STUDIES IN TRAUMATIC EPILEPSY*

3. VISUAL FITS

BY

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The study of epileptic phenomena in wounds of the visual cortex in the widest sense is of special interest, as the clinical features of such cases give relatively precise information regarding the parts of the brain injured.

The cerebral part of the visual mechanism consists of the optic radiation, in which there is a close cell-to-cell pathway between the cells of the lateral geniculate body, and the striate cortex (area 17), which differs from the main sensorimotor area in having no clearly defined projections from the thalamus proper. After this a further wide area of cortex is involved, with visual functions such as spatial orientation, visual recognition and reading, and the visual parts of the body scheme and memory in general. Such areas—the “visual association” or “visuo-psychic” areas—must be regarded as subserving “higher” visual functions in Jackson’s sense.

Lesions of the radiations will give sharp-edged visual field defects of hemianopic, quadrantanopic, or segmental outline. Lesions of the striate cortex itself will also cause sharp-edged homonymous defects which may be hemi- or quadrantanopic, but which often have a characteristic scotomatous pattern (Spalding, 1952). Lesions confined to the “higher” visual cortex may sometimes produce permanent changes in aspects of spatial orientation and visual recognition, or such defects as visual inattention, imperception of a stationary as opposed to a moving object, loss of ability to localize an object, or vague blurring of vision in part of a field (though it is possible that some of these may also occur in more peripheral lesions of the visual apparatus); but often they seem to be clinically silent. However, it should be remembered that such “silence” may be in part due to our present ignorance of the functions of these areas. It is thus evident that an exact study of visual fields is of considerable value in localizing the site of wounds,

and this is so even when more than one part of the visual pathway is involved. In consequence, the clinical localization in this group of cases is in some ways more accurate than in those involving the sensorimotor cortex.

We must regard the arrival of the neural impulses at the area striata as the first comparatively simple step of a progressively elaborate series of brain events which develop in neighbouring and more remote areas of the brain. This elaboration of what arrives at the striate cortex must be early concerned with the contralateral visual cortex, as well as the adjacent areas 18 and 19, so that the correlation of both sides may form a coordinated whole. Then, the complex mechanisms of memory leading to the ability to recognize, read, place in relation to body and its environment, etc., must be involved. It may be assumed that for an epileptic visual phenomenon (such as flashing lights appearing in one visual field) to occur, some part of the contralateral visual mechanism must be sufficiently active to cause this hallucination. It is, however, largely unknown which parts of the visual pathway must be intact to allow of the development of certain types of visual hallucination. Crude flashing phenomena are generally held to originate near the visual cortex, but this is a loose anatomical phrase and the matter should, if possible, be investigated more precisely. The appearance of hallucinations in blind half-fields is of special interest, because some part of the visual mechanism for that field must be preserved for visual phenomena to occur therein. It may be supposed either that the damaged area, although not able to function “normally”, has yet enough cells left to fire epileptically, or that areas separate from, and at a higher level than, the damaged area themselves fire spontaneously to produce the hallucination. The development of homonymous hemianopia as an epileptic phenomenon is also worth careful consideration, and suggests that some groups of cells have inhibitory or suppressor functions, and, since these cases have intact fields

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between attacks, they would seem to be in areas other than those concerned primarily with positive visual reception. Finally, the complex hallucinations with recognizable objects, people, places, or events give a glimpse into one of the highest cerebral mechanisms. Such hallucinations at times appear similar to visual memory and are referred to by the subjects experiencing them as "memories". Presumably all recognition must involve a memory process; however, these complex phenomena, which may form a static picture but often have a temporal element, in that events occur during hallucinated scenes while they last, would seem particularly to involve the highly integrated mechanisms that are usually referred to as "memory".

Reference to cortical function as a separate entity involves an over-simplification of a complex mechanism, for all cortical activity leads to a multitude of associations along many pathways, and in this connexion the cortico-thalamic connexions are no doubt specially important. Thus it is of interest to consider not only which part of the visual system is discharging, but also whether the discharging focus is in any way isolated from lower levels of the visual mechanism, or whether the pathways to the higher visual centres are intact or damaged, for functional damage to higher cortical mechanisms may presumably be caused by injury to white matter conveying cortical association tracts or thalamic connexions. The connexions of the occipital and parieto-occipital lobe with the pulvinar are obviously of great importance, and in many of our cases these connexions must be damaged. Furthermore, we must bear in mind the possibility that para-calcarine cells are just as likely to originate visual hallucinations as are those of area 17 itself.

In this study, therefore, these problems and possibilities should be kept in mind, and an attempt made to search for anatomical implications and correlations.

Case Material

This paper reviews 60 cases of brain wounds having epilepsy with a visual aura, and Table II gives a summary of the clinical details, including tracings of skull radiographs and visual field charts where available, for all cases. It can be seen that a wide variety of visual phenomena occur, from the simplest unformed visual hallucination to such complex effects as remembered scenes or agnosia for visual objects.

Evidence as to the exact site of a lesion is largely inferential and may be inaccurate. Bearing this in mind, we have nevertheless attempted to group the

cases into three main anatomical divisions: (1) Those with injury chiefly to the calcarine cortex, based on the type of field defect—a homonymous congruous scotomatous defect or defects—combined with the site of wound and direction of missile; (2) those with definite radiation lesions as indicated by a permanent hemianopia, quadrantanopia, or sharp segmental field defect, and the site and direction of the wounds; (3) those with lesions of the "higher" visual cortex—that is the parietal or occipital areas, but forward of the calcarine cortex itself, based on the site and direction of the wound together with an early transient field defect or some permanent change such as visual inattention, disorientation, agnosia, etc., but without the interruption of optic radiations which produces a permanent dense field defect. Such a grouping is not necessarily exclusive, since wounds of the radiations, for instance, will in many, if not the majority of, cases involve also "higher" visual cortex and some of the calcarine wounds may also involve other visual cortex or radiations. Nevertheless, it is of some practical value in attempting to correlate the site of wound and type of fit.

Group I: Wounds of the Calcarine Region

Nine cases have been placed in this group, but there are remarkably few small wounds of the calcarine cortex with focal visual fits, and in still fewer of these cases is the field defect confined to the dense paracentral scotoma characteristic of localized posterior calcarine wounds. Cases 1 and 2 are perhaps examples of this. Case 4 had some destruction of one occipital lobe as well as of the calcarine cortex. Case 9 had widespread damage to the left hemisphere as well as a lesion of the right calcarine area, while Cases 5, 6, and 8 almost certainly had additional radiation lesions. Case 3 had a small wound near the calcarine cortex though its exact anatomy cannot be determined. Case 5 is one of almost complete cortical blindness, and sparks of light appear as an epileptic aura. Presumably these discharging cells are situated in the upper occipital region at a physiologically higher level than the calcarine cortex.

In this group the visual hallucinations tend to be continuous unformed visual phenomena, usually with an element of movement in them: weaving patterns, zigzag lights, showers of sparks or coloured clouds are described, rather than interrupted flashes of light. The hallucinations appeared also in the segments of field loss due to damage to the calcarine cortex, and this supports the view that these crude visual phenomena originate at a higher level than area 17. In one case (No. 6), in which a missile was removed from the right calcarine cortex but

with additional more anterior cortical damage, there was a continuous sensation of coloured lights in the blind part of the field. Although varying in intensity, this had been present continuously for nine years since wounding. In some ways this is reminiscent of the continuous dyaesthesiae or pain sometimes seen in thalamic lesions. However, as has been pointed out, none of these cases had very sharply localized wounds and it may be assumed that cerebral damage extended beyond area 17.

In order to provide a background of the general incidence of epilepsy in these cases, Table I analyses the type of fit occurring in an arbitrary 225 cases which fall into the three anatomical groups we are considering.

In Group I there is a lower incidence of epilepsy in the unilateral wounds of the occipital pole than perhaps in any other area of the brain (six out of 24), but when fits occur they usually (four of the six) have a visual component.

The bilateral wounds of the occipital lobes also have a relatively low (11 out of 36) total incidence of fits and a strikingly low incidence of visual fits, which appeared only in one case.

There is therefore little to suggest that patients with small wounds of the calcarine cortex are specially liable to visual fits; indeed fits of any sort seem less likely to occur than with wounds in most other cortical sites.

Group II : Radiation Lesions

Wounds of the optic radiations will always involve parts of the cortex also. Thus they may injure the upper part of the occipital lobe and nearby parieto-occipital and posterior parietal cortex and spread down to involve radiations, or they may involve the temporal cortex more anteriorly and penetrate to sever the anterior part of the radiations. The pattern of visual fits that develops may therefore be affected both by the cortical wound and the radiation lesion. Of the 21 cases in this group, there are relatively few with wounds of the anterior part of the optic radiation and visual fits. The most clear cut are those in which chiefly the lower half of the optic radiation in the temporal lobe is damaged. There are only two examples in the series (Cases 22 and 23). In the first, the visual fit took the form of a negative phenomenon—hemianopia followed by flashing lights in the extinguished field: in the second, elaborate, formed visual hallucinations occurred. However, the total incidence of epilepsy in such wounds is by no means low, for in a group of 42 cases with this anterior type of radiation lesion no fewer than 22 had fits of some sort (Table I).

In eight of the cases with hemianopia or lower

TABLE I
TYPE OF FIT IN THREE MAIN GROUPS OF CASES

	Total	No Fits	Grand Mal with No Aura	With Visual Aura	With Non-visual Aura	Total Fits
Group I unilateral ..	24	18	1	4	1	6
(Calcarine) bilateral ..	36	25	6	1	4	11
Group II anterior radiations giving field defects ..	42	20	11	1	10	22
Group II more posterior wounds with field defects ..	37	14	3	11	9	23
Group III higher visual cortex without permanent field defect ..	86	40	15	13	18	46
Totals	225	117	36	30	42	108

quadrant defects there was also some permanent hemiparesis, while in 13 (including the two mentioned above) there was no permanent motor loss. Wounds in the former group were in general probably rather more anterior than in the latter, Nos. 22 and 23 being exceptions, but no obvious differences emerge in the pattern of visual fits which occur in the two groups. In all these cases the occipital poles were intact and the radiation lesions must have produced isolation of at least a part of the calcarine cortex from afferent visual impulses. Isolated neurons are known to show rhythmic spontaneous firing (Brazier, 1951), and "deafferentation" of an area of cortex might therefore be expected to influence both the pattern and frequency of epileptic fits firing from it. In Table I, of a total of 79 cases with radiation lesions and partially isolated calcarine cortex, 45 had fits of some sort, but only 12 of these had a visual aura. Such isolation does not therefore seem important in producing visual fits. These figures also make it unlikely that an irritative lesion of the radiation alone has epileptic potentialities. As Table I shows, when radiations are involved more posteriorly and the higher visual cortex is therefore probably also injured, the incidence of epilepsy rises and a higher proportion of those with an aura have a visual one.

When we consider the pattern of visual seizures in these radiation lesions, two broad groups can be recognized. First, complex phenomena such as formed visual hallucinations, distortions, and loss or blurring of a half-field, and secondly, crude phenomena such as interrupted flickering or flashing lights. Only one case showed the continuous moving lights commonly seen in calcarine wounds. As will be shown later, complex hallucinations and negative phenomena tended to occur in cases where

the higher visual cortex was damaged without there being any permanent dense field defect. It may therefore be supposed that such phenomena in this present group represent ictal firing from the cortex damaged at the wounds of entry and are not influenced primarily by the injury to the radiations. Formed visions or distortions occurred in five, and negative effects in six, of these 21 cases; in one continuous moving lights, and in one a ball of stationary light followed the negative effects. Interrupted flashes occurred in 10, and in a further one such flashes also occurred together with the extinction of part of a field as a negative effect. Interrupted light is thus a common phenomenon when visual fits do occur in this group. It may represent one aspect of the spontaneous firing of isolated calcarine cortex, though the following case makes this unlikely to be the whole explanation.

Case 17.—Fits would begin with the sudden flashing on and off at a rate of about two per sec. of a bright yellow light in the left (blind) visual field. This continued for a few seconds and the patient would lose consciousness and have a generalized grand mal attack.

Following surgical excision of a part of a right temporo-occipital brain scar and of an area at its anterior margin where a spike focus was defined by electrocorticography, the fits became less frequent and changed in character. Usually no aura of any sort occurred and the patient would suffer the abrupt onset of a grand mal convulsion. Rarely a brief aura, "a feeling of unsteadiness", would precede the convulsion.

In this case the removal of cortex well away from the occipital pole abolished the flashing. This cannot have affected whatever spontaneous activity occurred in the calcarine area. It may be argued that the hallucinations of flashing lights resulted from the arrival of calcarine discharge at the more anterior cortex, which was removed. However, the excision extended deeply and would have interrupted connexions between the pulvinar and peristriate cortex, and the consequent interruption of the thalamic contribution to cortical activity may just as well have been responsible for the result. The case serves to illustrate once more the complexity of cortical function.

That a high proportion of non-visual fits occurs with wounds in the anterior occipital or posterior parietal and temporal areas causing radiation lesions is understandable if we consider that these fits represent a discharge from cortex at the site of entry which may have only slight associations with visual function.

This group provides some interesting examples of complex hallucinations, and although they must be grouped anatomically as radiation lesions some of them will be discussed more fully in the section

dealing with cases of "higher" visual cortex lesions without permanent field defects.

Group III. Higher Visual Cortex Lesions

There are 30 cases which fall into this category, several with more than one kind of visual fit. Their most frequent and perhaps best defined clinical manifestations are the negative ones—complete blindness, or hemianopia, or sudden blurring in a half-field. This occurred as the onset to an attack in 16 cases. Sudden bilateral blindness, or hemianopia, or a gradual field defect spreading to involve one half-field or the whole of vision are all exemplified here. In one case the defect was a momentary affair like a shadow crossing one half-field. In addition, blurring of vision so that objects could not be seen clearly occurred both in one half-field and for complete vision. In four cases after the extinction of part of a field, interrupted flashes of light then appeared in the blind field, a sequence of events noted by Penfield and Jasper (1954) following electrical stimulation of certain peristriate points, and in two cases a continuous stationary light was experienced. It is noteworthy that no instances of formed hallucinations were recorded following a negative visual phenomenon.

These are posterior and often high parietal wounds and usually well above the optic radiation. There may be some sensory or motor loss but there is no dense visual field defect. Some of the negative phenomena appear to be examples of true inhibition and may be compared with the inhibition of motor activity as reported in our previous study of focal motor fits (Russell and Whitty, 1953). The tendency for a hemianopic or complete field distribution rather than any scotomatous or segmental type of defect to occur is striking.

In 18 cases distortions of vision occurred. Objects seemed far away, in the wrong place, unfamiliar, and distorted in shape, or actually appeared to be moving (both receding and moving to one side were described) when stationary. In one (Case 50) there was a definite spatial disorientation sufficient to make it difficult to move around in familiar surroundings. One other patient (Case No. 34) had a visual agnosia during his attacks. This patient had some permanent difficulty in localizing objects presented in the left half-field, and objects appeared less distinct on this side. However, there was no complete field defect of any sort. Nor was there any permanent difficulty in recognizing objects, though there was possibly a slight dyslexia and a definite dyscalculia. His attacks started with the abrupt onset of a left hemianopia, which was followed rapidly by a dysphasia, receptive and

expressive. Sometimes at this stage he would be unable to recognize objects though he could see them. This occurred only in some attacks, and was quite distinct from his dysphasia which constantly accompanied all attacks.

The case is of special interest because it illustrates the problem of what aspects of visual function must be regarded as a whole-field phenomenon. Agnosias are generally considered to be such, though recently Bay (1953) has suggested that they may, in fact, sometimes be only perceptual difficulties which may be limited to a part of the visual field, though producing a general effect on visual appreciation. In the case here described certain visual defects were permanently present in one half-field, and a complete hemianopia occurred during attacks, sometimes with and sometimes without agnosia. The hemianopia cannot therefore be a factor in the agnosia: and the case seems to support the view that agnosia is a defect of total vision fired from one hemisphere.

Attacks in which distortion of vision, apparent displacement of visual objects, and spatial disorientation occur, examples of all of which are recorded here, also illustrate this problem, since in all of them a localized field abnormality might be the basis of what the patient describes as a generalized effect.

In five cases aphasia occurred as part of an attack with visual element. In one case (Case 58) it initiated the attack and was followed by visual phenomena—disappearance of objects and a bright light. In the other four (Cases 34, 42, 50, and 57) visual phenomena appeared before the aphasia. Of these, one (Case 34, already detailed) had an occasional associated visual agnosia in his attacks and another (Case 50) occasional spatial disorientation. This association with aphasia is worth comment, for, unlike the focal attacks occurring in wounds of the sensorimotor cortex (Russell and Whitty, 1953), the present cases show little tendency for the content of fits to spread from visual to non-visual phenomena or vice versa. Thus, of the whole series, in only nine cases did an aura start as a visual one and spread to the non-visual sphere. In four cases the spread was to aphasia, in four to positive motor phenomena, and in one to sensory. Seven cases showed a spread from a non-visual to a visual aura, and of these one started as aphasia, four as a focal motor phenomenon, and two as a non-visual sensory one. So far as a change of a visual aura itself is concerned, it is interesting that 11 instances showed a progress from negative to positive visual phenomena, while only one showed a change from positive to negative and only two a change from one form of positive to another.

In two cases in this group interrupted flashes of light occurred as the whole content of a focal fit, in addition to the four cases where this was secondary to the extinction of part of a field. In one case also continuous moving lights, of the kind more commonly seen in calcarine lesions, were reported.

Many unusual varieties of focal fit may originate in the posterior parietal lobe. Thus in Case 54 the eyes were forced upwards, and in Case 40 adverse movements of the head and eyes occurred from small wounds in this region.

But perhaps the most interesting of any are those in which visual aspects of the body image and memory mechanisms appear to be involved. Two cases of the former and five of the latter are recorded in this group; however, as further examples of both these also appeared in the group of radiation lesions we consider them together in the present section. The anatomical group to which each case is assigned may be seen from Table II.

Before mentioning them in more detail, we may see from Table I that the general incidence of epilepsy in 86 cases of Group III is high (46 out of 86). However, of the 31 of these with focal fits or aura, only 13 were visual and 18 non-visual. This is probably a reflexion of the fact that some of these wounds impinge on the more posterior parts of the main sensorimotor cortex.

In four of our present cases the visual aspect of the body image was involved in their attacks. In two there was a localized involvement, and in two the upset was more general.

Case 40.—This patient had four types of attack: grand mal without warning; right adverse attacks involving the hand and eyes which led to momentary loss of consciousness; and a sudden shadow moving across the right half visual field, “like a cat running across” without loss of consciousness. In addition there were episodes in which his right hand would suddenly seem to be raised up above his head, and he would look up towards it and seem to see it momentarily, and on occasion had asked his wife to pull it down. The attacks lasted for less than 30 seconds, and ended abruptly; there was some confusion during them, but if his attention was directed towards his hand in its real position he would admit to seeing it there.

Case 25.—This patient's epilepsy was of sudden attacks in which his left hand looked queer and out of place; it did not appear to be in the place he felt it. He felt the limb did not belong to him, and would waggle his fingers and watch them moving to assure himself that what he was looking at was his own limb. This sensation lasted for a few seconds only, after which he lost consciousness and had a generalized grand mal convulsion. On other occasions he had the aura only, without any grand mal attack.

TABLE II: SUMMARY OF CLINICAL DETAILS

Clinical Features	Type of Fit	Skull Radiograph	Visual Fields
GROUP 1.—Calcarine Lesions			
1 (158). Midline. Handedness not known. No sensory or motor loss	<ol style="list-style-type: none"> 1. Zigzag ring of light in front→gets bigger till ring reaches limit of sight range→clears→headache 2. Head starts to spin→loss of consciousness 		
2 (373, 1st series). R. side. No sensorimotor loss	<ol style="list-style-type: none"> 1. Flickering bright colours appear in L. field (especially lower blind L. field)→pictures seem to appear in centre of colours flashing past horizontally (unable to recognize them)→suddenly stops 2. 1→loss of consciousness 		
3 (365). L. side—2 wounds. No sensorimotor loss	<ol style="list-style-type: none"> 1. Cry→general convulsion 2. Live wires dancing in front of eyes for a few minutes 		
4 (895). L. side. No sensorimotor defect	<ol style="list-style-type: none"> 1. Things seem more blurred, then white weaving pattern appears in defective field 		
5 (550). R→L. Sensory loss L. limbs, with some paresis. Cortical blindness	<ol style="list-style-type: none"> 1. Spasm of L. arm and leg 2. Thousands of sparks of light whirling about→head spinning→L. arm and leg spasms→loss of consciousness 		
6 (459) R. side. L. hemiparesis with some sensory loss	<ol style="list-style-type: none"> 1. Spasm of L. arm and leg→loss of consciousness 2. Sudden feeling of great anxiety for 5 min. 3. Continual flickering lights and colours in blind part of visual field—still present after 9 years 		
7 (491). R. side. No sensorimotor loss	<ol style="list-style-type: none"> 1. Like a windmill going round for 5 min. in L. (hemianopic) field 		

continued

TABLE II *continued*

Clinical Features	Type of Fit	Skull Radiograph	Visual Fields
3 (550). R. side. Early blindness, and weakness L. leg. Later: no sensorimotor signs	<ol style="list-style-type: none"> 1. Suddenly the blind half-field is filled with hundreds of tiny sparks of coloured light—red, blue, and green, moving downwards like snowflakes—loses consciousness—grand mal 2. Coloured fog appears in blind field—several overlapping colours float about—grand mal 3. Either 1 or 2 without loss of consciousness or convulsion
3 (817). R.→L. L. hemiplegia, severe sensory loss	<ol style="list-style-type: none"> 1. Continuous flickering lights, usually white, sometimes coloured, L.→R. passing into the blind field and lasting for 20 min. 2. Twitching L. forefinger and thumb 3. Visual loss—general convulsion
GROUP II.—Radiation Lesions with no Sensorimotor Loss			
3 (667). L. side. Early dysphasia and visual distortion. No sensorimotor loss	Sees people he knows telling him to get up → grand mal (Described more fully in text)
1 (456). L. side. No sensorimotor disorder. Some spatial difficulties	<ol style="list-style-type: none"> 1. Head and eyes to R. General convulsion 2. R. field blacked out → bright white or coloured lights moving up and down in R. blacked-out field affects whole R. field, including defective segment → general convulsion after 5–10 min. Severe headache on recovery 3. As above, but after coloured lights hallucination of remembered war scenes → loss of consciousness (Described more fully in text)
2 (362). R. side. L.-handed. No sensorimotor loss. Defective localization and colour sense in upper L. field	<ol style="list-style-type: none"> 1. Sudden flashing red light in L. field 2. As in 1 → loss of consciousness
3 (654). R. side. No sensorimotor loss	Flashing light in L. field → grand mal
3 (755). R. side. L.-handed. No sensorimotor disturbance. Attention and orientation defects in partially preserved L. upper field. Topographical difficulties, also with judging speeds and distances	<ol style="list-style-type: none"> 1. Flashing lights in hemianopic field, then to R. and obscures vision (lasts for 10 min.) 2. Attacks of loss of consciousness—no aura

continued

TABLE II—continued

Clinical Features	Type of Fit	Skull Radiograph	Visual Fields
15 (132). L. → R. No sensorimotor loss. Some dysphasia, and memory poor	1. Cry → general convulsion 2. Coloured lights flashing on and off in R. field → noise in head → loss of consciousness and convulsion
16 (179). L. side. Occasional slight dysphasia	1. Vision blurs 3-4 sec. → general fit 2. Above aura only
17 (R.I. 167943). R. side. No sensorimotor loss	Sudden flashing on and off of a bright yellow light in the left (blind) field. Loss of consciousness → grand mal (Described more fully in text)
18 (529). L. side. No sensorimotor loss. Dysphasia	1. Spasm R. hand → general convulsion 2. Short periods of flashing lights in blind field
19 (562). R. side. No sensorimotor disorder	1. A hot smell, and things go blurred 2. L. arm goes dead 3. Distorted hearing → loss of consciousness
20 (211). L. side. No sensorimotor loss. Dysphasia	Vision blurs → flashes of light for ½ hour, seen in lower field R. and L.
21 (883). R. side—developed abscess. Slight sensory loss L. hand	1. Sees a cabbage in L. lower field → general fit 2. Sees a mountain with snow on top which he climbs. When he reaches the top → general fit. May be followed by weakness of L. limbs

continued

TABLE II—continued

Clinical Features	Type of Fit	Skull Radiograph	Visual Fields
22 (547). R. side. No sensori-motor loss	1. Sudden giddiness, with loss of sight, and coloured lights and circles in L. field 2. General convulsion
23 (1030). R. side. No sensorimotor signs. R. eye destroyed	1. Major convulsion—no aura 2. Multiple hallucinations of himself (head and shoulders to waist) in miniature in L. field (Described more fully in text)
GROUP II.—Radiation Lesions with Sensorimotor Loss			
24 (534). R. side. Slight L. hemiplegia with sensory loss. Spatial difficulty and body image upset	1. Shock in L. hand, tonic L. arm → loss of vision with a "familiar" hallucination which he cannot remember. Lasts for 1-2 min. L. foot weaker after 2. 1 → Loss of consciousness (Described more fully in text)
25 (266). R. side. Slight L. hemiparesis with sensory loss. Topographical difficulty	1. Phantom movement of L. hand, partly visual 2. 1 → general convulsion (Described more fully in text)
26 (23). L. side. Slight R. hemiparesis with sensory loss and dysphasia	1. Twitching R. hand → general convulsion 2. Loss of feeling R. hand and loss of vision
27 (468). R. side. Severe L. hemiplegia, with sensory loss. Blurring and inattention L. upper field lower quadrantanopia	Head to L. → flashes of yellow light in L. lower field → loss of consciousness, but lies still
28 (781). R. side. Severe L. hemiplegia with sensory loss. Delayed but excessive reaction to pinprick	Flashing light to L. → general convulsion

continued

TABLE II—continued

Clinical Features	Type of Fit	Skull Radiograph	Visual Fields
29 (899). R. side. L. spastic hemiplegia with sensory loss	1. Flashing light to L. → clonic movements L. arm and leg 2. 1 → grand mal
30 (196). R. side. Slight L. hemiparesis with sensory loss. Blurring and inattention in L. upper quadrant	1. Flashing white light in L. field 2. Objects quiver → loss of consciousness (only on standing up suddenly)
GROUP III.—Higher Visual Cortex Lesions with some Permanent Visual Field Change			
31 (931). L. side. Slight weakness R. arm. Early R. lower quadrantanopia. Slight disorientation R. lower field later	R. hemianopia → grand mal
32 (721). L. side. Slight weakness R. leg. Slight dysgraphia and confusion of laterality. Early R. homonymous hemianopia. Later, fault in localizing objects in R. lower field	1. Little round things in front of eyes which get bigger and bigger till can't see. Gradually subside in 15–20 min. 2. Suddenly can't see straight ahead, but has to look out of corner of eye
33. (4122). R. side. Moderate sensorimotor loss. Weakness of L. leg. Disorientation in L. half-fields	1. Twitching of lips → blurring of vision like a shadow over L. field → general convulsion 2. L. hemianopia for ½ hour 3. Numbness of L. arm for 5 min.
34 (739). R. side. No sensorimotor loss. Dyscalculia. Slight blurring and spatial difficulty in L. field, more lower than upper	Loss of vision in L. field for ½ hour, with mental confusion and aphasia. Sometimes visual agnosia
35 (673). R. side. Slight sensory loss L. arm. L. upper half-field blurred, more marked lower quadrant	Everything starts to move → grand mal

continued

TABLE II—continued

Clinical Features	Type of Fit	Skull Radiograph	Visual Fields
36 (328). R. side. Early L. hemiparesis, with sensory loss and hemianopia. Now only blurring in L. lower quadrant	<ol style="list-style-type: none"> 1. Numbness L. arm → L. leg → clonic movements arm and leg 2. Numbness L. arm, arm feels strange and "will not obey commands" 3. Suddenly everything looks far away and out of focus
37 (584). R. side. Handedness not known. Early L. hemianopia. Later topographical disorientation and upper L. quadrant blurring	<ol style="list-style-type: none"> 1. Lot of coloured lights and flashes 2. 1 → grand mal
38 (898). L. → R. Weakness and sensory loss in R. leg. Vision blurred in L. field, with defective localization, especially in lower quadrant	<ol style="list-style-type: none"> 1. Blue light flashes in L. field → general convulsion 2. General convulsion with no aura
39 (293). L. side. No sensorimotor loss. Early R. central scotoma. Later, R. half-fields blurred and objects tend to disappear when not moving	Zigzag lights in R. half-field for 20 min. → pale and confused
40 (71). L. side. Slight cortical sensory loss. Early R. hemianopia, later localization defective in R. field	<ol style="list-style-type: none"> 1. General convulsion 2. Phantom movement R. hand 3. Head and eyes to R. → momentary impairment of consciousness 4. (Early only) sudden shadow across R. field (Described more fully in text)
41 (599). L. side. Slight sensory disorder R. hand. Occasional agnosia R. arm. Visual imagery impaired. Slight dysphasia. Some blurring in R. lower quadrant	<ol style="list-style-type: none"> 1. Rotation to R. 2. Autoscopic hallucination with feeling of detachment (Described more fully in text)
42 (2366). R. side. Sensory loss L. side. Defect lower L. field to stationary objects, also attention defect and failure to localize in whole L. field	<ol style="list-style-type: none"> 1. Hot → thoughts race → dreams he is back in Khartoum → sees and recognizes people → clonic movement of face 2. Jerking of L. face, aphasic → rotation to L. → reverse → loss of consciousness (Described more fully in text)

continued

TABLE II—*continued*

Clinical Features	Type of Fit	Skull Radiograph	Visual Fields
43 (554). L. side. No sensori-motor loss. Slight dysphasia—frontal lobe type of behaviour. L. upper quadrant visual blurring	<ol style="list-style-type: none"> Hallucinations of remembered scenes in R. half-field Sudden aphasia and word deafness Sudden compulsion to look to R. General convulsion 		
44 (401). L. side. No sensori-motor loss. Dysphasia and impaired topographical sense. Occasional perseveration of vision soon after wound; saw things after they had gone away. Blurring and attention defect in R. half-field	<ol style="list-style-type: none"> Hallucination of remembered scene on his R. (black and white)→general convulsion Dazed→general convulsion (Described more fully in text) 		
45 (865). L. side. No sensori-motor disorder. Slight blurring of R. half-fields	<ol style="list-style-type: none"> R. hemianopia, then flashing red light from R. spreading to centre and quickening to about one a second, continuing for 30 min. Both clear quite suddenly and are followed by L. occipital headache Hallucinations of remembered scenes of experiences after wounding in the R. half-fields→dazed and aphasic→weakness R. arm (Described more fully in text) 		
GROUP III.—Higher Cortex Lesions with Visual Field Defect but No Persistent Field Change			
46 (94). L. side, parasagittal. L. handed. No sensori-motor loss	<ol style="list-style-type: none"> Blurring of sight→grand mal Grand mal 		Early R. lower quadrant defect, fields full now
47 (103). L. side. Transverse occipital wound at calcarine level. Early: transient blindness, then R. hemianopia which cleared; L. tinnitus, also dysgraphia and confusion of laterality	<ol style="list-style-type: none"> Suddenly can't see to R. side, then red spot appears to R. L. tinnitus increases→loss of consciousness General convulsion—no aura 		Blurring in R. upper quadrant field, attention defect in R. field
48 (751). R. side. Severe L. hemiplegia and loss of sensation	<ol style="list-style-type: none"> Face twitches→feels hot above eyes Feels faint→vision goes black→flashing light in L. upper field for a few seconds followed by headache for an hour Twitching L. arm and L. leg→hot feeling 		Fields full, but early L. hemianopia
49 (127). L. side. No sensori-motor loss. Slight dyslexia	R. hemianopia, lasting for up to 2 days		Early hemianopia cleared
50 (702). L. side. L. handed. Moderate R. hemiplegia, with aphasia	<ol style="list-style-type: none"> Mind hazy→things moving to R.→unable to speak→R. field blurred→R. hand dorsiflexing and elbow flexing Grand mal Sudden hemianopia for ½ hour (during one he lost himself in familiar surroundings) 		No field defect now, but early R. hemianopia

continued

TABLE II—continued

Clinical Features	Type of Fit	Skull Radiograph	Visual Fields
51 (2782). L. side. Slight weakness R. hand	1. Jerking of R. arm and leg → weak arm 2. Dizziness → blackness of vision		Early R. hemianopia, momentary blindness. No permanent field defect
52 (881). Central. Severe paralysis both legs and R. arm, with sensory loss and some dysphasia	1. Turning head to R. → loss of consciousness 2. R. hemianopia with flashing lights in R. field		Fields full, but early R. hemianopia
53 (52). R. side. Horizontal high parietal scalp wound. Slight sensorimotor loss in L. arm	1. Hemianopia, then flashes of blue light to L. for 5 min. 2. 1 for 2 sec. → major fit		Early L. field defect; fields now full
54 (76). R. side. L. hemiplegia, with sensory loss	1. Eyes turn up → "loses sight" → calls out "I can't see, put me somewhere" → major fit 2. Everything seems to move → face twitches → loss of consciousness		No field defect, but early L. hemianopia
55 (214). R. side. Severe cortical sensory loss, slight weakness on L.	Objects appear misplaced → head to L. → grand mal		Fields full, but early L. hemianopia
56 (602). R. side. Handedness not known. Cortical sensory loss L. arm and leg	1. Early visions of platoon commander in blind field 2. Spatial disorientation → pain in L. hand and leg → twitching L. face (Described more fully in text)		Early L. hemianopia—fields now full

GROUP III.—Higher Cortex Lesions with Fields Always Full

57 (280). L. side. No sensorimotor defect	1. Sight goes → aphasic → fall → general fit 2. General fit; no aura 3. Eyes blur and empty feeling inside		Fields full
58 (102). L. side. No sensorimotor loss. Dysphasia	1. Scream and half-turn → fall with convulsion 2. Aphasia → objects seem to disappear and bright light in front of eyes		Fields full
59 (894). R. side. No C.N.S. signs	1. Objects suddenly appear to recede → loss of consciousness 2. Rarely, grand mal attack		Fields full
60 (391). R. side. Slight weakness of L. leg	1. Everything looks distorted → fall (? unconscious) 2. Sudden fall and unable to move for 3–5 min.		Fields full

All cases are right-handed unless otherwise stated.

In the first case phantom movement of one limb occurred and the attack involved mainly kinaesthetic sense though there was a visual element to it in that he would at times look up and seem to see the limb in its phantom position. In the second the feeling was more ill-defined; the limb looked queer and was in the wrong position, though the visual element to the dysfunction was more definite. These may be compared with localized or lateralized visual field phenomena such as hemianopia, etc. The following two represent a more intrinsic upset of the body scheme.

Case 41.—The patient's attacks were of two sorts. In one there was a sudden intense feeling of rotation always to the right. If walking, he would be forced to veer to the right in a circle. This would last for a few seconds only and end abruptly. In the second and less frequent one he would have a sudden feeling of being detached from his body, so that he was observing it as if looking at someone else. There was a definite visual quality about the attacks, which he described as, "I see myself as separate and watch myself from outside and slightly above me". On one occasion this had led on to loss of consciousness and falling.

Case 23.—This patient had a variety of epileptic phenomena. Early after wounding he had sudden grand mal attacks without any aura. Later, attacks occurred in which objects he was looking at would suddenly fade out and be replaced by a black or grey spot which would spread to cause a complete left hemianopia. This would sometimes lead on to a grand mal attack. In others, the left half-field was suddenly filled with coloured balls of light, and as he looked at them they changed to multiple tiny figures of men. On later occasions he saw these figures and recognized them as multiple images of himself, tiny replicas which seemed to move towards him and then recede again: they were head and shoulders only, and if he moved his arms the figures seemed to move theirs in a similar way. For a limited period of several days together he also had attacks of a sudden black spot obscuring his vision, but, instead of remaining a negative phenomenon, the area was suddenly occupied by a normal life-size picture of a man's head and shoulders: as he looked, "it suddenly dawned on me it was myself". It was a mirror image, and again could be made to imitate movements he himself made. On occasion all of these hallucinations had been the aura to grand mal attacks. (This case has already been mentioned in a study of autoscopia by Dewhurst and Pearson, 1955.)

In both these cases the mechanism for central integration of the body image itself seems to be involved, but in two different ways. In the first, Case 41, he is watching the visual impression of his own body image from without, as if the visual (exteroceptive) and postural and kinaesthetic (interoceptive) elements of the scheme had become separated. In the second (Case 23) we see a gradual

build-up of a fully formed "autoscopic hallucination" from what at first seems to have been a simple, almost unformed, visual hallucination. In the one case the discharge seems to occur directly within the areas concerned with formulating the visual body image. In the second, a discharge originally from some relatively primitive visual area is secondarily elaborated by other parts of the cortex at first into figures and finally into figures of the self. Such a process may well be important in the formation of one type of autoscopia, and it would be of interest to know whether it represents a gradual spread of some epileptic discharge in recurring attacks so that more widespread areas of cortex are involved, or whether the discharge remains confined to its original circuits, but the normal cortex around it gradually reacts to the discharge in such a way as to produce the feeling of autoscopia.

Finally, in the formed visual hallucinations of remembered scenes, the problem of both whole-field organization and the possible "elaboration" of the primary hallucination occurs.

In nine cases, five stated that their hallucinations were in only one half of the visual field, always that half which had some loss of function either early after wounding or permanently. All were able to describe the hallucinations in detail. Of these cases, Nos. 43 and 45 have already been reported as examples of epileptic hallucinations of remembered scenes (Robinson and Watt, 1947) and No. 44 has been referred to in connexion with loss of "number form" following brain wounds (Spalding and Zangwill, 1950).

Case 45.—This patient, in addition to attacks of intermittent, red, flashing lights in the right half-field, would suddenly see in this field pictures of his experiences immediately after wounding. He would see stretcher bearers walking past and then the figures of nurses whom he would recognize. The figures were smaller than normal and seemed flat and uncoloured rather like a cinema film. They appeared in addition to real objects seen in the left half-field.

Case 44.—In his first fit only, there was a clear-cut visual aura at the onset. Suddenly in the right half-field a scene appeared in black and white. It was an outdoor scene with fields and trees and it seemed like a part of the landscape near his home, though he could not place it precisely. There was some vague movement of objects—a swaying of trees, etc.—in the hallucination, but just as this began he lost consciousness and fell. A generalized fit followed, in which there was some stiffening with tongue biting, but no clonic movements were recorded.

Case 56.—His visual attacks consisted of sudden spatial disorientation followed by pain in the hand and

leg and then twitching of the left face with some clouding of consciousness. In attacks early after wounding when he still had a partial left hemianopia, the blind field would suddenly be occupied by a vision of his platoon commander walking towards him. This might cease abruptly or go on to loss of consciousness, probably with some generalized convulsion.

In another case (Case 43) the visions were superimposed on the preserved part of the field, which was also still seen clearly, and produced a most bizarre experience. While in the cinema he suddenly saw in the right half-field various objects in the hospital ward. These appeared as a continuation to the right of the screen, on which he could still see the film he was watching to the left. The images were similar in colouring to the pictures on the film. On another occasion, while in bed in the ward, he suddenly saw a motor bicycle and then a convoy of lorries passing from right to left in his right half-field. They appeared to be passing over the end of the bed, which he could still clearly see in the left half-field. On this occasion, the attacks proceeded to a grand mal convulsion.

In another case, the hallucination was more widespread and involved a visual sequence in which the patient was taking part.

Case 21.—In some attacks the left lower (quadrantanopic) field was suddenly occupied by what seemed to be a large white cabbage: the outlines and crenations of the cabbage leaves could be clearly seen. This lasted for a few seconds then consciousness was lost and a grand mal attack ensued. In others, the left field was filled by a snow-covered mountain, which seemed familiar, and as he gazed at it he seemed to be climbing up it, with appropriate changes in its appearance as he climbed. This lasted a few seconds only, and when he seemed to reach the top he lost consciousness and a generalized convulsion occurred.

In all these, although the visual phenomena were strictly unilateral, the discharge appeared to activate a clear-cut memory mechanism. In three, the dominant hemisphere, but in two the minor hemisphere, was involved. These cases, especially perhaps the last two, raise questions of great interest about the organization of "higher" cerebral function. It would seem as if such an integrated and holistic mechanism as memory can at times be fragmentarily activated while still retaining all the qualities of "memory".

In Case 21 the possibility of subjective elaboration of a simple primary hallucination into a more complex scene arises. Here, the hallucination was sometimes of a cabbage, but white not green, and sometimes of a snow-covered mountain top. Both in fact were white, rather amorphous objects. When the latter occurred, the patient felt himself to be climbing, with the contours of the mountain changing as he did so. However, it is not certain whether changes and movement in the

original amorphous visual pattern were not interpreted as the visual impressions of climbing up the mountain.

In the other four cases, the hallucinations appeared to fill the whole visual field, though they might start in the defective half. In three, the hallucinations could be recalled and described in detail.

Case 11.—This patient had right adverse attacks leading to generalized grand mal: also sudden right hemianopia followed by flashes of light in this field. On other occasions coloured pictures of various battle experiences in Italy would seem to move across his vision like lantern slides. They filled the whole of his vision and were the prelude to loss of consciousness and grand mal convulsions.

Case 10.—He suddenly saw people he knew and could name at the time, standing round him and telling him to get up. He seemed to be lying down at the time. These figures, three or four at a time, filled the whole visual field and the real visual world was obliterated. The hallucination lasted for a few seconds only and then he lost consciousness and had a grand mal attack.

Case 42.—In addition to attacks of clonic facial movement leading to left adersion and loss of consciousness, this patient had attacks in which he suddenly felt hot, his thoughts seemed to get out of control, and he felt that as if in a dream he was back in Khartoum during the war. He saw and recognized friends around him. The experience was accompanied by strong apprehension and frustration which exactly reproduced the emotions he had in fact felt in 1940 in Khartoum when he thought that he ought to be in England taking a more active part in the war. During this time he could not see real objects around him. The attack led to clonic movement of the face, but very rarely to a generalized convulsion.

In one case, however, although the hallucination was familiar and clearly defined when it occurred, it could not be recalled or described afterwards.

Case 24.—This patient, in addition to left-sided clonic motor attacks, also had two types of visual attack. In one, the remaining right half-field of vision suddenly became blurred so that nothing could be seen clearly. In the other there suddenly appeared in the left (blind) half-field, but spreading rapidly to the right also, the picture of some familiar scene. The picture was in black and white and at the time of its occurrence was clear cut and recognizable, but as soon as it faded only the feeling of familiarity could be recalled. The description here was similar to the "sense of familiarity" of uncinate attacks.

In one case also, the involvement of a memory mechanism seems more fundamental. In this case (Case 42) the whole experience was more mental and less purely visual; he described it himself as "like a dream". This contrasts especially with the case (Case 21) where the memory element seems

rather incidental to the primary, rather ill-defined, visual hallucination. It may therefore be seen that epileptic discharge from the parieto-occipital areas provides a wide variety of visual phenomena ranging from crude hallucinations of flashes and coloured lights, through more complex phenomena such as extinction of part or the whole of vision and distortion or disorientation of visual perception, to a highly integrated hallucination of a familiar remembered scene. Some of the problems raised by the kind of attacks occurring in this and the two preceding groups are discussed further below.

Discussion

The content of visual fits shows great variation in minor detail: so much so that in some respects no two fits are alike. Lights may be white, multi-coloured or monochromatic—green, yellow, orange, red, and purple are all described. They may be continuous or interrupted, stationary or moving, varying in intensity, scintillating or sparkling, and of varying shapes. Visual objects may appear smaller or larger than normal, they may seem displaced in space, or simply distorted, strange or unreal, or without meaning as in agnosia. They may appear blurred and indistinct; or vision may be actually extinguished in part or whole. Formed hallucinations may be in black and white, or coloured. They may be of simple objects or of complex scenes, either static pictures or with movement and action in them. Indeed, there is scarcely any aspect of normal vision which is not represented in one or other of these fits. It is as well to emphasize this variation and complexity, which is in general also a feature of the responses obtained from electrical stimulation of the cortex (Penfield and Jasper, 1954) and to regard it as a reflexion of the flexibility of function of the normal cortex in which discharge from a given cortical point has many possible paths of conduction, depending on the exact state of excitation or inhibition at the various synapses involved.

Nevertheless, it is possible to recognize certain broad similarities in differing fits, and to group them accordingly. Amongst crude hallucinations, these groups may be segregated: (1) Continuous moving lights which may be single- or multi-coloured and of various shapes and patterns, the essential feature being that they are continuous and moving; (2) continuous, steady, motionless light; (3) interrupted flashes of light; (4) negative phenomena—blurring or extinction of vision—form another definite group; (5) distortions of vision—micropsia, macropsia, apparent displacement of objects, etc.—also emerge; (6) finally, a number of complex

positive phenomena may be seen, consisting of formed hallucinations of familiar common objects or remembered scenes.

Of the whole 60 cases, 31 had crude phenomena of some sort—17 interrupted flashes, 11 continuous moving lights, and three continuous stationary lights—as part of a fit. Twenty-four showed negative phenomena, 11 distortions, and 12 complex hallucinations.

In Table III an attempt is made to relate these six

TABLE III
PATTERN OF FITS IN THREE MAIN GROUPS OF CASES

Pattern of Fits	Calcarine (9 cases)	Radiation (21 cases)	Higher Visual (30 cases)
Continuous stationary lights	0	1 (1)	2 (2)
Continuous moving lights	9 (1)	1 (1)	1
Interrupted flashes ..	0	11 (1)	6 (4)
Negative phenomena ..	2	6	16 (1)
Distortions	0	1	10 (1)
Complex hallucinations	1 (1)	5 (1)	6

Figures in parentheses denote the number of cases within the total in which phenomena occur *after* another visual phenomenon in a given attack. Total types of fit are greater than total number of cases.

types of fit to the three anatomical groups given above. It appears that crude phenomena of continuous moving lights are especially associated with calcarine lesions. Interrupted flashes seem more common when permanent defects of radiation have occurred, while negative phenomena, distortions, and complex hallucinations seem related to "higher" visual cortex lesions.

It is usual to associate unformed visual hallucinations with lesions of the occipital pole and calcarine region, and although our findings support such a view, they suggest that some forms of crude visual phenomena are also common with lesions well anterior to the occipital pole and impinging at times on the posterior temporal cortex.

Negative phenomena deserve special consideration. The inhibition or extinction of preceding vision or visual imagery must play an important part in normal visual activity in "clearing the decks" for the next object of visual attention. The cases noted here certainly suggest the possibility of a special anatomical site for such negative activity and raise once more the question of a special visual inhibitory area (area 19 S, McCulloch, 1949) already mentioned by one of us (Russell, 1947) in connexion with the general distribution of epileptogenic wounds. These negative fits may affect a complete half-field but in some cases this spreads rapidly to involve all vision and in others complete visual loss

seems to occur *ab initio*. Such effects, presumably originating in one hemisphere, underline once more the functional integration of the two visual cortices which is an essential part of normal vision, and suggest the rapid transmission of impulses between the two occipital poles. Complex hallucinations also show a similar variety of distribution and spread within the visual fields.

These findings may be compared with those from electrical stimulation of the cortex producing visual effects (Penfield and Rasmussen, 1950; Penfield and Jasper, 1954). Direct stimulation of the calcarine cortex is technically difficult because of its relative inaccessibility, and is recorded in only a few instances, but, in general, responses from this and the nearby cortex of the occipital pole were crude, unformed phenomena of lights of various colours and shapes, but usually with a quality of movement—undulating, quivering, dancing, or glistening were adjectives commonly used. Intermitting flashing lights certainly occurred occasionally from the immediate vicinity of the pole, but these seemed more frequent when stimulation was applied further forward to the lower parts of the lateral occipital cortex. Information about negative phenomena is more scanty. Responses of shadows across vision and areas of “blackness” were occasionally recorded, but the points stimulated were in the lateral aspects of the occipital lobe and did not seem differentiated from those producing crude positive responses. Complex visual hallucinations, involving at times remembered scenes, were obtained from stimulation of points on the lateral aspect of the temporal lobes, both anteriorly and near its posterior extremity, but not from the occipital or parietal areas. These hallucinations were, however, often accompanied by auditory phenomena also, a feature which was not recorded by our patients. Distortions of visual perception, micropsia, macropsia, etc., were also noted, again generally from the temporal cortex.

So far as crude visual phenomena are concerned, therefore, our findings seem in general agreement with the results of stimulation. When formed hallucinations and remembered scenes are involved, however, some of our cases certainly suggest firing from posterior parietal and occipital cortex. Indeed, it is of special interest that these cases involving remembered scenes seem to fall into two distinct groups from the anatomical point of view, for some of the cases have wounds in the temporal lobe, and others have lesions in the posterior parietal or parieto-occipital region.

Cases 43, 44, 23, and 24 are all remarkable instances of temporal lobe visual hallucinations

which are often in the contralateral field and often involve the reproduction of remembered scenes or are associated with a feeling of familiarity. These cases may have division of the lower half of the optic radiation (Meyer's loop), or complete hemianopia. In Case 44 the missile has entered just above the radiation on the right and the metal fragment has passed into the posterior temporo-parietal region on the left. There is no dense field defect. It is therefore possible that in this case the hallucinatory phenomena may originate from the left parietal region rather than more anteriorly in the temporal lobe. In none of this group were there any associated attacks with crude visual flashes.

In the other cases (Cases 11, 45, 41, 21, and 42) the wound was usually limited quite clearly to the parietal or parieto-occipital region (Case 21 is difficult to classify), and in three the visual field defect was slight and indicated that the amount of brain damage was slight. In Cases 11 and 45, which fall most certainly into this group, there were also at times crude visual discharges of the type reported in Group II cases.

In most of these cases the hallucinated “memory” could be recalled in exact detail, but in one (Case 24) with a temporal wound and hemianopia, the effect was more of a “*déjà vu*”, and, although the hallucinated scene was entirely familiar, its details could never be described.

Here, therefore, is convincing evidence that hallucinations of remembered scenes may originate from two widely separated anatomical areas: first, the temporal lobe, which is well known for its association with a sense of reminiscence and “*déjà vu*”, and secondly the posterior parietal or even occipital region which is known to be connected with visual imagery and visual memory.

It is easy to understand that discharge from the parieto-occipital region can lead to formed visual hallucinations, though there is a tradition in clinical neurology that temporal lobe lesions are more often associated with this. However, temporal lobe lesions may involve hallucinations of other special senses with a characteristic feeling of familiarity. There seems, therefore, in this region to be a special function associated with remembering in general which applies to many senses—visual, auditory, olfactory, etc. This may well be a function of the hippocampus and its connexions, and if this is so then when we observe the same complex hallucinations appearing in both temporal and parietal lobe lesions we are in fact seeing the results of stimulating two parts of the same mechanism, or perhaps we are demonstrating that a visual memory depends

not only on a certain pattern in the parieto-occipital region, but that, like other memory processes, its effective functioning depends in some way on hippocampal activity.

A further observation which may have anatomical implications is the strikingly low incidence of visual fits in occipital pole wounds. The calcarine cortex differs from the parastriate and other cortical areas in having no direct thalamo-cortical connexions. Such an area would be expected to have only a small epileptogenic capacity if thalamo-cortical discharges are important for initiating and maintaining or enhancing cortical discharge. The conception of a "centrencephalic" system whose activity plays an important part in elaborating epileptic discharges in the cortex is one which has some support from animal experiments (Jasper and Droogleever-Fortuyn, 1947). The very limited epileptic properties of the injured striate cortex might be considered as further evidence in its favour.

It is interesting that in four cases in this series unformed hallucinations were followed by severe headache, sometimes throbbing and unilateral, the whole sequence resembling what might be called a traumatic migraine. However, we were not able to recognize any special physiological or anatomical features in these unusual cases.

This study provides some evidence both for the quality of activity and method of function of the occipital cortex and related areas. But such

findings must be used with discretion in formulating theories of cerebral activity. For when a group of cortical cells fires abnormally the subjective symptoms experienced need not necessarily arise at the site of firing but may be produced in some intact part of the cortex at which the discharge arrives. Moreover, a brain wound may not only give rise to an epileptic focus, it may also interrupt some of the normal channels of discharge from the "firing" area and this may further alter the apparent "function" of the area. The correlation of such evidence as we have brought forward with that derived both from cortical stimulation and non-traumatic extirpation where possible is thus important for a proper evaluation.

REFERENCES

- Bay, E. (1953). *Brain*, 76, 515.
 Brazier, M. A. B. (1951). *The Electrical Activity of the Nervous System*. Pitman, London.
 Dewhurst, K. E., and Pearson, J. R. (1955). *Journal of Neurology, Neurosurgery and Psychiatry*, 18, 53.
 Jasper, H. H., and Droogleever-Fortuyn, J. (1947). *Res. Publ. Ass. nerv. ment. Dis.*, 26, 272.
 McCulloch, W. S. (1949). In *Precentral Motor Cortex*, 2nd ed. Ed. Paul C. Bucy. Chapter VIII, Cortico-cortical connections. University of Illinois Press, Urbana, Illinois.
 Penfield, W., and Jasper, H. H. (1954). *Epilepsy and the Functional Anatomy of the Human Brain*. Churchill, London; Little, Brown and Co., Boston.
 —, and Rasmussen, T. (1950). *The Cerebral Cortex of Man*. Macmillan, New York.
 Robinson, P. K., and Watt, A. C. (1947). *Brain*, 70, 440.
 Russell, W. R. (1947). *Ibid.*, 70, 225.
 —, and Whitty, C. W. M. (1953). *Journal of Neurology, Neurosurgery and Psychiatry*, 16, 73.
 Spalding, J. M. K. (1952). *Ibid.*, 15, 169.
 —, and Zangwill, O. L. (1950). *Ibid.*, 13, 24.