiety lest a fit ensue, but this anxiety is different. The first attack was 5 years ago. On waking in the morning he felt funny, then got out of bed to feel indescribably frightened for no apparent reason - a "terrible feeling of fear." He returned to bed and lost consciousness. The duration is not known, but on regaining consciousness he was clear mentally but was confused as to which was his right or left side. Weakness of the left hand has been present since.

CASE (F23) Age: 57 years Sex: Female

HISTORY: Nine attacks of loss of consciousness
during the preceding 18 months. No aura is recalled preceding 8 of these attacks. Preceding
one she was very frightened for no apparent
reason. Over the same period she has had many
attacks in which she has a peculiar indescribable
sensation during which she is intensely afraid,

is not sure where she is and is not completely conscious. She sits still, staring directly to the front.

She is right-handed.

<u>PAST MEDICAL HISTORY</u>: Nervous all her life but had no previous attacks of loss of consciousness or feelings of fear like these.

EXAMINATION: A very anxious excitable woman of low average intelligence who preferred to consider that her attacks were the result of "nerves."

There was no neurological abnormality.

## INVESTIGATIONS:

X-ray Skull and Chest: Normal

Cerebro-spinal fluid: Normal constituents

ELECTROENCEPHALOGRAM: Showed a slow wave, 2-3

per second, irregular activity focal to the right
low frontal, anterior temporal and post-central

areas on the right. Spiking potentials occurred
in both anterior temporal areas.

DIAGNOSIS: Epilepsy, cause unknown
DRUGS: Dilantin 0.03 grams, t.i.d. and phenobarbital 0.03 grams, t.i.d. This resulted in no

further attacks of loss of consciousness during 4 months, but occasional very brief episodes of "terrible feelings," "feelings of fear," occurred.

CASE (F24) Age: 32 years Sex: Male

HISTORY: He was subject to "fits! for 15 years.

During the day he would develop a feeling of fear which appeared to rise in a wave from the epigastrium, followed by a generalized convulsion.

Occasionally at the onset, according to his wife, he might sing a few snatches of a song in a strange way. Nocturnal attacks were preceded by waking from sleep screaming, immediately followed by a generalized convulsion lasting a few minutes. On waking he would immediately "demand sexual satisfaction." He showed several aggressive outbursts unrelated to the fits.

PAST MEDICAL AND FAMILY HISTORY: Revealed no significant facts.

He is right-handed.

EXAMINATION: A man of low average intelligence with poor command of verbal expression and rather paranoid in his attitude. No neurological abnormality was noted.

### INVESTIGATIONS:

X-ray Skull: Normal

Electroencephalogram: Showed, in the frontal region, runs of 6 per second irregular activity perhaps rather more on the left.

DIAGNOSIS: Temporal lobe epilepsy, cause unknown.

DRUGS: Epanutin, gr. 1½ t.i.d., completely

controlled the attacks for 2 years, but there

was no personality improvement.

CASE (F25) Age: 21 years Sex: Female HISTORY: Since aged 5 years she had frequent generalized convulsions with loss of consciousness. She has no recollection of an aura during the first few years. Now she has an aura in which she has a "funny feeling" in the epigastrium and a feeling of fear "all over." fear is different to "just being afraid of having an attack." "The fear is frightening." On some occasions during this phase, near-by objects have appeared to be at a great distance though at the same time she was fully aware that they were not. She has complained during this phase of things "stinking." On one occasion she

told her mother to "get away, you stink." During the early part of the attack she scratches her abdomen or head in an automatic fashion. On one occasion she had an isolated weakness of her right hand, otherwise there have been no focal motor components to the auraes.

PAST MEDICAL HISTORY: Some months prior to the onset of the attacks, she fell, striking the back of her head on a concrete floor. She was unconscious for 30 minutes.

FAMILY HISTORY: Her maternal aunt had convulsions when aged between 7 to 16 years.

EXAMINATION: A rather nervous young woman of average intelligence, but with good insight.

There was no neurological abnormality.

# INVESTIGATIONS:

X-ray Skull: Normal

Electroencephalogram: A high voltage focus associated with irregular delta activity appeared in the temporal, low-frontal and post-central area on the right, and was exaggerated during hyperventilation.

<u>DRUGS</u>: Mesantoin, gr.  $1\frac{1}{2}$ , b.i.d. and phenobarbital, gr.  $\frac{1}{2}$ , b.i.d., with partial control.

CASE (G1) Age: 21 years Sex: Male HISTORY: For about 12 years he has been subject to attacks in which he suddenly feels frightened, sweating, and trembling. He recognizes the feeling as one of fear, feels that he has been in the same circumstances and places before, without being afterwards able to recall where, and yet feels quite secure, "I know there is nothing to be afraid of. " At this stage he prefers to be alone because he knows an attack is imminent. The anxiety lest he have an attack is more upsetting in recent years than the "fear." The aura lasts some 10-15 seconds and then he may have a generalized convulsion or there ensues a period of a few minutes duration for which he is later amnesic. During these "amnesic" minutes, he has acted in an automatic way, e.g., climbed stairs or walked into another room. The frequency may be 4-5 per day.

He is right-handed.

PREVIOUS MEDICAL HISTORY: Eszema at 2-3 years; asthma started at 3-4 years and he still has frequent attacks. Liable to urticaria. No head injuries.

<u>FAMILY HISTORY</u>: His sister has eczema, brother has hay fever; father suffers from asthma.

<u>EXAMINATION</u>: An intelligent student of archi-

tecture. No neurological abnormality.

### INVESTIGATIONS:

X-ray Skull: Normal

Electroencephalogram: Showed a fair amount of paroxysmal bilateral 5-7 per second activity best seen in the frontal regions. Compatible with a deep mid-line lesion.

<u>DIAGNOSIS</u>: Temporal lobe epilepsy, cause unknown.

?? result of cerebral allergic lesion.

<u>DRUGS</u>: Phenobarbital, gr.  $\frac{1}{2}$ , q.i.d., Epanutin, gr.  $1\frac{1}{2}$ , t.i.d., without any improvement in the frequency of attacks.

CASE (G2) Age: 51 years Sex: Female

HISTORY: For 5 years she has been liable to

attacks of sudden loss of consciousness without

warning. Interspersed with these are more frequent attacks in which she finds herself unable to think or speak, able to hear a conversation but not understand it; hearing a hub-bub of conversation inside her head and feeling intensely that she knows what is said, but is never able to repeat it. For some 4 months she has been liable to similar attacks but with the addition of an indescribable taste and smell, more unpleasant than anything real and a "feeling of dread." She is anxious about the attacks but is frightened by the feeling of dread, by the nightmare. These attacks last several minutes. During some of them she may carry on doing her work without recollection later of having done so. One attack was personally observed. She suddenly complained of a horrible smell, appeared agitated and afraid. She exclaimed, "Oh. I am afraid." To the query, "What are you afraid of?" she replied, "I don't know. I don't know." To the question, "Are you afraid of the smell?" she replied, "No. I am just afraid. I am so afraid." She now began to smack the lips,

and stated that she heard voices. To further questions she replied in a jargon aphasia. After about  $l_{2}^{\frac{1}{2}}$  minutes she lost consciousness and had a generalized convulsive seizure with pallor, sweating, increased pulse rate and noisy passage of flatus. On regaining consciousness, she was amnesic for the whole event.

She is right-handed.

PREVIOUS MEDICAL AND FAMILY HISTORY: Revealed no relevant facts.

EXAMINATION: She was rather depressed and now despaired of her sanity" on account of the attacks. She showed a mild intellectual deterioration. No neurological abnormality was noted.

# INVESTIGATIONS:

X-ray Skull: Normal

Ventriculogram: Normal

Bilateral Carotid Air Arteriogram: Normal

Electroencephalogram: Showed a bilateral 5-7 per second dysrhythmia more marked on the left side, but not definitely focal.

<u>DIAGNOSIS</u>: Epilepsy. In view of the progressive intellectual deterioration over the succeeding two years, cerebral atrophy was considered as

the likely pathology.

<u>DRUGS</u>: Phenobarbitone, gr. 1, t.i.d., and Epanutin, gr.  $1\frac{1}{2}$ , **4.i.d.**, led to a marked decrease in the frequency of the attacks.

CASE (G3) Age: 12 years Sex: Male

HISTORY: He complained that for 12 years he was liable to occasional attacks of loss of consciousness of a few seconds duration. These were preceded, for 2-3 minutes, by a feeling of fear arising on its own without any general discomfort, severe, but in itself not frightening, so that he felt that he was looking at it in an objective way. He could continue thinking and acting despite it except that he had a vivid prescience of what was about to happen. Thus, if he watched a television show during this phase, he would know exactly what would next be said or done. The loss of consciousness was not invariable. He is right-handed.

<u>PAST MEDICAL HISTORY</u>: An accident 6 months prior to onset, resulted in a fractured skull and vertebra and he was unconscious for several

minutes.

FAMILY HISTORY: Revealed no relevant facts.

EXAMINATION: Well adjusted male of average intelligence. No neurological abnormality noted.

INVESTIGATIONS:

X-ray Skull: Normal

Electroencephalogram: Showed a dominant unstable 9-10 per second activity slowing to 6 per second. The abnormality remained generalized.

<u>DIAGNOSIS</u>: Temporal lobe epilepsy, post-traumatic.

<u>DRUGS</u>: Anticonvulsants were started, but the patient did not return for further evaluation.

CASE (G4) Age: 42 years Sex: Male

HISTORY: For 12 years he was liable to sudden

feelings of fear accompanied by a momentary cold

feeling in his epigastrium. This would give way

to a state in which he felt that whatever he

was doing was being relived and that he knew

precisely what was about to happen. This, though

it at the time felt very long, never lasted more

than a few seconds, and was always followed by

a loss of consciousness.

He is right-handed.

FAMILY HISTORY: Revealed no relevant facts.

PAST MEDICAL HISTORY: He was injured in a motor car accident 13 years previously, following which he was unconscious for several minutes.

EXAMINATION: He was of average intelligence with good insight. Neurological and general examinations were normal.

## **INVESTIGATIONS:**

X-ray Skull: Normal

Electroencephalogram: Showed a paroyxsmal 6 per second generalized dysrhythmia with some slower waves as well as low voltage fast activity, chiefly in the frontal region.

<u>DRUGS</u>: Temporal lobe epilepsy, post-traumatic.

<u>DRUGS</u>: Phenobarbital, gr. 3/4, t.i.d. and Epanutin, gr.  $1\frac{1}{2}$ , t.i.d., resulted in control of the attacks during the 6 months he was observed.

CASE (G5) Age: 29 years Sex: Female

HISTORY: She complained of generalized convulsions of varying frequency for 11 years. These were preceded by aurae: onset with a hallucination of rotation of surroundings of a few moments! duration, followed by a hallucination of hearing voices.

The voices might occur without being followed by a fit and then would last up to 5 minutes. She at the time "knows" what the voices are saying, but is unable later to recall what though she feels that they are unfriendly. These aurae may be followed by a feeling of fear, or the fear may occur by itself. She then feels that there is something above and is afraid to look up. She has looked up, seen nothing there, and has still been afraid. The generalized convulsions last a few minutes. She is right-handed.

PAST MEDICAL AND FAMILY HISTORY: Revealed no significant facts.

EXAMINATION: A female of low average intelligence fairly well adjusted. There were no abnormal findings on neurological and general examination.

INVESTIGATIONS:

X-ray Skull: Normal

Electroencephalogram: Showed a bewildering museum of epileptic outbursts of various kinds.

Otological investigations were reported by Dr. C. Hallpike, The National Hospital, as being normal.

<u>DIAGNOSIS</u>: Epilepsy of temporal lobe kind, probably cause unknown.

DRUGS: Phenobarbitone, gr. 1, t.i.d., Epanutin, gr. 1½, t.i.d., gave good control of the major attacks; the aurae were not so well controlled.

On changing her doctor she was referred to a mental institution, diagnosed as schizophrenia.

The anticonvulsants were withdrawn. She then had very frequent attacks (serial epilepsy) and was discharged. She again responded well to Phenobarbitone and Epanutin.

CASE (G6) Age: 34 years Sex: Female

HISTORY: For 20 years she was liable, without
warning, to generalized convulsions and to
frequent jerking of limbs in the early morning.

In addition, she had more frequent attacks of
clouding of consciousness, of a few minutes!
duration, preceded by a feeling of fear "rising
suddenly from nowhere," lasting a few minutes.

She would then feel "unreal and strange," as
though living in a strange, unreal world she
could not describe, at the same time knowing
that she, herself, was quite real and that there
was a "real world."

She is right-handed.

PAST MEDICAL AND FAMILY HISTORY: Revealed no relevant features.

EXAMINATION: A well adjusted female of average intelligence. There were no abnormal neurological findings.

## INVESTIGATIONS:

X-ray Skull: Normal

Electroencephalogram: Showed a generalized paroxysmal 5-? per second dysrhythmia.

<u>DIAGNOSIS</u>: Epilepsy, possibly multifocal with temporal lobe features.

DRUGS: Response to therapy was not observed.

CASE (G7) Age: 16 years Sex: Female

HISTORY: Since aged 12 years she was liable

to attacks of loss of consciousness with generalized

convulsions and to frequent petit mal. From

the age of 9-10 years she recognized that prior

to the attack she felt afraid, but did not

know what of. She stated that the fear "just

came" but was unable to elaborate. At times

the fear would be followed by a feeling of the

room "turning round and round."

She is right-handed.

PAST MEDICAL AND FAMILY HISTORY: Revealed no relevant facts.

EXAMINATION: Of average intelligence but unable to give a good description of her aurae. There were no abnormal findings.

### INVESTIGATIONS:

X-ray Skull : Normal

Electroencephalogram: Showed some generalized slowing for this age; mildly abnormal.

<u>DRUGS</u>: Tridione, 0.3 gms., t.i.d., resulted in complete control of the petit mal. Phenobarbitone, gr.  $\frac{1}{8}$ , t.i.d., and Epanutin, gr.  $1\frac{1}{8}$ , t.i.d. resulted in only slight control of the attacks.

<u>DIAGNOSIS</u>: Epilepsy, cause unknown.

CASE (G8) Age: 56 years Sex: Female

HISTORY: For the past 4-5 years she was subject
to attacks preceded by a sudden feeling of
restlessness so that she felt compelled to move
from one room to another; this was associated
with a horrible fear and a queer feeling in the

stomach; occasionally she might feel dizzy. Her husband stated that during an attack chewing movements of the jaws occurred, and that she might behave as though in a trance, undressing herself, starting to prepare a meal even though a meal may have been finished within the past few minutes.

She is right-handed.

PAST MEDICAL AND FAMILY HISTORY: Revealed no significant facts.

EXAMINATION: An intelligent female with complete insight, rather amused by her odd behavior.

She had come to recognize the attacks as part of her life. There were no abnormal neurological findings.

# **INVESTIGATIONS:**

<u>Left-carotid arteriogram</u>: Normal

Air encephalogram: Normal

<u>Electroencephalogram</u>: Showed a generalized 5-7/sec. irregular slowing.

Otological investigations by Dr. C. Hallpike,
The National Hospital, showed a well-marked
directional preponderance of nystagmus to the
left on caloric testing, and a slight directional

preponderance to the left of optokinetic responses, suggestive of a temporal lobe lesion. DRUGS: On Rutonal, gr. 3, t.i.d. and Epanutin, gr.  $1\frac{1}{8}$ , b.i.d., the frequency of the attacks diminished slightly.

CASE (N1) Age: 67 years Sex: Male HISTORY: He complained that for some 16 years he had frequent attacks of fear, sudden in onset, occurring during the day or night. He might wake up screaming during the night. These attacks would last 2-3 minutes. The feeling would start in the epigastrium, and then a fear "a sort of something terrible going to happen," "I cannot compare it with any fright I have ever had." After the attacks, he has "a fear that I am going to die." but that is a different fear, an "angor animi," the sense of dying. The fear had no shape or form but "appeared to be in front of me." It was usually associated with vertigo, a marked sensation of rotation of self. On several occasions, not related to the attacks, he had a visual hallucination of his fiancee

of old appearing in front of him, her face turned away. At times, again not related to the fear, he has had vivid 'deja vu' experiences. For 15 years his attacks were considered to be psychogenic, even by two neurologists. In November, 1950, he had a similar attack of fear, crying out that he was going to die, and developed a severe occipital headache but without loss of consciousness.

EXAMINATION: On admission to the hospital he had marked neck rigidity and bilateral papilloedema.

Cerebro-spinal fluid: Showed a pressure of 200 mms. water and was heavily blood stained. He died 27 days later and on post-mortem an aneurysm was discovered at the junction of the anterior cerebral and anterior communicating arteries.

Electroencephalogram: Recorded one year prior to death showed no significant abnormality.

Dr. Blackwood, The National Hospital, reported that sections of the frontal cortex, mid-brain and upper pons showed no lesions histologically.

CASE (N2) Age: 64 years Sex: Male

HISTORY: He was for 3 years subject to brief

attacks of loss of consciousness. Preceding

these attacks, and usually isolated, were

"peculiar feelings," a feeling of intense fear

and a desire to run out of the house. The fear

would arise irrespective of his thoughts, mood,

or situation.

He is right-handed.

PAST MEDICAL AND FAMILY HISTORY: Revealed no relevant facts.

EXAMINATION: When first seen, he was depressed and weepy, afraid lest he might become insane. No neurological abnormality. B.P. 180/90, with evidence of generalized arteriosclerosis.

X-ray Skull: Showed the pineal calcified and in mid-line.

X-ray Chest: Normal

Electroencephalogram: Normal

<u>DIAGNOSIS</u>: Epilepsy, probably on a cerebro-vascular basis.

DRUGS: Phenobarbitone, gr. 3/4, and Epanutin, gr.  $1\frac{1}{2}$ , each b.i.d., resulted in a complete remission of

the attacks during the 7 months he was observed.

CASE (N3) Age: 47 years Sex: Male

HISTORY: For 28 years he was liable to generalized convulsions, usually nocturnal. The

attacks during the day were commonly preceded
by a feeling of "vagueness," a feeling of "not
being real." Some attacks, but not all, were
preceded by a feeling of fear "coming out of
the blue," "as though being attacked without
knowing how to defend myself." When any aura
occurred, he admitted to being afraid, "but this
fear is different." Occasional attacks were
preceded by vertigo without fear. On one
occasion, a similar fear woke him up during the

He is right-handed.

night.

PAST MEDICAL HISTORY: When aged 7 years he fell downstairs, injuring his head. He stated that the injury was severe but could not recall further details.

FAMILY HISTORY: Revealed no significant facts.

EXAMINATION: A patient of higher than average intelligence who, in a way, enjoyed describing his attacks. No abnormality was noted on neurological and general medical examinations.

X-ray Skull and Chest: Normal

Electroencephalogram: Normal

<u>DIAGNOSIS</u>: Epilepsy with temporal lobe features, possibly post-traumatic.

DRUGS: Phenobarbitone, gr. 3/4 t.i.d., and Epanutin, gr. 1½ t.i.d., gave moderately good control of attacks.

CASE (N4) Age: 28 years Sex: Female

HISTORY: She complained of blackouts for 12

years, each lasting 2-3 seconds and preceded by
an aura. The aura consisted of an epigastric

sensation and a few involuntary lip smacks without
a gustatory or olfactory component. On several

occasions she had a marked fear, a fear which
came on its own, without apparent reason, and
was quite indescribable. More frequently, and
usually not related to the fit, her surroundings
would suddenly appear "far, far away" but without

"any change in size," so that "everything seemed unreal as though I was not in this world," and yet during the episode she knew that she was quite real. This would be accompanied by voices "muttering," "going up-and-down in a boring monotonous way."

She is right-handed.

PAST MEDICAL AND FAMILY HISTORY: Revealed no significant facts.

EXAMINATION: A female of average intelligence, emotionally not disturbed. No neurological abnormality was noted.

### INVESTIGATIONS:

X-ray Skull - Normal

Electroencephalogram: Normal

DIAGNOSIS: Temporal lobe epilepsy, cause unknown.

<u>DRUGS</u>: Poor response to phenobarbitake and Epanutin.

CASE (N5) Age: 40 years Sex: Male

HISTORY: For 9 months liable to attacks which woke
him from his sleep with a "peculiar emotion,"

"a feeling of fear," though not related to a dream

or thoughts. It was intense and in a way he felt apart from it. Accompanying it would be an odd hallucination as though a "motor had started inside my head." This was not a noise, but an "odd feeling." He also had an auditory hallucination of water dripping from a tap in the distance, but though at a distance unusually clear. In the early attacks he was almost convinced that the water was in fact "dripping," though "it was something different from an ordinary dripping, it was too clear." The duration of the attack varied from 5-10 minutes. He was right-handed.

PAST MEDICAL AND FAMILY HISTORY: Revealed nothing relevant.

EXAMINATION: He was seen by Dr. Eliot Slater, consulting psychiatrist to The National Hospital, who found no somatic manifestation of anxiety, and considered the condition to be epileptic.

# INVESTIGATIONS:

X-ray Skull and Chest: Normal

Electroencephalogram: Normal

DRUGS: After investigation he was not seen again.

CASE (NEI) Age: 17 years Sex: Female HISTORY: She complained that since aged 16 months she was liable to occasional attacks of loss of consciousness without convulsions. From an early age she also had "trembling" spells in which she would follow her mother, clutching at her. During these she appeared pale and frightened. spells would last about one minute. The girl now says that she gets a peculiar feeling in the stomach and feels frightened, but does not know what she is frightened about, "just afraid." This is at times associated with dizziness, "things going round." After an attack, whether associated with loss of consciousness or not, she is immediately afterwards free from fear and ravenously hungry, eating anything she can get hold of in a manner, according to the mother, most unusual for her.

She was right-handed.

PAST MEDICAL HISTORY: At 10 months she fell, raising a lump over the vertix, and was unconscious for a few minutes.

FAMILY HISTORY: No relevant features.

EXAMINATION: Of low average intelligence.

No neurological abnormality was revealed.

X-ray Skull: Normal

This case was seen before the investigation started and an electroencephalogram was not obtained.

<u>DRUGS</u>: She had for years taken phenobarbitone and Epanutin in varying doses but with poor effect.

CASE (NE2) Age: 52 years Sex: Male

HISTORY: For over 3-4 months he had several

attacks of loss of consciousness with generalized convulsions lasting 5-6 minutes. Preceding
these attacks, according to his wife, he appeared
startled and pale and his speech was a jargon
aphasia. At the onset of the attack he smacked
his lips. He described his aura as being a
smell, a very small, ugly, dirty, black woman
smelling of onions, a feeling in the epigastrium,
and a feeling of fear. He at the time was
dysphasic and ill, and the detailed sequence of
the aurae could not be obtained. He denied

being afraid of the hallucination though the fear occurred apparently at the same time. This olfactory aura he related to his visual hallucination. He denied ever having seen the woman before.

He was right-handed.

PAST MEDICAL AND FAMILY HISTORY: Revealed no relevant facts.

EXAMINATION: Bilateral papilloedema; partial right homonymous hemianopia, moderate expressive and less marked receptive aphasia, and slight weakness of the right side of his face and of his right hand. An urgent operation was performed by Dr. Harvey Jackson, The National Hospital. A large glioma was encountered in the left temporal lobe. Removal was not attempted.

Histology of a biopsy was reported by Dr. Black-wood as showing a malignant glioma, Astrocytoma, Type IV. A post-mortem examination was not permitted.