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STUDIES IN TRAUMATIC EPILEPSY

2.* FOCAL MOTOR AND SOMATIC SENSORY FITS: A STUDY OF 85 CASES

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Contents

When both focal and generalized epileptic attacks occur in the same patient it is not uncommon for the content of the focal attack to occur also as an aura to the generalized seizure. This probably indicates that in each case the site of initial cortical discharge is the same, but that it remains localized in one and spreads widely in the other to involve sub-cortical centres which seem to play an essential part in the spread of the epileptic process. For this reason we here consider together isolated focal fits and the localized aura to major seizures, an "aura" being taken to mean any abnormal motor or sensory change which precedes the loss or clouding of consciousness of a fit and which the patient remembers clearly and can subsequently describe. A focal fit may affect almost any of the bodily functions or sensations, and indeed may affect more than one of these during a single attack. The present study is concerned with those fits which involve the motor and somatic sensory functions. These form by far the largest group of focal epilepsies. Of the 360 cases of post-traumatic epilepsy in this series, over 110 have had one or more epileptic attacks of the type which falls into this group; 85 of these are considered in this paper. The variety of pattern which these cases exhibit is so great that it seems desirable to describe briefly the features of a considerable number of cases. Classification is difficult, and though many types of fit are described, this collection of cases does not by any means cover all the possible varieties which may occur.

The majority of focal motor attacks have both a tonic and clonic element. In the upper limb, for instance, the common pattern is of clonic jerking of the fingers with tonic flexion of the hand and forearm, the whole limb often being drawn up towards the face. Clonic movements may also occur in the contractions of forearm and arm muscles, which are initially tonic. Pure tonic or clonic fits are relatively uncommon, but the beginning of movement in an attack may be either tonic or clonic, and it is often possible to establish the initial type of movement for a given case. Examples of "pure" tonic attacks are seen in the so-called adversive fit when this remains localized (see case no. 31*), while *epilepsia partialis continua* often consists of a "pure" clonic movement. In some ways the pattern of the common tonic-clonic focal

* Part 1 appeared on p. 93, Vol. 15, 1952.

*Except where otherwise stated case numbers refer to those in Table IX. Cases described also in the text are marked "T" in Table IX.

fit suggests that tonic contraction is the presenting abnormality which is interrupted repeatedly by sudden momentary inhibition to cause the clonic element, and as is shown later, an attack often changes from tonic to clonic, but rarely from clonic to tonic during the aura phase. This change is well seen in those cases where tonic spasm initiates the attack but is rapidly followed by clonic beats at first of considerable frequency, but gradually slowing, as each inhibitory interval with relaxation of muscles becomes longer, till the limb falls in a flaccid state and the fit is over. On the other hand in some cases of *epilepsia partialis continua* a rhythmic twitching may occur sometimes of a single muscle or part of a muscle which is continued for hours or days, and in which there appears to be no background of tonic spasm. The impression here is of rhythmic discharge rather than of intermittent inhibition, but of course the short duration of each twitch might possibly depend on sudden inhibition.

A study such as this may not only describe the various forms of aura and focal fit and the relative frequencies with which they occur: it may also attempt to correlate the type of aura with the site of wounding and the neurological deficit shown on clinical examination. Of special interest in this series is the information available with regard to the transition of one type of aura to another, or to a major convulsion, or to some mechanism which arrests the fit. The physiological basis for epileptic phenomena is still largely unknown, but, as will be mentioned later, the circumstances under which a focal fit ceases should not be neglected, as this may be just as much a positive physiological process as the spread from one part of the cortex to another. The latent interval between injury and the onset of fits is also of interest as is evident from Table VII.

TABLE VII

INTERVAL AFTER WOUNDING OF FIRST FIT IN CASES WITH FOCAL CLONIC FITS WITH AND WITHOUT PERMANENT SEVERE HEMIPLEGIA

Time after Wounding of Onset of Focal Clonic Fits	Cases with Permanent Major Hemiplegia	Cases without Major Hemiplegia	Total
0– 1 month	1 (1)*	13 (5)*	14 (6)*
1– 6 ,,	8	7	15
6–12 ,,	5	4	9
1–2 years	3	2	5
Over 2 years	—	1	1
Not known	3	5	8
Total	20	32	52

* The figures in parentheses give the number of cases which had focal fits within a month of wounding but not later (5 year follow-up).

It must of course be emphasized that the information analysed in this study, based chiefly on the careful questioning of patients and their relatives, cannot always be accurate. It is to be hoped, however, that errors due to such inaccuracies will be more than offset by the number of reliable case records available.

In describing our observations we consider first the general features of the common motor and sensory attacks, then note the ways in which the initial discharge spreads in relation both to the body and the cortex, and the ways in which it may cease, then return to a more detailed review of the content of some of the focal attacks. Finally we consider briefly the evidence for the cortical site of discharge of the various forms of attack.

GENERAL FEATURES OF INITIAL MOTOR AND SENSORY ATTACKS AND THEIR SUBSEQUENT COURSE

The material from which we have drawn our conclusions is presented briefly in Table IX, where particulars are given of the patterns of fit reported in each case and also a rough indication of the permanent paralysis, sensory loss, or field defect caused by the wound. Table IX also gives tracings of the skull radiographs for nearly all cases.

Clonic Attacks

On the motor side, the clonic attacks (Table VIII) are sometimes highly localized in origin, as in those

TABLE VIII

PARTS OF THE BODY TO WHICH VARIOUS FORMS OF PRESENTING AURA ARE REFERRED

	Motor Clonic	Motor Tonic	Adversive Tonic	Motor Inhibitory	Sensory Positive	Sensory Negative Numbness and Agnosia	Totals*
Face	11	3	—	—	4	2	20
Head and eyes	4	1	12	—	2	1	20
Thumb ..	2	—	—	—	—	—	2
Fingers ..	3	—	—	—	4	1	8
Hand	10	3	—	4	9	5	31
Arm	11	5	—	1	8	3	28
Abdomen ..	1	1	—	—	—	1	3
Leg	5	1	—	2	2	1	11
Foot	4	—	—	—	2	—	6
Whole side ..	2	—	—	—	1	3	6
Total ..	53	14	12	7	32	17	135

* All *attacks* with differing onset are here recorded, so that one case may be represented more than once.

starting in thumb, index finger, or face, and the movement usually spreads to some extent, but it may remain relatively localized for long periods. The following cases illustrate these varieties of clonic aura :—

CASE 16. **Clonic Aura with Very Focal Onset and Sensory Aura to Grand Mal**

A right anterior parietal brain wound caused at the moment of wounding a well formed visual hallucination " like a playing card all lit up ". Soon after, there was a slight spastic weakness of the left hand. Passive movement sense was reduced and stereognosis was largely absent in the left hand. Pin-prick was poorly localized and appreciation was delayed in the hand and arm. There were no motor or sensory changes in the leg. Some years later, some reduction of passive movement and two-point touch in the left thumb and index were the only abnormalities.

Fits.—**1.** Sudden clonic jerking of the left thumb, then the whole left arm showed tonic stiffening, with fingers extended at the wrist and the elbow flexed : then clonic jerkings of the whole forearm occurred, consciousness was lost, and generalized tonic stiffening with opisthotonos was followed by generalized clonic movements.

2. Sudden numbness of the left hand and forearm was immediately followed by tonic flexion of the fingers and wrist, then clonic movements of these but nothing further, and no loss of consciousness.

CASE 4. **Very Localized Clonic Fits and Generalized Convulsions**

A right Rolandic brain wound soon after wounding caused left hemiparesis and agnosia for the left arm together with complete loss of all types of sensation. Sensation was also reduced in the left leg, but here pain was appreciated as an intense and radiating sensation. Some years later there was slight weakness with increased tone in the left arm. The appreciation of two-point touch and passive movement was reduced, stereognosis was impaired and pin-prick appreciation changed in the left upper limb.

Fits.—**1.** Occasional clonic twitching occurred of the left side of the mouth.

2. The left hand, then the left face twitched, and then felt hot.

3. A sudden feeling of faintness was followed by loss of consciousness, and onlookers describe reddening of the face with generalized stiffening of the whole body followed by clonic movements.

CASE 79. **Partial Continuous Epilepsy of Hand**

A right posterior parietal brain wound soon after wounding caused a severe flaccid left hemiplegia. All forms of sensation were lost in the upper limb for which there was an agnosia, but pain was occasionally appreciated in the leg. There was left homonymous hemianopia. Some years after there was a left spastic hemiparesis more marked in the arm than in the leg, with athetotic movements of the hand. Two-point discrimination was lost throughout the left side, including the face. Localization of touch was very poor in the arm and leg, less so in the face. Stereognosis was abolished in the left hand. Pin-prick sensation was diminished, and hot and cold were not distinguished on the left face, arm, and leg. Dense left hemianopia was present.

Fits.—Fits occurred only early after wounding. There was a constant rhythmic clonic movement of the left hand and forearm. This was a flexion-extension movement with slight supination accompanying the flexion. Tone was increased in all muscles, but there was no tonic contraction of flexion or extension behind the clonic movement. This persisted for 10 to 12 days. The movements then became confined to the left index finger and continued thus for a further three days. It was an active rhythmic contraction of the extensor indices with a slow return to the slightly flexed position of rest. At each movement a flicker of extensor muscle contraction could be seen and felt at the rate of about one per second.

Tonic and Adversive Attacks

Tonic or adversive auras tend to involve major groups of muscles in such a way as to cause strong postural reactions such as turning of the head and eyes or flexion at the elbow with abduction at the shoulder, though rarely it may be more confined while retaining its tonic character. The head, eyes, and upper limb are commonly involved first in tonic fits.

CASE 35. **Adversive Movements as Aura to Loss of Consciousness**

A left frontal to parietal brain wound soon after wounding caused a marked motor dysphasia and right hemiparesis. There was sensory loss of all types in the right hand, but passive movement sense was more severely impaired than appreciation of pin-prick. These changes were less marked in the leg. Some years later there was slight dysphasia and slight weakness of the arm and leg with some increase in tone. There was impaired two-point touch and passive movement sense and also some change in appreciation of pin-prick more in the arm than in the leg. The right hand showed astereognosis.

Fits.—The head was forcibly turned to the right, then the right arm, wrist, and elbow were slowly flexed and bent up towards the head. There was some jerking of the leg, first in the foot, then in the knee, without any tonic element. Then consciousness was lost, but no further movements were recorded. After the attacks there was paralysis of the right arm and leg for some 20 minutes.

CASE 65. **Focal Tonic Fits Affecting One Muscle Only and Generalized Convulsion with Aura of Tonic Stiffening**

A severe and deep left fronto-parietal brain wound soon after wounding caused right hemiplegia and right hemianopia and aphasia. Some years later right hemiparesis persisted. Sensory testing was difficult owing to dysphasia and lack of cooperation, but pin-prick was

TABLE IX.—SUMMARY OF PERMANENT SENSORI-MOTO

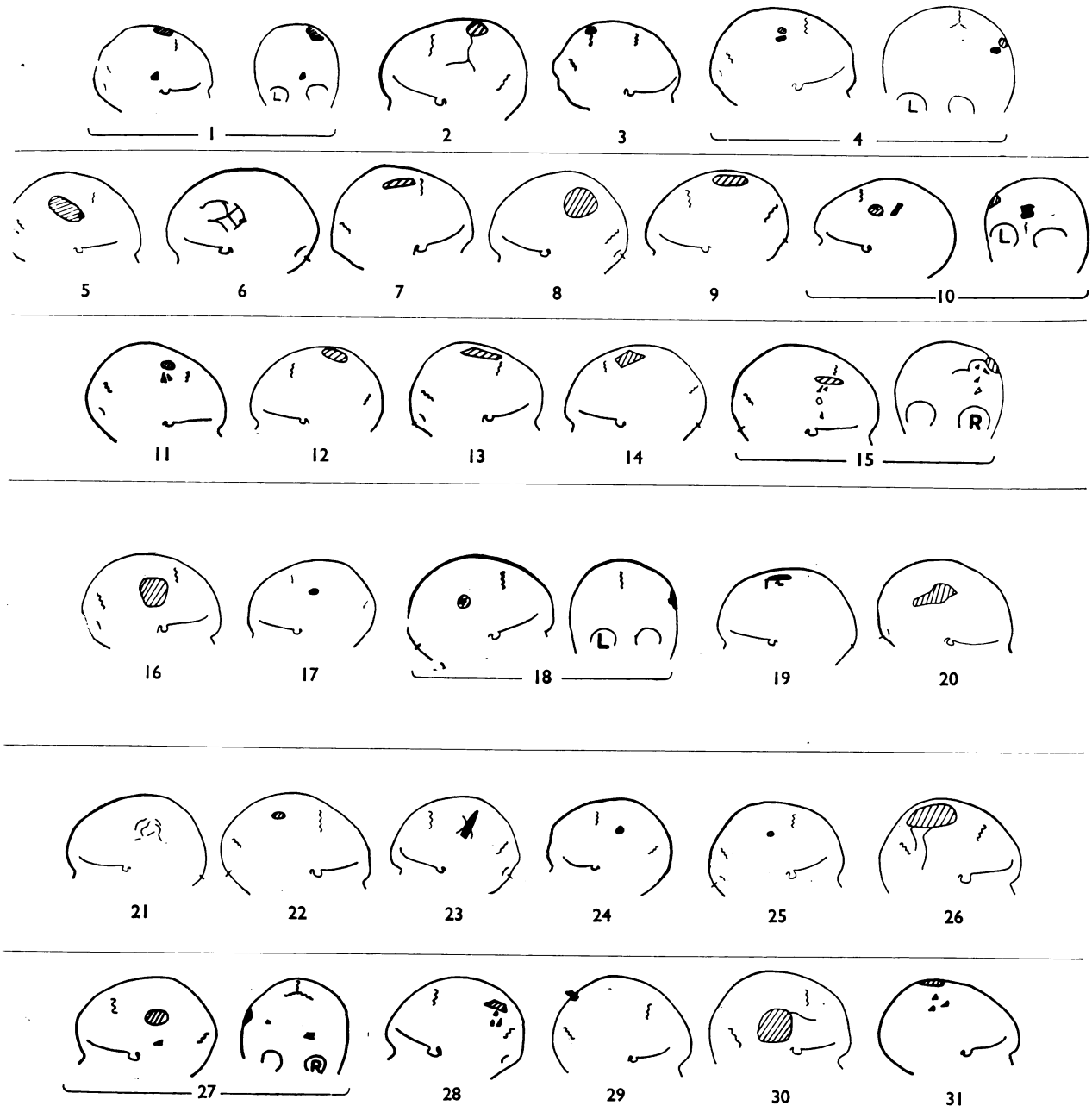
Case and Index Nos.	Side	Motor Loss	Cortical Sensory Loss	Other	Type of Fit
			GROUP 1.—*Wounds in the Rolandic region causing limited motor (and often also sensory) loss*		
1 (162)	R.	L. arm, moderate	L. arm, slight	—	L. hand tonic→ L. face clonic→ loss of consciousness†→ general toni (clonic L. side only)
2 (2789)	L.	R. leg, severe	Nil	—	(1) Tingling R. arm→ R. face→ aphasia. (2) Sudden loss of balance
3 (328)	R.	L. arm, L. leg, slight	L. fingers, slight	L. spatial disorientation	(1) Tingling L. hand, L. arm. (2) Numbness L. fingers→ L. arm→ L. leg clonic L. hand→ L. foot. (3) Dizzy→ loss of consciousness, pale, n movements. (4) Tonic and numbness L. abdomen→ clonic L. abdome
4 (404)	R.	L. arm, slight	L. arm, slight	—	(1) L. face clonic. (2) Clonic L. hand→ L. face→ heat L. hand, L. face (3) Faintness→ general convulsion T.
5 (100)	R.	L. arm, moderate	L. arm, moderate	—	(1) Aphasia→ tonic adversive head, eyes to L.→ loss of consciousness
6 (915)	L.	R. arm, R. leg, moderate	R. arm, R. leg, moderate	Aphasia	(1) Pins and needles R. leg→ R. thigh→ clonic R. foot, R. arm→ toni jaw T
7 (860)	R.	L. arm, L. leg, moderate	L. hand, moderate	Aphasia	(1) Tonic L. leg, painful cramp→ clonic L. leg. (2) Pain L. face→ cloni L. face→ L. leg→ L. electric shock sensation (not arm). (3) (2) followe by loss of consciousness. No general convulsion
8 (512)	L.	R. arm, moderate	R. arm, moderate	Aphasia	(1) R. arm very cold→ R. leg (2) Cry→ general convulsion. (3) R. arı jerks with noise T
9 (49)	L.	R. leg, R. arm, L. leg, moderate	R. leg, L. leg, R. arm, moderate	R. visual inattention	(1) Paralysed or weak R. arm→ R. leg→ falls to right→ loss of cor sciousness (brief general convulsion) T
10 (742)	L.	R. hand, moderate	R. hand, moderate	Slight aphasia	(1) Agnosia R. arm, R. leg for 20 mins.
11 (176)	R.	L. hand, moderate	L. hand, moderate	—	(1) Clonic L. hand→ tonic L. hand, L. fingers. (2) General convulsio in sleep
12 (209)	L.	R. arm, R. leg, moderate	R. arm, R. leg, moderate	—	(1) Clonic R. thumb→ R. fingers (index first)→ tonic R. hand→ paralyse R. hand
13 (816)	R.	L. arm, L. leg, slight	L. arm, L. leg, slight	—	(1) Clonic L. foot→ tonic L. arm→ loss of consciousness. (2) Cloni L. foot, L. arm
14 (935)	L.	R. arm, R. leg, severe	Nil	—	(1) Tonic-clonic R. arm, R. hand. (2) Clonic R. hand→ R. arm→ R. fac →loss of consciousness
15 (776)	R.	L. arm, moderate	L. arm, moderate	—	(1) Clonic L. arm→ L. leg→ loss of consciousness. (2) Numbnes L. side→ clonic→ loss of consciousness. (3) L. arm numbness, tonic– L. side (" horrible feeling ")
			GROUP 2.—*Wounds in Rolandic region causing more sensory than motor loss*		
16 (458)	R.	Nil	L. thumb and index	—	(1) Clonic L. thumb→ tonic hand→ clonic L. arm→ loss of con sciousness. (2) Numbness L. hand→ tonic L. hand→ clonic L hand T
17 (273)	L.	Nil	R. hand, R. face, severe	Aphasia	(1) Clonic R. face→ aphasia→ R. hand weak for ½ hr. (2) Adversiv tonic head→ tonic R. arm→ loss of consciousness. (3) Tinglin R. foot→ as in (2)
18 (2656)	R.	Nil	L. hand, moderate (ulnar)	—	(1) Stinging L. hand→ L. shoulder→ L. leg→ L. abdomen→ cloni L. face→ choking→ loss of consciousness (L. side convulsed) (2) Electric shock sensation L. neck. (3) Electric shock sensatior L. hand, L. foot
19 (815)	L.	Nil	R. hand, moderate (ulnar)	—	(1) Pins and needles R. upper lip→ R. hand→ R. elbow→ R. foot→ R. flank→ vision defective. (2) Visual blackout (? postural) T
20 (975)	R.	L. arm, L. face, slight	L. hand, L. face, moderate	Lower quadrantanopia	(1) (early only) Electric shock sensation L. hand to L. shoulder→ cloni R. shoulder. (2) Severe pain L. arm→ face→ eye→ loss of con sciousness (general convulsion). (2) May be preceded for a day or tw by numbness L. arm T
21 (884)	L.	R. arm, slight	R. hand, severe	Aphasia	(1) Clonic R. hand→ ringing R. ear→ loss of consciousness and genera convulsion. (2) Intense change of temperature R. fingers→ R. arm→ R. cheek (R. hand sweats). (3) Agnosia R. face and head T
22 (775)	R.	Nil	L. hand, moderate (ulnar)	—	(1) Numbness L. hand→ paralysed or weak L. hand
23 (231)	L.	R. hand, slight	R. hand and upper lip moderate	Sensory aphasia	(1) Numbness R. hand→ clonic R. hand→ cold R. face→ R. eyelid and lip (R. leg may twitch). (2) Eyes to R→ cry→ loss of consciousness→ tonic fit
24 (432)	L.	Nil	R. hand, severe (early)	Aphasia	(1) Clonic head→ adversive tonic→ loss of consciousness with genera convulsion
25 (8)	R.	Nil	L. arm, L. leg, moderate	—	(1) Clonic L. face. (2) Clonic L. arm→ L. arm paralysis 3–4 min.
26 (44)	R.	L. arm, L. leg, slight	L. arm, L. leg, severe	Slight loss L. visual field	(1) Clonic L. leg→ L. arm
27 (171)	L.	R. arm, R. leg, slight	R. arm, R. leg, severe	R. hemianopia	(1) Tonic R. arm R. face→ clonic R. arm, R. face, aphasia
28 (184)	L.	R. arm, R. leg, slight	R. arm, R. leg, severe	—	(1) Clonic R. leg→ R. arm (numbness after). (2) (1)→ loss of con sciousness. (3) (1)→ aphasia
29 (905)	R.	Nil	L. leg, slight	—	(1) Clonic L. leg→ clonic L. abdomen. (2) (1)→ L. arm, L. hand→ tonic adversive head to L.
30 (189)	R.	L. arm, L. leg, slight	L. hemianopia	—	(1) Pins and needles L. face. (2) Pins and needles L. hand→ L. face→ L. leg→ heat L. arm, L. leg. (3) Adversive tonic head to L.→ loss of consciousness→ general convulsion T.
31 (17)	R.	L. arm, L. leg, slight	L. arm, L. leg, moderate	—	(1) Numbness L. leg→ L. arm→ L. chest. (2) (1)→ tonic adversive head to L. (dribble saliva)

*The cases are divided into seven groups according to type and degree of deficit. The second case number refers to the case index. " T " indicates that the case is described in the text.

†When a focal fit proceeds to loss of consciousness, this is always indicated. If a cerebral abscess developed this is mentioned.

ND VISUAL DEFICIT, AND TYPES OF FIT IN 83 CASES*

TRACINGS OF SKULL RADIOGRAPHS‡



‡Site of skull penetration is shaded ; metal foreign bodies are dense black. Bone chips in the brain appear as triangles (where pre-operation radiographs are available). An outline of the ventricles as seen by air encephalography is sometimes shown.

[*Continued overleaf*

TABLE IX

Case and Index Nos.	Side	Motor Loss	Cortical Sensory Loss	Other	Type of Fit
GROUP 3.—*Wounds in which a metal fragment travelling in the sagittal plane undercuts the Rolandic area but only causing a limited degree of sensori-motor loss*					
32 (187)	R.	L. arm, L. leg, moderate	L. leg, moderate	—	(1) Tonic eyes→ L. arm. (2) Clonic foot. (3) Dizzy→ fall (no loss c consciousness)
33 (782)	R.	L. arm, L. leg, severe (early)	Nil	Aphasia early	Clonic L. foot→ L. leg→ L. shoulder→ sternomastoid→ L. trice (focal status for 10 days)
34 (168)	L.	R. face, slight	Nil	L. frontal abscess	(1) General convulsion, no aura. (2) Clonic R. hand→ aphasia→ cloni R. face→ general convulsion (clonic on R. only)
35 (891)	L.	R. arm, R. leg, slight	R. arm, R. leg, slight	—	Adversive tonic head to R.→ tonic R. arm, wrist, elbow→ clonic foot— L.→ loss of consciousness (weak R. arm and R leg for 20 min. after). T
36 (399)	R.→ L.	R. arm, R. leg, slight	Nil	—	(1) Numbness L. arm→ agnosia L. arm→ paralysed or weak L. arm (2) As in (1)→ L. leg. (3) Giddiness. T
37 (2746)	L.	R. arm, slight	Nil	—	(1) Numbness, R. hand→ R. arm→ shoulder→ R. leg and jaw→ loss c consciousness (general convulsion). (2) (early) Clonic R. face→ R. leg L. leg
38 (463)	R.	L. arm, L. leg, moderate	L. arm, L. leg, moderate	Hemianopia	(1) Tonic L. hand→ clonic→ loss of consciousness (general convulsion) (2) Electric shock sensation L. arm→ L. face→ clonic L. face
39 (2859)	R.	L. arm, L. face, slight	L. arm, L. face, slight	—	(1) Feeling of band L. wrist→ tonic L. hand→ clonic L. fingers— →face→ loss of consciousness. (2) Tonic jaw→ burning L. hand— arm→ loss of consciousness. (3) (early) R. "focal motor"
40 (849)	R.	L. hand, slight	L. hand, slight	—	(1) Numbness all L. side (giddy)→ jerk L. arm, L. leg. (2) Pins an needles L. arm→ burning (severe)→ trunk, L. leg→ cold
41 (522)	R. & L.	Nil (early R. arm, R. leg)	Nil	Lower R. quadrantanopia, aphasia	(1) Numbness R. arm→ R. face (paralysed or weak R. arm)→ twitchin R. arm→ R. face. (2) Light R. visual field→ R. triceps feels torn (3) Sudden giddiness→ staggers
GROUP 4.—*Frontal or temporal wounds with little or no sensori-motor loss*					
42 (733)	L.	R. hand, slight	—	Lower quadrantanopia, aphasia	(1) Aphasia→ hot R. hand and shoulder→ clonic R. hand, R. arm→ general convulsion. (2) Aphasia→ loss of consciousness (genera convulsion) T
43 (704)	L.	R. arm, R. leg	R. arm (slight)	—	(1) Clonic with pins and needles R. fingers→ hand→ arm. (2) (1)→ clonic R. face, R. leg→ aphasia. (3) General convulsion (no aura)
44 (864)	L.	R. arm, slight	R. arm, slight	Aphasia	(1) Hot R. hand→ R. arm. (2) (1) plus clonic R. hand. (3) Clonic 4th an 5th fingers→ R. hand, arm→ general convulsion T
45 (723)	R.	—	—	—	(1) Pins and needles R. fingers→ numbness R. hand→ clonic R. fingers→ tonic R. arm. (2) as (1)→ loss of consciousness
46 (948)	L.	—	—	Slight aphasia	(1) Tonic adversive head to R.→ tonic R. arm→ tonic L. arm→ clonic R. arm L. arm
47 (371)	L.	—	—	—	Clonic eyes→ general convulsion (clonic R. face, arm, leg)
48 (207)	L.	—	—	—	Tonic eyes→ fall (spiral movement), no general convulsion
49 (813)	R.	—	—	—	Adversive tonic head to L.→ loss of consciousness (clonic L. arm, L. leg
50 (593)	R.	—	—	—	(1) General convulsion in sleep. (2) Clonic L. face, L. arm→ loss o consciousness (general convulsion)
51 (814)	L.	R. face, slight	—	—	(1) Numbness R. face→ R. arm→ paralysed or weak R. hand. (2) (1)→ loss of consciousness. (3) (2)→ general convulsion
52 (36)	R.	R. face, slight	—	—	(1) General convulsion, no aura. (2) Clonic R. arm, R. face, eyes adversive clonic to R. (between 2 general convulsive attacks) T
GROUP 5.—*Posterior parietal wounds with little or no sensori-motor loss*					
53 (510)	L.	—	R. hand, slight	Lower quadrantanopia	(1) Visual spot→ phantom R. arm movements. (2) R. arm numb→ clonic R. arm. (3) general convulsion T
54 (922)	L.	—	—	Lower quadrantanopia, aphasia	(1) Hot R. hand→ arm→ edge of face→ tonic hand→ clonic R. arm T
55 (467)	R.	—	—	Radiograph, normal	(1) Paralysed or weak R. leg, L. leg→ fall→ loss of consciousnes (momentary). (2) Epigastric sensation→ burning L. hand→ arm→ shoulder
56 (390)	L.	—	—	R. attention hemianopia, aphasia	(1) General convulsion (no aura). (2) Paralysed or weak R. hand
57 (571)	R.	L. arm, L. leg (early)	L. arm, L. leg (early)	L. hemianopia. Abscess	(1) (early only) Adversive tonic head, eyes to L.→ tonic L. arm→genera convulsion. (2) Paralysed or weak L. hand→ loss of consciousnes (general convulsion)
58 (71)	L.	—	R. arm, slight	Lower quadrantanopia	(1) Pale→ loss of consciousness (general convulsion). (2) Phantom R. arm above head (clenched) T

Continued

TRACINGS OF SKULL RADIOGRAPHS‡

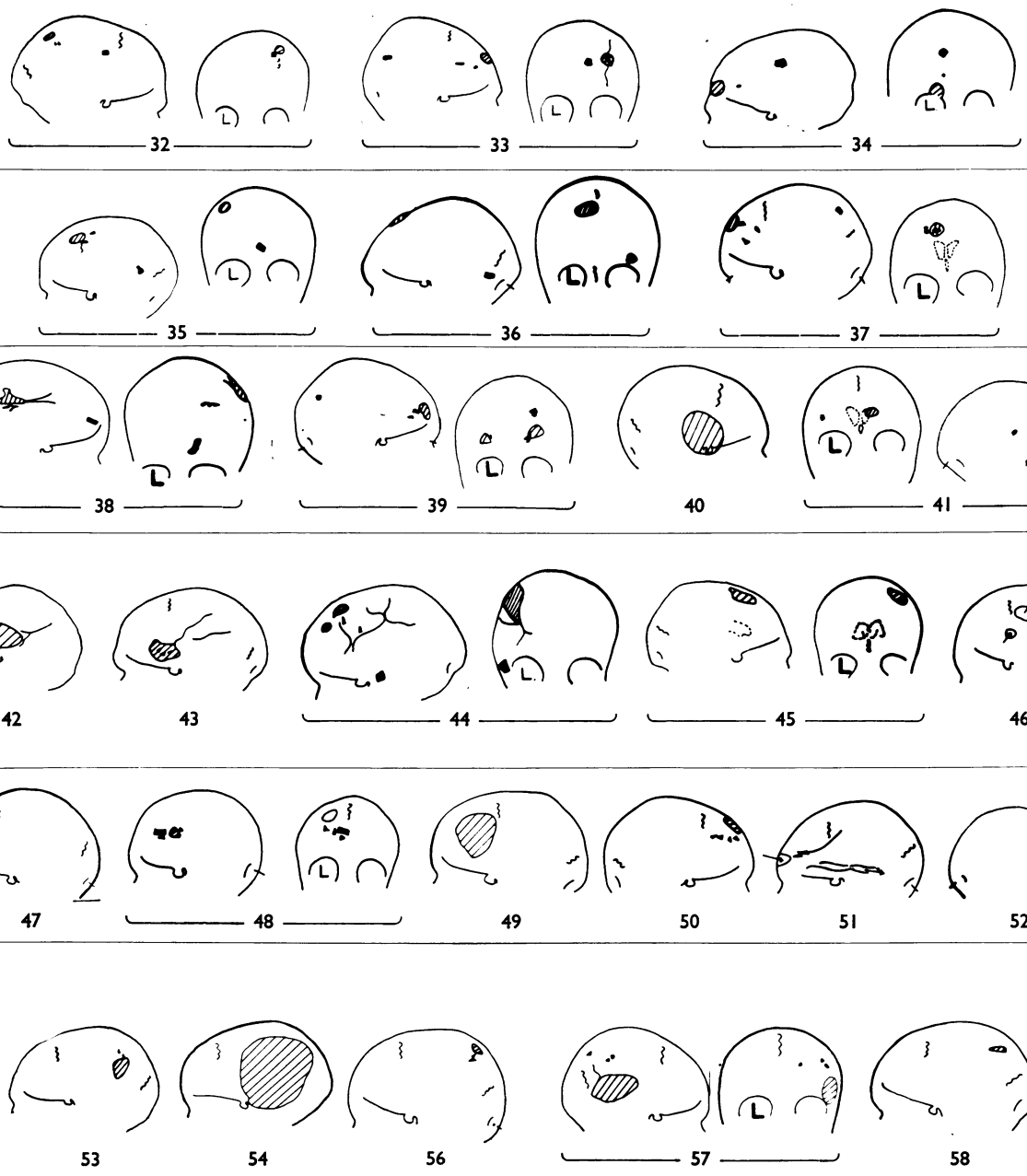


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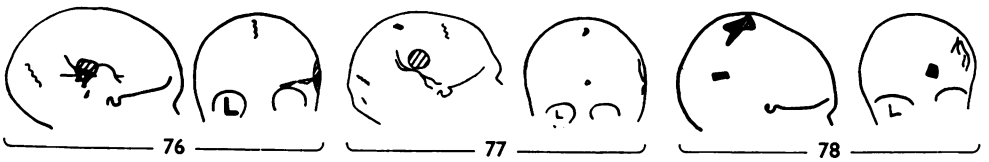
TABLE IX—

Case and Index Nos.	Side	Motor Loss	Cortical Sensory Loss	Other	Type of Fit
59 (266)	R.	R. arm, L. leg, slight	L. arm, L. leg, slight	Hemianopia	(1) Agnosia L. hand. (2) Agnosia L. hand → visual → general convulsion
60 (931)	L.	—	—	Lower quadrantanopia	(1) R. hemianopia, clonic eyelids → loss of consciousness. (2) Clonic R. arm → clonic R. leg
61 (485)	R.	—	—	L. hemianopia	(1) Pins and needles L. side. (2) (1) → clonic L. hand, L. leg → hemianopia (3) as (2) → loss of consciousness (general convulsion)
62 (800)	L.	—	—	Dyslexia	(1) Funny feeling R. face → loss of consciousness (general convulsion) (2) Tinnitus → loss of consciousness (general convulsion)
63 (896)	L. → R.	—	—	Aphasia bilateral, visual field defect	(1) Clonic L. face → L. hand. (2) Clonic L. leg → L. hand → L. face (3) As in (1) then → loss of consciousness. (4) General convulsive status with focal clonic L. hand, L. face intermittent between general convulsion attacks. (5) Aphasia → general paralysis or weakness T
GROUP 6.—*Cases with severe hemiplegia, sensory and motor, but no hemianopia*					
64 (470)	Midline	R. leg, L. leg, R. arm, severe	R. leg, L. leg, severe	—	(1) Adversive tonic head to R. → R. shoulder tonic → R. shoulder clonic
65 (708)	L.	R. arm, R. leg, severe	R. arm, R. leg, severe	Aphasia	(1) General tonic → loss of consciousness. (2) Tonic R. arm T
66 (53)	L.	R. arm, R. leg, moderate	R. arm, R. leg, moderate	Aphasia	(1) Tonic-clonic R. hand → R. foot. (2) Cry → general convulsion clonic R. side only
67 (419)	R.	L. arm, L. leg, severe	L. arm, L. leg, severe	Aphasia	(1) Tonic adversive head to L. → loss of consciousness → clonic L. face arm, leg → R. leg, arm, face. (2) Epigastric → vertigo → clonic L. hand → L. leg
68 (853)	R.	L. arm, L. leg, severe	L. arm, L. leg, moderate	—	(1) Tonic adversive head to L. (2) Clonic L. foot focal status (early only) T
69 (702)	L.	R. arm, R. leg, moderate	R. arm, R. leg, severe	—	(1) Vertigo to L. → aphasia to hemianopia → tonic R. hand (stopped by gripping hand). (2) Adversive tonic head to R. → loss of consciousness (3) General convulsion → no aura
70 (2697)	R.	R. arm, R. leg, severe	R. arm, R. leg, severe	—	(1) L. fingers clonic → L. arm. (2) As (1) → adversive tonic head to L. → loss of consciousness (general convulsion)
71 (54)	L.	R. arm, R. leg, severe	R. arm, R. leg, severe	—	(1) Tonic-clonic R. shoulder → R. leg. (2) As (1) → loss of consciousness (3) General convulsion, no aura (early)
72 (928)	R.	L. arm, L. leg, severe	L. arm, L. leg, severe	—	(1) Fall → loss of consciousness. (2) Twitching L. leg → loss of consciousness
73 (959)	L. → R.	L. arm, L. leg, severe	L. arm, L. leg, moderate	—	(1) (Early) painful spasms L. leg. (2) Vibration R. arm → R. leg
74 (706)	L.	R. arm, R. leg, severe	R. arm, R. leg, severe	Aphasia	(1) Tonic R. face → R. arm. (2) Clonic R. face → R. arm → R. leg (3) Clonic R. abdomen, thorax → pain R. abdomen, R. thorax R. neck T
75 (821)	R. → L.	L. arm, L. leg, severe	L. arm, L. leg, severe	—	(1) Clonic L. arm → numbness L. arm → loss of consciousness → general convulsion. (2) Sudden jerk T
GROUP 7.—*Cases with hemiplegia and hemianopia*					
76 (534)	R.	L. arm, L. leg, moderate	L. arm, L. leg, moderate	Hemianopia, spatial loss	(1) Phantom movements L. fingers. (2) As (1) → tonic L. hand → clonic L. arm. (3) As (2) → visual hallucination (formed) → loss of consciousness T
77 (937)	R.	L. arm, L. leg, severe	L. arm, L. leg, moderate	Hemianopia	(1) Electric shock sensation L. arm → clonic L. arm → face → leg (L. arm L. leg paralysed or weak 20 min.). (2) Tonic L. arm → loss of consciousness (general convulsion) T
78 (157)	R.	L. arm, L. leg, severe	L. arm, L. leg, severe	Hemianopia	(1) Tonic-clonic L. face, tonic adversive head to L. → L. arm, L. leg (2) As (1) → loss of consciousness
79 (762)	R.	L. arm, L. leg, severe	L. arm, L. leg, severe	Hemianopia, athetosis	(1) Early focal status L. hand T
80 (173)	R.	L. arm, L. leg, moderate	L. arm, L. leg, severe	Lower quadrantanopia	(1) Clonic L. side. (2) General convulsion, no aura
81 (518)	R.	L. arm, L. leg, moderate	L. arm, L. leg, severe	Lower quadrantanopia	(1) Clonic L. shoulder → L. leg → loss of consciousness (general convulsion)
82 (861)	L.	R. arm, R. leg, severe	R. arm, R. leg, severe	Hemianopia	(1) Clonic R. hand (15 min.) → loss of consciousness. (2) Clonic R. hand → clonic R. leg
83 (917)	R.	R. arm, L. face, severe	R. arm, R. leg, severe	Lower quadrantanopia	(1) Agnosia L. arm, L. leg, L. face → adversive tonic head to L. → loss of consciousness (clonic L. arm, L. leg). (2) Loss of vision → dizzy

Continued

TRACINGS OF SKULL RADIOGRAPHS‡



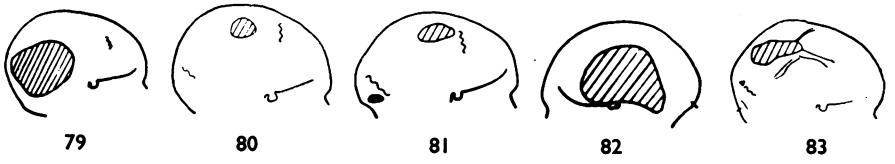


TABLE X

PRESENTING FOCAL AURAS COMPARED TO SENSORI-MOTOR DEFICIT USING SEVEN GROUPS IN TABLE IX

Groups from Table IX		Small Wounds					Hemiplegia	Hemiplegia and Hemianopia	
		M = S M > S	S > M	Undercutting	Fronto-temporal	Posterior Parietal			
		1	2	3	4	5	6	7	
	Totals	20	25	14	13	1	14	8	106
Motor	Adversive	—	3	1	3	1	4	—	12
	Tonic	5	1	3	—	—	4	2	15
	Clonic	6	8	4	5	2	4	3	32
	Jerk	1	—	—	—	—	2	—	3
	Inhibitory	1	—	—	1	3	—	—	5
Sensory	Tingling, pins and needles	3	4	1	1	2	—	—	11
	Numbness	1	4	4	1	1	—	—	11
	Pain	1	1	—	—	—	—	—	2
	Hot and cold	1	1	—	2	1	—	—	5
	Electric shock	—	2	1	—	—	—	1	4
	Agnosia	1	1	—	—	1	—	1	4
	Phantom	—	—	—	—	1	—	1	2

S = sensory deficit. M = motor deficit.

appreciated. Aphasia was more marked on the motor than the sensory side.

Fits.—**1.** Intermittent slow tonic contraction of the triceps muscle in the right arm occurred. This caused a slow extension of the arm which subsequently relaxed to the position of semi-flexion.

2. There was a sudden generalized tonic stiffening, then loss of consciousness and generalized clonic movements.

Focal Sensory Attacks

The various types of sensory discharge also show some differences in their capacity for detailed reference (Table VIII). For example, the sensation of pins and needles may be referred precisely, as in the following cases (Nos. 6, 19). Sensory auras seem to differ, however, from the clonic motor auras in the rapidity with which they spread, for though they may begin in some localized part of a limb, it is usual for them to spread quickly to involve at least the whole limb. The occurrence of a sensory aura which remains localized for hours or even days, as in focal motor status, has never been recorded in these cases. Another difference is seen in the fact that a motor fit will readily develop after destruction of the motor cortex, but as will be considered later, sensory fits are only described when a part at least of the sensory cortex is intact.

CASE 6. **Focal Sensory Attacks with Rapid Spread Leading to Focal Motor Attacks**

A left fronto-parietal wound soon after wounding caused right hemiparesis with global dysphasia. There was sensory loss in the right hand, stereognosis and movement sense being markedly reduced: the leg was less affected. The permanent disability included spastic hemiparesis with reduction of joint sense in fingers and toes. There was some hyperpathia in the left hand, and some residual dysphasia, especially at times of emotion.

Fits.—**1.** A pins-and-needles feeling suddenly appeared in the right shin. This passed rapidly up the leg to the thigh, and was followed by a jerking of the foot and then of the arm. The jaw was then tightly clenched, but there was no clonic movement. There was no loss of consciousness.

These attacks could sometimes be stopped by using the muscles of the affected limb as strongly as possible. This applied both to the leg and arm.

CASE 19. **Focal Sensory Attacks with Extensive Spread and Focal Visual Epilepsy**

In a left anterior parietal wound soon after wounding there was a right hemiparesis and aphasia and some continuous, ill-defined, spontaneous pain in the right upper limb. Passive movement sense was reduced in this limb, but pin-prick was appreciated. Some years later there was slight right hemiparesis, with reduction of passive movement sense and two-point touch in the right hand, mainly on the ulnar border. No weakness was present, and there was no dysphasia.

Fits.—**1.** A sudden pins-and-needles feeling appeared in the right upper lip. This then passed to the hand and up the arm to the elbow. Then it started in the right foot and the right flank. At this time the patient found difficulty in concentrating, and complained of not being able to see properly.

2. A sudden visual blackout lasted for a few seconds only. There was no loss of consciousness.

Focal Attacks in the Lower Limb

One striking feature of Table VIII is the relative infrequency of attacks beginning in the lower limbs.

Of the total 135 types of attack analysed, only 17 started in the lower compared with 69 in the upper limb. The disparity occurred in both motor and sensory attacks. This does not represent simply a lower percentage of wounds involving the leg area, as may be seen from Fig. 2 (Part 1), when the distribution of all wounds with or without epilepsy was considered. Indeed, detailed consideration of the only four cases which showed clonic motor attacks starting in the foot suggests that special physiological conditions may be required for ictal firing of this area. In one the attacks only occurred just before going to sleep, while in two others they only occurred during the first few weeks after wounding.

CASE 32. **Focal Motor Attacks Starting in the Foot and Adversive Attacks**

A right posterior parietal to frontal wound undercutting cortex early after wounding caused left hemiplegia and homonymous hemianopia. There was loss of all forms of sensation in the left leg below the knee, and to a lesser degree in the left hand. Some years later spastic left hemiparesis persisted, the leg being more affected than the arm. There was cortical sensory impairment in the left leg and also slight change in the appreciation of pin-prick. No marked sensory change in the arm was noted.

Fits.—**1.** There was occasional clonic jerking of the left foot only with no tonic stiffening. This only occurred just before going to sleep.

2. The eyes suddenly turned to the left, then the left arm jerked and was drawn up to the face. There was no loss of consciousness, and the leg was not involved.

3. A sudden dizzy feeling occurred with falling but no loss of consciousness.

CASE 13. **Clonic Focal Fits of Foot and Occasional Loss of Consciousness**

There was a right anterior parietal brain wound, and, some years after wounding, slight spastic weakness of the left arm and leg with some sensory loss of cortical type, persisted.

Fits.—**1.** Clonic movement of the left foot occurred at the same time as clonic jerking of the left hand and arm. The fit then ceased.

2. There were sudden clonic movements of the left foot, the left arm was then drawn up in a tonic movement and consciousness was lost.

Cases 33 and 68, of partial continuous epilepsy of the foot, also illustrate this point, and are considered later. The foregoing case records illustrate certain general features which may be seen at the *onset* of any focal motor or sensory attack. However, the subsequent course of the attack which is next considered, and the detailed content of various types of sensori-motor fit, especially some of the more unusual ones mentioned later, also provide material of interest in the study of cerebral function.

SPREAD OF INITIAL ATTACK

Great efforts have been made in this study to collect reliable information as to the spread of the epileptic aura, and to analyse the results in relation to possible directions, rates, or patterns of spread of the different varieties described. Several features of this analysis have been recognized and described, but we are fully conscious of the likelihood that the facts collected supply additional information of importance which we have so far failed to recognize and extract from the recorded observations.

Extent and Rate of Spread

Definite information about the rate of spread is scanty. It is usually a matter of seconds, for instance, for the common sensory aura of pins and needles starting in the hand to reach the shoulder or the edge of the face. In the case of focal clonic fits, however, the rate of spread is often much slower, and in the following case, where accurate timing was done, lasted for about eight minutes.

CASE 585 (not included in tables). **Focal Clonic Motor and Focal Sensory Attacks**

A right frontal brain wound (Fig. 3), with indriven bone fragments which had reached the head of the caudate nucleus, caused left spastic hemiplegia with athetotic posture of the limbs, exaggerated by any attempts at voluntary movement. In the foot this produced a spastic extension of the great toe so uncomfortable as to require amputation. Fits started some months after wounding as follows. There were sudden clonic movements of the face, which passed down to the shoulder, then to the elbow (but not the hand), to the thoracic muscles, to the thigh, and to the foot. The attacks then ceased. Eight minutes elapsed between the onset of facial movements and those in the foot. Sometimes after a motor attack there was a sudden tingling sensation in the left fingers which passed rapidly up the arm to the shoulder, to the edge of the face, then down the left flank and from the thigh down to the toes.

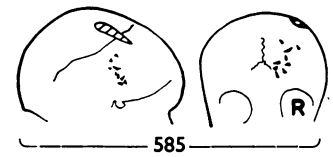


FIG. 3.—Skull tracing of Case 585.

It has already been pointed out that sensory auras spread quickly, also that sensations of pins and needles or tingling may have a very local and precise reference. Other types of sensory aura such as pain, heat or cold, often affect a larger area

from the start, but they too show the tendency to rapid spread. These features are shown in the following cases :—

CASE 8. **Focal Thermal Sensory Attacks of Wide Reference and Grand Mal attacks and Myoclonic Jerks**

A left parietal brain wound shortly after wounding caused right hemiparesis with sensory loss in the right limbs, and global dysphasia, but no field defect. Some years later there was spastic weakness of the upper limb with cortical sensory loss and some loss of pain sense in the finger tips only.

Fits.—**1.** The right arm suddenly felt intensely cold. This spread rapidly to the leg, and the attack ceased.

2. The right arm would suddenly give an uncontrollable jerk. This commonly followed some stimulus such as an unexpected noise.

3. There was a sudden cry, then loss of consciousness and a generalized convulsion.

CASE 54. **Focal Sensory Attacks of Heat with Extensive Spread followed by Motor Involvement**

A left posterior parietal brain wound soon after wounding caused gross global dysphasia with right homonymous hemianopia and some weakness of the right hand. Some years later dysphasia was present, though dysgraphia and dyslexia were more marked. There was a lower right homonymous field defect, but no weakness or sensory loss.

Fits.—A sudden hot feeling occurred in the right hand. This was not painful, but like holding it near a fire. It passed rapidly up the hand and arm to the edge of the face, and then the hand was drawn into a tonic spasm and there was some clonic jerking at the elbow.

Inhibitory auras, whether sensory or motor, seem always to affect a large area *ab initio*. The whole of a limb is involved, and this usually spreads to the whole of one side of the body. Indeed, inhibitory auras tend to be more widespread also in the sense that they often involve both sensory and motor functions.

CASE 36. **Inhibitory Sensory Attacks of Wide Reference and Some Motor Inhibition**

A right frontal to left occipital brain wound early after wounding caused slight weakness of the left arm and leg, but no detectable sensory change. Some years later there were no abnormal signs in the nervous system.

Fits.—**1.** A sudden numbness occurred in the left arm. This was a negative feeling as if the arm were not fully there. While this lasted the arm could not be used properly.

2. As above, but the sensation passed rapidly after onset in the left arm to the left leg. The same motor disability was also noted here.

CASE 9. **Motor Inhibitory Attacks with Wide Reference as Aura to Generalized Tonic Fit**

A left mid-parietal parasagittal wound shortly after wounding caused spastic paralysis of both legs, with flaccid weakness of the right arm, and severe loss to all forms of sensation including pain and light touch in those limbs. There was an attention defect in the right visual field. Some years later there was still weakness of the right arm and both legs, with some increase in tone. Passive movement sense and two-point touch were lost in the toes, and impaired in the right hand.

Fits.—The right arm suddenly became powerless and fell to the side. This rapidly spread to the right leg, so that the patient fell to the right side. Shortly thereafter consciousness was lost, and a brief general tonic stiffening occurred.

Direction of Cortical Spread

When a number of cases are available for study the order in which focal phenomena may follow each other is clearly of significance in relation to the study of cerebral physiology, especially if, as appears from this series, the spread tends to be in certain specific directions. Table XI analyses this spread of the focal phenomena one to another. For this table a group of cases with sensory or motor fits is analysed as regards all the focal phenomena which could be described in detail. (It is the change from one type of aura to another which is recorded so that more than one entry often appears for one case.) It must also be emphasized that the *cessation* of an epileptic phenomenon is just as positive an event (inhibition) as is the spread to another form of discharge, so that special attention is devoted to those forms of aura which precede a complete arrest of the fit. Also the spread to cause loss of consciousness and a general convulsion is of special physiological interest, and those auras proceeding to loss of consciousness are indicated.

Some remarkable facts emerge from Table XI. In the first place it becomes apparent that a sensory aura frequently passed to a motor aura, whereas spread from motor to sensory is rarely recorded. In no case did tingling or pins and needles follow a motor aura, and in only one case is there a clear record of clonic focal movements on one side being succeeded by the common somatic sensory aura of numbness. In the other few instances of sensory phenomena following motor, they either involved the special senses, auditory or visual, or a sensation such as heat or pain, which are uncommon types of aura.

Case 76 illustrates in some of the fits the spread of clonic movement to a visual aura, and the following three cases also illustrate some of these unusual types of spread.

TABLE XI

SEQUENCE OF EVENTS FOLLOWING MANY FOCAL AURAS*

Focal Auras			Totals	Subsequent Event: Tingling, pins and needles	Pain	Shock	Heat and cold	Numbness	Agnosia	Phantom	Adversive	Tonic	Clonic	Jerk	Paralysis	Aphasia	Loss of consciousness	Fit ceased	Other
	Sensory	Tingling, pins and needles	24	**9**	—	—	2	—	—	—	1	—	5	—	—	1	1	4	1
		Pain	6	—	—	—	—	—	—	—	—	—	1	—	—	—	1	3	1
		Electric shock	4	—	—	**1**	—	—	—	—	—	—	2	—	—	—	—	1	—
		Heat or cold	16	—	—	—	**6**	—	—	—	—	1	2	1	—	—	1	5	—
		Numbness	19	—	—	—	—	**5**	1	—	1	1	4	2	3	—	1	1	—
		Agnosia	7	—	—	—	—	—	**1**	—	1	—	—	—	1	—	—	3	1
		Phantom	4	—	—	—	—	—	—	—	—	1	—	—	—	—	—	3	—
	Motor	Adversive	17	—	—	—	—	—	—	—	—	**6**	—	—	—	—	8	3	—
		Tonic	38	—	—	—	1	—	—	—	—	**3**	16	—	1	—	7	10	—
		Clonic	94	—	1	—	2	1	—	—	3	6	**23**	—	3	3	20	30	2
		Jerk	6	—	—	—	—	—	—	—	—	—	1	—	—	—	1	4	—
		Paralysis	12	—	—	—	—	—	—	—	—	—	—	—	**1**	—	4	7	—
		Aphasia	7	—	—	—	1	—	—	—	1	—	1	—	1	—	1	1	1
		Other	13	—	1	—	1	—	—	1	—	1	2	—	—	1	4	2	—
		Totals	267	9	2	1	13	6	2	1	7	19	57	3	10	5	49	77	6

* The " subsequent " events may be a spread of the same type of aura to another region (squared bold numbers), or spread to another type of aura, or to loss of consciousness, or to cessation of the fit.

CASE 75. **Clonic Motor Attack followed by Sensory Aura followed by Grand Mal**

A right frontal to parietal wound (through and through) early after wounding caused spastic left hemiplegia, with gross sensory loss. Some years later there was still severe hemiparesis more in the arm than in the leg, and cortical sensory loss of the same distribution.

Fits.—Sudden clonic movements occurred in the right hand and leg, a numb dead feeling followed in the hand and leg, then consciousness was lost and clonic movements ceased, while generalized tonic stiffening occurred, which was then followed by generalized clonic movements.

CASE 21. **Focal Clonic Attacks Passing to an Auditory Sensation before Grand Mal and Focal Thermal Focal Agnosia Attacks**

A left parietal wound soon after wounding produced severe global dysphasia. There was also weakness of the right arm and leg and marked cortical sensory loss in the right hand and face. Some years later there was a residual dysphasia of expressive and receptive types. There was loss of position sense and stereognosis in the right hand together with some change in the appreciation of pain. There was a very slight spastic weakness of the arm, but the leg appeared normal.

Fits.—**1.** There was a sudden twitching of the right hand, then a ringing noise in the right ear, then loss of consciousness. This was followed by generalized stiffening, but no generalized clonic element.

2. Sudden intense feeling in the finger-tips ran rapidly up the right arm to the cheek. The feeling was of change of temperature, but was so intense that it seemed sometimes freezing and sometimes burning. The hand sweated profusely during this attack.

3. A sudden feeling as if the face and head on the right had ceased to exist.

CASE 74. **Focal Clonic Aura followed by Focal Pain and Other Types of Motor Attack**

A left high parietal to inferior frontal wound caused early right hemiplegia and hemianaesthesia. Later a marked spastic paresis in the right hand with cortical sensory loss persisted. The right leg was a little weak and spastic with some passive movement sense loss. There was gross dysphasia and some dementia.

Fits.—**1.** Twitching of the right abdomen proceeded to the right intercostal muscles and then severe gripping pain occurred in the abdomen and chest wall and passed up to the neck.

2. There was sudden stiffness and numbness of the right face but no movements.

3. Clonic twitching occurred in the right face, then in the right abdomen and in the right leg.

4. (early) A right-sided clonic fit occurred in the arm and then in the leg.

It might be argued that the occurrence of involuntary movement is so striking for the patient as to allow subsequent numbness in the part affected to pass unremarked, whereas pain or a special sense aura, being more obtrusive, would be noted. However, this is unlikely to account entirely for the great difference in frequency of these two types of spread: apart from the case quoted above (No. 75) there were two others in which numbness developed and spread *pari passu* with a clonic or tonic movement (Cases 3 and 43). It seems clear that while the spread of a somatic sensory aura to a motor one is quite usual, the reverse method of spread is quite exceptional.

While all forms of sensory aura often spread quickly to a motor aura, there is little tendency for one type of sensory aura to spread to another type. This suggests that the cortical cells concerned with these various sensory functions have easier anatomical access to the motor cortex than to post-central cells concerned with other types of sensation. The only type of sensory aura in this series which proceeded to a general convulsion without report of a motor phase of the aura were Cases 20 and 39, in which the aura was of intense local pain or heat; Case 39 was remarkable in the spread from focal tonic to focal burning, and then loss of consciousness with convulsions involving only the one (the affected) side.

CASE 20. **Aura of Focal Pain Immediately Followed by Grand Mal and Attacks of "Electric Shock" with Clonic Movement**

A right parietal brain wound soon after wounding produced a left hemiparesis with sensory loss in the left hand, and a left homonymous visual field defect. Some years later there was a left inferior quadrantic field defect, a slight spastic weakness of the left hand and face, with sensory loss in the hand and the face of discriminative cortical type.

Fits.—**1.** There was a sudden, intensely unpleasant pain in the whole left arm, which passed up to the face and the corner of the left eye, then consciousness was lost and a general convulsion ensued. The pain in the arm was hard to describe, but phrases used were "unnatural", "intensely unpleasant", "a torture of pain".

2. Sometimes the pattern was as in (1) but preceded by a dead feeling in the arm for a day or so, and was considered as the herald of an attack.

3. The third manifestation was seen only in early fits. Sudden, shock-like feelings occurred in the left hand, which spread rapidly up to the shoulder, and were followed by twitching of the shoulder only.

CASE 39. **Motor Tonic Aura Spreading to Burning Sensation and Other Types of Attack**

A right frontal to posterior parietal brain wound soon after wounding caused left hemiparesis with sensory loss. Some years later there was a slight spasticity and weakness of the left hand and face, some reduction of stereognosis and passive movement sense in the hand, and some two-point discrimination loss in the face.

Fits.—**1.** Tonic clenching of the jaw was followed by intense burning of the left hand. This marched up the arm to the shoulder, consciousness was lost, and a generalized tonic stiffening occurred with clonic movements of the left side only.

2. There was a sudden feeling of constriction as if a tight band was around the left wrist. There was then some tonic stiffening of the hand followed by clonic movements which proceeded up the arm to the face. Consciousness was then lost and convulsions occurred as in (1).

3. Right-sided convulsions occurred with no involvement of the left side; this pattern was only seen in early fits.

The relationship between a clonic and tonic aura is also of interest (Table XI). Sixteen out of 39 tonic episodes became clonic, but only six of the 95 clonic auras changed to tonic. Such "paths of election" for spread are further emphasized in Table XIII so far as the clonic motor auras are concerned. Here it may be seen that in seven out of 18 instances clonic movement in the face passed to loss of consciousness, while loss of consciousness followed directly in only one case of 27 where clonic movement occurred in the thumb or fingers. This is a reflection of the fact that movements starting in the digits tend to show a Jacksonian march if they spread, as can also be seen in this table. It would appear that the areas for face and head representation have very ready access to these mechanisms concerned with loss of consciousness, an observation already implicit in much earlier clinical literature on epilepsy where the common occurrence of loss of consciousness as the aura reached the head was noted.

Table XII extracts some of the information in Table XI from the point of view of comparing the behaviour of motor and sensory auras. Here again there is evidence that the epileptic process moves more easily in one direction than another.

These findings indicate certain physiological patterns for the spread of the epileptic process which deserve further study.

TABLE XII
COMPARISON OF SPREAD OF MOTOR AND SENSORY AURAS

	80 Examples of Sensory Aura	174 Examples of Motor Aura
Spread to sensory..	Same* : 22 (27%) Other : 3 (4%)	6 (3%)
Spread to motor ..	28 (35%)	Same* : 27 (15%) Other : 42 (24%)
Fit ceased	20 (25%)	55 (31%)
Spread to loss of consciousness ..	4 (5%)	41 (24%)
Other	3 (4%)	3 (2%)

* " Same " indicates spread of the same type of aura to another part of the body (see Table XI).

Types of Spontaneous Cessation of Focal Attacks : Post-epileptic Paralysis

The ending of a focal attack is also of interest, and is recorded as occurring after all varieties of aura (Table XI). Clonic motor attacks usually die out gradually with a gradually lengthening interval between each clonic movement. Sometimes they are followed by weakness, though not complete paralysis, of the affected limbs which may last 20–30 minutes, long after all movements have ceased (Todd's paralysis, 1861). There seems nothing in the intensity of the clinical attack to suggest an " exhaustion paralysis " and it seems more likely to be due to involvement or release of some inhibitory mechanism. The following case illustrates this point.

CASE 77. **Focal " Electric Shock " with Clonic and Inhibitory Fits followed by Local Paralysis ; Possible Spread to Grand Mal**

A right parietal brain wound was sustained. Some years after wounding there was a left homonymous hemianopia and left spastic hemiparesis, more in the arm than in the leg. No gross sensory change was noted early, but later some loss of stereognosis and two-point touch in the left hand was found.

Fits.—**1.** There was a sudden feeling as of an electric shock in the left elbow, then twitching of the muscles of the left forearm, followed immediately by twitching of the left face and of the left leg below the knee, in that order. At the same time loss of power in the arm and weakness and incoordination on attempted movement of the left leg occurred. This motor effect lasted about 20 minutes.

2. Attacks started as above, but the left arm was raised at the shoulder and the elbow forced backwards. This was followed by loss of consciousness with left-sided generalized clonic movements.

Sensory attacks, on the other hand, when they do not pass over to motor, as they often do, usually end abruptly. At one moment the sensation is still there, and the next moment it has cleared entirely. There seems to be no gradual fading away. This is well shown in the following case where the aura was an inhibitory one of sensory agnosia.

CASE 10. **Focal Inhibitory Sensory Attacks with Agnosia and with Abrupt Ending**

A deep left parietal wound soon after wounding caused right hemiplegia with sensory loss to all modalities, more in the arm than in the leg. There was also dysphasia, more expressive than receptive. Some years later the right hand was weak with increased tone, and there was loss of stereognosis, two-point touch, and passive movement sense. There was also a slight motor dysphasia which emerged when the patient was emotionally upset.

Fits.—The left arm and almost immediately afterwards, the left leg, suddenly seemed to disappear. " There is a feeling that they don't belong to me, and I

TABLE XIII
PATTERN OF SPREAD OF CLONIC FITS*

		Focal Movement or Other Subsequent Event												
		Face	Head and Eyes	Thumb	Hand and Fingers	Arm	Shoulder	Abdomen Trunk	Leg	Foot	Loss of Consciousness	Fit Ceased	Other	Totals
Focal Aura	Face	X	1	—	1	2	—	—	1	—	7	5	1	18
	Head and eyes ..	—	X	—	—	—	—	—	—	—	2	—	1	3
	Thumb	—	—	X	1	—	—	—	—	—	—	—	2	3
	Hand and fingers ..	5	—	—	X	5	—	—	2	2	1	7	2	24
	Arm	4	—	—	1	X	—	—	5	—	5	6	6	27
	Shoulder	—	—	—	—	1	X	—	2	—	1	2	—	6
	Abdomen or trunk ..	—	—	—	—	1	—	X	—	—	—	1	1	3
	Leg	—	—	—	1	2	2	1	1†	—	4	8	1	20
	Foot	—	—	—	—	2	—	—	4	X	—	4	—	10
		9	1	—	4	13	2	1	15	2	20	33	14	114

* Each clonic feature is entered according to list at left of Table. There may, therefore, be several entries for one case, and indeed for one fit.

† Spread from one leg to the other.

look at them with surprise ". This attack lasts about 20 minutes, and ends abruptly. There is no loss of consciousness and no further ictal phenomena.

" Jacksonian " Spread

In addition to the transcortical spread from one variety of aura to another, there is the better known type of spread from one part of the body to another. This " Jacksonian march " occurs most classically in some cases of focal clonic attack. In many cases of this group the aura, in fact, begins with a sensory focus and proceeds to clonic motor convulsion. A typical sensory march may also be seen, but, as has been mentioned, this tends to spread more quickly than do the clonic motor attacks.

The following cases illustrate these points, which are also shown in Cases 33, 16, and 6 detailed above.

CASE 66. **Spreading Focal Tonic and Clonic Fits, Grand Mal Attacks, without Aura**

A left parietal brain wound early after wounding caused dysphasia, with right spastic hemiplegia, including the face, and partial right hemianaesthesia to all types of sensation. Some years later there was slight residual dysphasia, most marked in emotional situations, with slight spastic weakness of the arm more than of the leg. Two-point touch appreciation was reduced in the right hand and stereognosis was markedly affected. Passive movement sense was grossly reduced in the right hand and arm up to the elbow. Pin-prick was felt as cold and painful, but was not localized. The leg showed very slight spasticity with sensory changes similar to those in the arm but less marked, and confined mainly to the foot.

Fits.—**1.** The right arm suddenly jerked up to the face, the hand was flexed, and clonic jerks appeared at the wrist for a few seconds. The thumb and fingers were flexed in tonic spasm but did not jerk. Just after the arm started moving, the foot also jerked, but without tonic spasm. There were no facial movements. The movements of the hand could be controlled usually by forcibly extending the right arm with the left. Standing up and taking the weight on the right leg will similarly abolish the movements in that limb.

2. A sudden cry occurred with loss of consciousness, falling, and generalized tonic stiffening. This was followed by jerking of the right side only.

CASE 3. **Focal Fits with Spreading Sensory and Motor Element, Loss of Consciousness with Aura of Dizziness**

A right posterior parietal brain wound soon after wounding caused slight left hemiparesis and reduction of passive movement sense in the left fingers and toes. Some years after wounding there was slight spatial disorientation in the left half visual field and some left finger agnosia. There was minimal weakness of the left arm and leg with slight loss of passive movement sense in the left fingers.

Fits.—**1.** Numbness passed up the left arm from the fingers, then into the left leg, starting with the toes and moving upwards, followed by jerking of the left hand at the wrist and the left foot soon afterwards. There was no loss of consciousness.

2. A sudden tingling in the fingers of the left hand passed rapidly up to the elbow and then disappeared.

3. Tonic rigidity and a numb feeling of the left abdominal wall was followed immediately by clonic jerking of this and of the cremasterics. There was no loss of consciousness.

4. There was a sudden dizziness and the patient had time to lie down before losing consciousness. There was pallor but no movements.

CASE 45. **Spreading Focal Sensory and Motor Attacks. Adversive Aura to Tonic Fit**

A left posterior frontal wound soon after wounding caused jargon aphasia and right hemiparesis with some loss to all forms of sensation on the right. Some years later there was still slight dysphasia and slight weakness and increased tone in the right hand. Stereognosis was reduced in this hand, as was two-point touch, and this reduction was also found on the right upper lip.

Fits.—**1.** The right hand suddenly went numb. This was followed in a few seconds by a jerking of the hand and the right eye-lid. The right side of the face felt intensely cold. Sometimes the lip also twitched at this stage. Speech was thick but there was apparently no dysphasia. Sometimes as the right hand started jerking, the right leg also twitched. There was no loss of consciousness.

2. The eyes seem forcibly turned to the right. The patient cried out, then lost consciousness, and a general tonic fit ensued without any clonic element.

CASE 37. **Spreading Focal Sensory and Motor Attacks sometimes Proceeding to Grand Mal**

A left frontal to posterior parietal brain wound soon after wounding caused right hemiparesis with right-sided sensory loss to all modalities and global dysphasia. Some years later there was a very slight spastic weakness of the right hand, but no definite sensory loss. Slight dysphasia occurred in emotional situations.

Fits.—**1.** A sudden numbness of the right hand spread up the arm to the shoulder, then the leg and jaw started jerking, and consciousness was lost with a generalized convulsion.

2. Jerking of the right face, and then both legs, was continuous for about four hours and then ceased abruptly. This was observed only in the early fits.

However, the march of events in the spread of an attack seems at times to jump the expected anatomical sequence as in Case 77 (above). The variation

which may occur in the Jacksonian march presumably depends on the continual changes in the setting of the motor cortex.

Multiple Forms of Aura in One Case

In some cases of small Rolandic wounds two or more types of fit may occur. A fit must originate in relatively normal cells adjacent to the traumatic lesion, and the appearance of more than one type of fit seems at first sight to indicate the development of more than one discharging focus around the scar. However, it may be due to different responses from the same focus as the intensity or some other quality of the discharge changes, just as the neurophysiologist can elicit a variety of motor response from a given cortical point by changing the conditions of the experiment (Liddell and Phillips, 1950).

These points are demonstrated in the following case, and in Cases 4 and 32 among others.

CASE 53. **Focal Fits with Varying Sensory and Clonic Motor Onset. Adversive Onset to Grand Mal Attack**

A left posterior parietal brain wound early after wounding caused a right lower quadrantic homonymous field defect. The right arm was weak and atonic with loss of the appreciation of passive movement. The right face was also weak. Some years after wounding the right quadrantic field defect remained together with slight reduction of passive movement sense and stereognosis in the right hand.

Fits.—**1.** Suddenly a spot as of reflected light appeared in front of the eyes, which was difficult to localize in the visual field. The right arm then felt as if it were moving below the elbow, and the triceps region felt as if the muscle were being torn, though this was not described as painful. There was no objective movement, nor loss of consciousness.

2. A sudden feeling of numbness in the right hand was followed immediately by twitching of the right hand, but no tonic movement.

3. A generalized convulsion started with adversive movements of the head to the left. During the generalized convulsion movements occurred more on the right than on the left side.

Epilepsia Partialis Continua

Partial continuous epilepsy seems to occupy a special position amongst the motor auras, particularly so far as the spread of the epileptic process is concerned. It has been pointed out that nothing comparable seems to occur in the sensory sphere. Moreover, it tends to occur mainly in the first few weeks after wounding when the patho-physiology of the cortex must be quite different from the more chronic condition of some years later. The tendency to continued clonic movement which neither spreads nor ceases would suggest perhaps the activity of a region of cortex relatively isolated so far as its afferent and transcortical connexions are concerned. The individual focal clonic attack—and for that matter the isolated grand mal attack also—is presumably terminated in such a positive manner that the discharging focus is rendered refractory for a considerable time. The following cases (and also No. 79) illustrate this type of attack.

CASE 33. **Partial Continuous Epilepsy of Foot**

A right frontal wound with a foreign body passing to the right occipital lobe subsequently became septic and caused death in three weeks. Soon after wounding there was marked left hemiparesis, stupor, and dysphasia. The right hand showed continual groping and grasping movements.

Fits.—Early after wounding there were clonic movements in the right foot and ankle, which passed up the leg, and then to the shoulder, the sternomastoids, and down to the triceps muscles, but no further. The movements were continued for 10 days uninterruptedly. In addition to this, during the period of continuous movement, there were occasional isolated twitches of the right face and thumb.

CASE 68. **Partial Continuous Epilepsy of Foot. Focal Adversive and Tonic Fits.**

A right fronto-parietal brain wound (through and through) soon after wounding caused left hemiplegia with reduced tone. Passive movement sense and light touch were abolished in the left hand and foot. Some years later there was a slight spastic hemiparesis, more marked in the arm than in the leg, but no sensory changes.

Fits.—**1.** Early after wounding rhythmic contractions of the medial plantar and posterior tibial and peroneal muscles of the left foot at a rate of about 6 per second were maintained uninterruptedly for four days. Movements were sudden isolated contractions with slower relaxation.

2. A feeling came over the patient that he had to look at the left hand. Then the eyes and the head started moving slowly round to the left. Consciousness was fully preserved, but he had to remain still till the forced turning ended " in a few minutes ". There was no clonic element.

" Focal status ", as it is sometimes called, should be sharply differentiated from generalized status epilepticus. The latter is a medical emergency calling for urgent treatment to arrest the attacks, which may otherwise rapidly exhaust the patient and lead to death. The former seems to incommode the patient little, and is, in fact, so resistant to the usual anticonvulsants that determined endeavours to end it by these means may do more harm than

good. A type of continuing focal discharge may occasionally be seen during a grand mal status. Here a continuous local clonic discharge occurs between each grand mal attack. However, the clinical site of discharge in this type of case is not constant, but flits from group to group of muscles, and the impression is gained that this represents a reduced continuation of the grand mal seizures, and that the physiological background of this and true partial continuous epilepsy is different. Cases 63 and 52 illustrate this.

CASE 63. **Status Epilepticus with Focal Clonic Movements between Fits. Focal, Clonic Attacks in Left Limbs**

A left parieto-occipital parasagittal brain wound early after wounding caused global dysphasia, dysgraphia, and dyslexia, with a right hemianopia and some defect in the left lower visual field also. Some years later there was receptive and expressive dysphasia which was much worse when the patient was emotionally upset. There was dyscalculia and dysgraphia, but he could read slowly and write accurately with correction. A right hemianopia with a left lower quadrant defect was also present.

Fits.—**1.** There was status epilepticus with generalized tonic stiffening but clonic movements mainly on the left side, though the head and neck were involved as well. In the intervals between the more generalized clonic movements there was a sort of intermittent partial continuous epilepsy, sometimes in the left hand, sometimes in the face—just a continuous clonic flickering of a few muscles.

2. A sudden twitching of the left face passed to the left arm with no tonic movement. Sometimes twitching began in the hand and sometimes in the left leg, and then passed to the arm and face. After this there was residual weakness in the left arm for half an hour or so.

3. The pattern of the fit was as in 1, but consciousness was then lost, and continuous clonic movements confined to the left side occurred with occasional brief spreads to the right arm also.

4. On one occasion only a sudden loss of speech was followed at once by complete motor loss with falling. There was no movement and no loss of consciousness.

CASE 52. **Generalized Grand Mal with Continuing Focal Clonic Movements between Consecutive Attacks**

A right frontal wound soon after wounding caused left facial weakness. Some years later there were no abnormalities in the central nervous system.

Fits.—**1.** There was occasional serial epilepsy with some hours' interval between each attack. Sometimes between attacks focal clonic movements would occur, shifting from the right face to the right arm or to the eyes. In these consciousness was preserved.

2. Generalized grand mal attacks occurred with no aura.

Methods of Arresting Focal Fits

It is often thought that a " busy " area of cortex is less likely to discharge spontaneously, and in this connexion it is interesting to note that patients sometimes discover a method by which application of a peripheral stimulus to the affected part will arrest a clonic motor attack. The manoeuvre usually involves forcible tension applied to the muscles involved or attempted strong voluntary movement of them. Cases 6, 66, and 69 illustrate this. Penfield and Kristiansen (1951) have observed in a case of focal status affecting one foot that electrical stimulation of the foot area of the motor cortex abolished the attack. Possibly the strong voluntary effort or stimulus described above has a similar effect in forcing through an afferent stimulus to a relatively isolated area of cortex. Indeed these phenomena may be considered as evidence in favour of the view that focal fits develop in areas of the cortex which have been deprived of some of their afferent connexions. No case was recorded where a sensory fit was so arrested, though patients did sometimes attempt this by rubbing or moving the affected part.

FURTHER DETAILS OF VARIETIES OF MOTOR AND SENSORY ATTACKS

Myoclonic Jerks and " Electric Shock " Sensations

The myoclonic jerk may also occur as a variety of motor aura, though it is infrequent in this series (Table XI). Such an attack seems similar to the normal start reflex, and in fact in one case it occurred as a local aura in one limb only, in response to sudden noise. It was, however, more than a simple " start ", as the movement would sometimes develop into a short clonic attack. This motor aura seems paralleled on the sensory side by the sudden feeling as of an electric shock. This may be purely sensory, or it may be associated with a few clonic jerks in the part affected (Table XI). The rapidity of the phenomenon and the fact that it may involve the whole of a limb almost instantaneously suggest that it may represent some " short circuiting " process in the central nervous system, by which pathways become momentarily available for discharge. The following two cases illustrate both the pure sensory shock and the mixed motor-sensory type. Case 77 above also brings this point out well.

CASE 18. **Focal " Electric Shock " Feelings possibly Spreading to Focal Clonic Movements and Grand Mal**

The patient received a right Rolandic wound. Some years later there was slight spasticity of the left upper

limb, with cortical sensory loss in the left hand, more marked on the ulnar than the radial border.

Fits.—**1.** A sudden feeling like an electric shock, which ran round to the back of the head, occurred in the left neck : nothing further.

2. A sudden brief feeling like an electric shock occurred simultaneously in the hand and foot : nothing further.

3. A sudden, stinging feeling like pins and needles in the left hand passed rapidly up to the shoulder. Then a similar feeling appeared in the left leg, which ran up the leg to the side, the abdomen, and then the loin. Twitching then began in the left face around the eye. A choking sensation occurred in the throat and consciousness was lost, and left-sided convulsions followed.

4. There was a sudden feeling of being unable to breathe properly. It was momentary only, and always occurred just at the time of falling asleep.

CASE 38. **Focal " Electric Shock Feeling " with Clonic Fits**

A right posterior parietal to frontal brain wound early after wounding caused left hemiplegia with sensory loss and homonymous hemianopia. Some years later there was a spastic hemiparesis, more marked in the arm than in the leg, and homonymous hemianopia. All forms of sensation were impaired, in the arm more than in the leg. This patient developed a schizophrenic reaction with paranoid features.

Fits. **1.** A sudden feeling like an electric shock in the left arm passed rapidly up to the left face, when twitching of the face occurred. There was no further progress and no loss of consciousness.

2. The left hand was suddenly clenched ; then clonic jerking occurred, followed by loss of consciousness and general convulsions.

Motor and Sensory Inhibitory Attacks

The inhibitory attacks require special mention. It has been suggested that they represent a discharge through a cortical inhibitory mechanism. As has been mentioned earlier, they tend to involve a larger area of the body than the focal clonic or positive sensory attacks, and this is in keeping with the physiological observation that stimulation of the suppressor areas in monkeys tends to produce widespread rather than localized motor inhibition. Both motor and sensory types of attack are observed, though in some cases the two seem closely associated in a given attack. Examples of motor inhibition are seen in Case 9 (above) which illustrates the widespread involvement first of the whole upper limb and rapidly thereafter of the leg also, and in Case 55 where both legs are involved.

CASE 55. **Focal Inhibitory and Focal Thermal Fits**

A right posterior parietal parasagittal wound soon after wounding caused weakness of both legs, the right more than the left. There was a slight sensory loss of cortical type in the right foot. The bladder was distended, and urination was difficult. Some years later there were no abnormal signs in the central nervous system.

Fits. **1.** There was sudden loss of power in the legs but no sensory change was recalled. The patient fell, and there was a momentary loss of consciousness.

2. A queer feeling occurred in the epigastrium. Then came a hot, burning feeling in the left hand, which spread rapidly up the arm to the shoulder. The attack then ceased. The sensation was one of heat but was not painful.

A focal aura of numbness probably represents a sensory inhibitory attack. Though the subjects' powers of accurate description vary, there seems to be a well maintained differentiation of these types of aura from the more positive pins and needles or tingling. In Case 3 (above) the patient described quite separately a " numb feeling " in some attacks and a tingling feeling in others, the former having a distinctive negative quality. Again in Case 40, widespread " numbness " in the whole of one side is distinguished from " pins and needles ", which have a more focal reference. This may also be distinguished from agnosia which is referred to later.

CASE 40. **Focal Sensory Attacks sometimes Inhibitory and sometimes " Positive " with Focal Motor Element**

A right fronto-temporal brain wound soon after wounding caused some weakness with marked sensory change in the left hand. Any stimulus to the hand caused a strange, spreading feeling of an unpleasant character. Pin-prick was appreciated but diffusely, and there was marked cortical sensory loss. Some years later the left hand was a little weak and awkward to use, and always felt colder than the right. However, two-point touch and passive movement sense were well preserved, though stereognosis was reduced. There was an upper quadrant left field defect.

Fits.—**1.** A sudden, giddy feeling with numbness down the whole of the left side was usually followed by local jerking of the left arm and leg.

2. A sudden pins-and-needles feeling in the left arm almost immediately became a severe, burning sensation, which was very painful. This passed up the left arm, and then involved the left trunk, and sometimes also the left leg. The sensation of heat passed in a few seconds, leaving an unpleasant cold feeling in the same situation.

In these cases motor or sensory inhibitions are separate, but in Case 22 the two appear together again with a wide involvement of the whole upper limb. While in Case 829 sensory inhibition leads on to motor, and the process spreads rapidly to the whole of one side.

CASE 22. **Sensory and Motor Focal Inhibitory Attacks**

A right Rolandic brain wound soon after wounding caused weakness with sensory change in the left hand. Light touch and pin-prick gave a tingling, widely radiating response; though touch was fairly well localized, passive movement sense was reduced, as were stereognosis and two-point touch. Some years later there was slight cortical sensory loss in the left hand, mainly on the ulnar border.

Fits.—The left hand suddenly felt dead and numb. There was no tingling associated with this; the sensation seemed to be a negative one. At the same time attempts to use the hand were unavailing, and no movement occurred.

CASE 829 (not in tables). **Focal Sensory and Motor Inhibitory Attacks followed by Focal Clonic Movements and sometimes by Loss of Consciousness**

A left frontal brain wound (Fig. 4) soon after wounding caused global aphasia, with some weakness of the right arm and loss of facility of movement. No sensory changes were found. Some years later there was still slight expressive dysphasia, especially when the patient was emotionally upset. There was a slight increase in tone in the right hand but no other abnormalities.

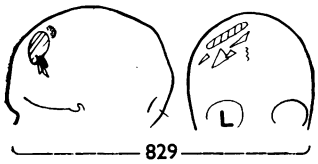


FIG. 4.—Skull tracing of Case 829.

Fits.—**1.** There was a sudden feeling in the right arm, difficult to describe, but as if the arm were weak and nothing could be felt in it. This was accompanied almost immediately by an actual loss of power, so that anything held in the hand would be dropped, and the arm would fall to the side. A few seconds later the leg would also be involved; first the feeling, then loss of power, so that the patient would fall to the right side. At this stage there were usually, but not always, a few clonic jerks of the right arm, and less so of the right leg. The attack then ceased.

2. As above, but on falling to the ground there was loss of consciousness with convulsions, involving mainly the right side.

Auras of Agnosia and Phantom Sensations

A further and most interesting example of sensory inhibition is provided by the auras of focal agnosia. Here patients describe the sudden feeling that some part of the body is no longer there, and they may look at the continuing physical reality of the part with surprise. Here again, the areas of the body involved are usually fairly large as in other inhibitory attacks. In Cases 10 and 36 (see above) the arm suddenly seemed to disappear, to be followed shortly by the leg. In Case 84 the attack involved half of the face and the arm and leg, while in Case 59 only the arm was involved and in Case 21 (above) only one half of the face. In these attacks, although power is preserved in the limb, there may be some disability and clumsiness of movement.

These attacks presumably represent a discharge affecting the body image mechanism in the posterior parietal lobe, and the disposition of wounds in such cases on the whole supports this localization. Contrasted with these negative phenomena, but still involving areas concerned with organization of the body image, are the auras of phantom sensation. In Case 58 the aura took the form of a highly organized phantom arm. In Case 76 the aura was more limited in extent involving a sensation of movement in fingers which were, in fact, still, while in Case 53 (see above) the aura was even more limited and bizarre.

CASE 58. **Focal Attack of Phantom Limb. Grand Mal with no Aura**

A left posterior parietal brain wound soon after wounding caused a right homonymous field defect with sensory loss in the right hand—reduced passive movement sense, two-point touch, and stereognosis. Pin-prick was appreciated, but was radiating and intense. The reflexes of the right arm and leg were increased compared with the left. Some years later there was a right homonymous lower quadrant field defect. Stationary objects would be missed from this field, though appreciated when moving. Localization of objects in the right field was also reduced. There was occasional attention defect in the somatic sensory sphere also with astereognosis in the right arm. Tone in the right limbs was slightly increased, and there were bilateral equivocal plantar responses.

Fits.—**1.** A sudden feeling occurred that the right arm was in a position elevated above the head with the hand clenched and the elbow semi-flexed. The patient looked up in this direction to find the hand was not there but by his side. The feeling might be so strong that he would, in bed, ask his wife to pull the arm down, though in fact it was still by his side.

Sometimes, but rarely, there is a slight feeling of pins and needles in the phantom arm in the raised position.

CASE 76. **Focal Fits of "Phantom Movement". Visual Aura with Sense of Familiarity followed by Occasional Loss of Consciousness**

A right temporo-parietal brain wound soon after wounding caused left hemiplegia with left sensory loss to all modalities and hemianopia.

Some years later there was a left hemiparesis, more marked in the arm than in the leg. There was reduced appreciation to light touch and passive movement sense in the left hand. Hot and cold were appreciated but pin-prick was changed and radiating. These changes were present but less marked in the leg. There was a

complete left homonymous hemianopia and also some difficulty in appreciating spatial relations.

Fits.—**1.** A sudden feeling occurred as if the fingers in the left hand were moving, clenching, and unclenching, though in fact no movement was occurring.

2. As above, then there was actual stiffening of the hand in a clenched position with some flexion at the elbow, followed by clonic movements at the elbow.

3. The above phenomena were followed by a curtain suddenly appearing over the normal half visual field. This was accompanied by a picture of something which could not be described, but was always the same. This might proceed to complete loss of consciousness, but without further motor phenomena.

Painful and Thermal Auras

Two other sensory auras remain to be mentioned, both of them of special interest to cerebral physiology. These are concerned with sensations of pain and temperature.

In four cases pain was mentioned as some part of an aura. In Case 20 (see above) this was a severe and intensely unpleasant, rather indefinable sensation which occurred at the onset and was the main part of the aura. In Case 7 (below) pain was more clearly defined and described as like a painful spasm, though no actual muscular spasm occurred, while in Case 73 pain was actually associated with tonic spasm. In Case 74 pain followed a clonic motor aura and was again severe and hard to describe—it showed a spread from the abdomen to the thorax and then to the neck on one side—following the march of the preceding clonic movement.

CASE 7. **Focal Aura of Pain with Clonic Element**

A right anterior parietal parasagittal brain wound soon after wounding caused complete motor aphasia and left hemiplegia with some sensory loss in the left hand. Some years later there was a spastic weakness of the left arm and less so of the left leg. There was some hyperpathia in the left hand, and joint sense and two-point touch were reduced.

Fits.—**1.** A painful feeling as of cramp occurred in the left face, though there was no noticeable stiffening or movement. This was followed by painless twitching of the face. Directly after this there was twitching of the left leg, and then of the shoulder but not of the arm, with no further spread.

2. As in 1 but afterwards there was spread of movement to the leg, consciousness was lost, and the patient fell, but no generalized convulsion occurred.

3. There was sudden stiffening of the left leg with painful cramp, then clonic movements of the leg, but no further spread.

No fewer than 11 patients mentioned definite thermal sensations as part of an aura. Of these nine had sensations of heat or warmth, at times so intense as to be very painful. Two had feelings of cold, and in one temperature change was so intense that it was impossible for the patient to decide whether it was hot or cold. In 10 cases auras were referred to the arm, and in one the face was involved. Cases 4, 8, 21, 39, 40, 54, and 55, have already been referred to above. The following three cases also illustrate this aura :

CASE 30. **Focal Sensory Attacks, Tingling and Thermal. Adversive Aura to Grand Mal**

A right parietal wound soon after wounding caused slight left hemiparesis with sensory loss in the hand and hemianopia. Some years later there was no discernible motor abnormality but some reduction of temperature appreciation in the left arm and leg and the hemianopia persisted.

Fits.—**1.** Tingling in the left hand passed up the arm to the face, and then involved the leg. This was followed almost immediately by a feeling of intense heat running up the arm and at the same time in the leg. Then the attack ceased.

2. Sudden pins and needles occurred in the left face—nothing more.

3. The head and eyes were slowly turned to the left, consciousness was then lost, and a grand mal convulsion ensued.

CASE 42. **Focal Fits with Aphasia, Thermal, Sensory and Clonic Aura in Right Hand and Possible Loss of Consciousness**

A left fronto-temporal brain wound some years after wounding caused a right homonymous lower quadrantic field defect, dysphasia, dyslexia, and dysgraphia, with loss of dexterity in movements of the right hand, though little actual weakness.

Fits.—**1.** There was sudden loss of speech for a few seconds, then loss of consciousness with a generalized convulsion.

2. Sometimes after the speechlessness and before loss of consciousness a hot feeling occurred in the right hand which spread up the arm to the shoulder and was followed at once by clonic movements in the hand and forearm.

3. Sometimes attacks occurred as in 2, but there was loss of consciousness after the clonic movement.

CASE 44. **Thermal, Sensory, and Clonic Focal Fits. Grand Mal with Aura of Focal Clonic Movements**

A left frontal to temporal brain wound was sustained, one foreign body having passed down to the temporal fossa. Early after wounding there was aphasia, motor more than sensory, with right hemiplegia. Some years

later the patient still had marked motor aphasia, and the right hand showed ataxia, but with little actual weakness. The leg was hypertonic, but power and movements were well preserved. He could now draw and write well with his left hand. On the sensory side, pin-prick and temperature appreciation were reduced, though not abolished, in the right hand. Two-point touch and stereognosis were also reduced in this limb. The leg showed no sensory defect.

Fits.—**1.** A sudden burning sensation occurred in the right hand running up the arm. This feeling was intense and unpleasant, and the patient rushed to put the arm under a cold water tap as this helped.

2. Sometimes this sensation was followed immediately by local jerking of the right hand and arm.

3. Sudden local twitching of the fourth and fifth fingers of the right hand, which travelled up the arm, occurred. Consciousness was lost and a generalized convulsion ensued.

It may be seen from these examples that thermal sensations can occur at the onset of an aura or at the end. Although a feeling of heat is the usual form, in two cases hot and cold feelings occurred in the same attack. In two also there was a change of aura from pins and needles to heat during an attack. This group also shows some of the rare cases in which a motor aura proceeds to a sensory, and both clonic and tonic movement immediately preceding the temperature changes have been recorded. The anatomical implications from the study of these cases is considered later.

ANATOMICAL CONSIDERATIONS

Extent of Wound and Clinical Deficit in Relation to Fit

The site of wounding and the type of sensori-motor deficit clearly play an important part in determining the type of fit which will occur spontaneously. An abnormal discharge can only act through a functioning cell so that a fit can only originate in, say, the motor cortex if a part at least of that cortex is functioning.

In this connexion the occurrence of clonic focal auras appears to be far more frequent in the small Rolandic wounds where permanent sensori-motor deficits are not extensive than in those cases where wounds are larger and permanent severe hemiplegias result. This lends some support to the general view, derived in part from experimental studies of the fit pattern after cortical ablation, that the cortex is specifically concerned with clonic movements. In Table VII cases with severe residual hemiplegia are separated from those with less extensive signs, and the more frequent occurrence of clonic focal seizures and their onset relatively early (under one month) after wounding in the latter group is clearly shown. Further, among the 52 cases with focal clonic fits, there were 14 which had *only* focal attacks on a five-year follow up, while the remaining 38 cases had also developed general convulsions on one or more occasions. Of the 14 cases with focal clonic fits alone (at the five-year follow-up) only three had a permanent severe hemiplegia. The remaining 11 had less paralysis, and in eight of these 11 cases the focal fits began within a month of wounding.

It seems therefore that the focal fits which remain focal are more likely to be those which follow the less severe wounds of the sensori-motor cortex, and these are also more likely to develop very soon after wounding.

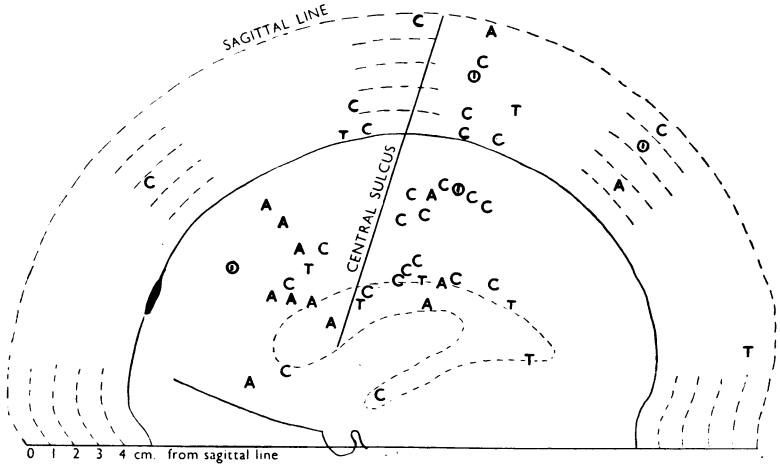
A great variety of sensory and motor auras is observed in the small wounds included in this series, and we can conclude that many of these can only appear when certain parts of the sensori-motor cortex are intact.

Many of the cases of hemiplegia in groups 6 and 7 of Table IX must have had most of both the sensory and motor cortex destroyed. In contrast to the smaller wounds these cases show few in which there are focal clonic motor auras, while the tonic type of motor aura is the more common (Table X). Whether this tonic discharge originates in other parts of the cortex, such as the pre-motor region or in deeper motor centres, cannot, however, be determined from this material, though several of the adversive auras with their strongly tonic setting clearly originate from the pre-motor region (Fig. 5).

Another and most striking feature of the hemiplegic cases is that sensory auras are very uncommon (Table X). It seems reasonable to associate this with disappearance of the sensory cortex, and if this is a correct assumption, as in most of these cases the thalamus cannot have been directly injured, it may be concluded that sensory discharges require integrity of the post-central gyrus for them to occur, or at any rate for them to be appreciated in consciousness.

Site of Wound and Type of Fit

The effects of electrical stimulation of the motor cortex suggest that the pre-central gyrus is the site of origin for most cases of focal clonic fits, but we have little information as to whether the spontaneous occurrences of these focal fits represent an irritative or a release phenomenon. The common appearance of a considerable latent interval between injury and the onset of fits in traumatic epilepsy is perhaps more in favour of an area of cortex gradu-



FIGS. 5 and 6.—Charts to show the site of skull penetration of a series of wounds causing focal motor (Fig. 5) and sensory (Fig. 6) auras. Cases of severe hemiplegia are excluded. Wounds lying within 5 cm. of the sagittal line are charted in the halo. Method of charting is as described by Russell (1947).

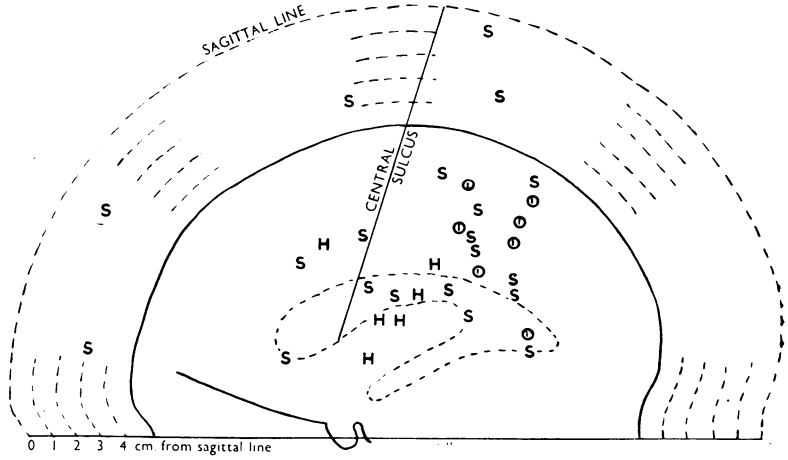
Types of aura: A = adversive, C = clonic, T = tonic, (I) = inhibitory (motor in Fig. 5, sensory in Fig. 6), S = sensory positive, H = aura of heat or cold.

ally becoming over-active through absence of control or through physical isolation, and with this in mind it is by no means impossible that damage to the *sensory* cortex plays an important part in the development of focal clonic *motor* discharges. In Fig. 5 the site of penetration of wounds (not causing severe hemiplegia) but which were followed by motor fits is charted, and it is evident that most of these points for cases with clonic fits lie behind the central sulcus. From this it is at least evident that the post-central gyrus is injured in most cases of focal clonic fits, an observation which is, in general, confirmed by reference to individual cases in Table IX.

Adversive attacks have already been referred to as often originating around posterior frontal wounds (Fig. 5). It may be noted, however, that they may occur in cases of severe hemiplegia and thus seem to be independent of the Rolandic region.

The anatomical aspects of the various sensory auras present difficulties. The main somatic sensory projection reaches the post-central gyrus via the thalamus, and circuits back to the thalamus. But what then? Presumably there is a complex "development" of what arrives at the cortex in the adjacent more posterior parietal lobe, but at what stage does the sensation result in the mental recognition and localization of a sensation?

The position of penetration of small wounds causing positive or negative (inhibitory) sensations is charted in Fig. 6. These wounds are, in general, a little more posterior than those which cause clonic focal fits, but we can say little more. Both types of sensory aura often spread to focal clonic movements (Table XI), and this probably provides the best evidence that the post-central gyrus is concerned, but from Table XI it is seen that all forms of sensory aura may spread to clonic movements except the phantom and agnosia sensation.



Most of the sensory auras described are so precisely localized that the body image mechanism must be intact even though cortical sensation, by which it is largely "fed", is disrupted. This mechanism correlates the somatic sensory and visual plan of the body in relation to the environment, and

the several cases with auras of agnosia have sustained injury involving the posterior parietal lobe, which is the generally accepted anatomical site for this function. Case 59 is a good example, and the subject will be considered again in the next part of this study.

The description of an aura of agnosia indicates the disappearance of a sense of body existence and position which is at other times preserved. Being inhibitory without any positive accompaniments, an aura of agnosia probably originates in the midst of the body image mechanism. In three instances the sensation was self-limited (Table IX), in one it passed to a visual aura, in one to an inhibitory motor episode, and in one to a tonic adversive fit, but in no instance did it pass to a positive sensory experience.

A phantom sensation suggests a positive stimulus to an intact body image mechanism, but in no instance was there a record of phantom spreading to agnosia or the reverse. Indeed, neither of these phenomena was associated with other sensory attacks of any kind.

The four cases which show an aura of local pain are of great interest. Two of them (Nos. 7 and 20) had small wounds near the Rolandic fissure with a part of the sensory cortex clearly intact, and may thus confirm the recent evidence that the sensory cortex may, in some cases, play an important part in the appreciation of pain (Marshall, 1951).

In both these cases the pain occurred as the initial part of an aura. In the other two cases (Nos. 73 and 74), the injury was more extensive but was so situated that a part of the sensory cortex was also probably intact.

Sensations of heat or cold form a remarkable group, and must be studied from the point of view that they might throw light on the cerebral mechanisms concerned with temperature sense, at present little known. The 11 cases concerned were wounds associated with little sensory loss or motor disorder, and there is certainly no evidence of deep cerebral injury being a feature of these cases. The site of skull penetration in six of these, where the wound was relatively small, is shown in Fig. 6. From Table XI certain special associations emerge. In the first place two cases with a positive sensory aura spread to a thermal sensation, and these provided the only examples of spread from one type of sensory aura to another. The other remarkable feature is that thermal sensations formed four out of the only six examples of motor to sensory spread. These sensations therefore of hot and cold seem to form a unique link between clonic movements and pins and needles, or more strictly between the motor and sensory cortex from which these auras respectively originate. The apparent grouping of the wounds around the lateral fissure raises the possibility that the so-called " secondary sensory area " is concerned. The tendency for these auras to spread to other sensory auras and to be initiated by motor auras would also suggest a less discriminative and more " primitive " level of firing, which might also be in keeping with what is known of these secondary areas.

The inhibitory auras are represented by paralysis, numbness, and agnosia. The site of wounding in some of these cases is shown in Figs. 5 and 6, but this indicates no obvious anatomical distinction from the site of wounding in cases with positive sensory and motor attacks.

Table XI indicates that an aura of focal paralysis never passed to another kind of aura, but was either self-limited or proceeded to loss of consciousness. Auras of numbness on three occasions, and an aura of agnosia in one case did pass to focal paralysis. These were the only examples of a sensory aura proceeding to an inhibitory motor episode, and they clearly suggest a linkage between these various inhibitory forms of attack.

DISCUSSION

The present study is concerned with the analysis of the various features observed in spontaneously occurring focal fits following wounds of the sensorimotor cortex. Aphasic features are not considered here, as they are studied in a later paper. The observations reported are in some ways complementary to the careful observations on cortical stimulation by Foerster, Penfield, and others during the past half century. The variety of sensation, movement, and inhibition is as great in the spontaneously occurring fit as in those obtained by cortical stimulation, while the spread of the cortical discharge from one sensation to another may be more vividly described than can be hoped for by the patient on the operating table. Not that these spontaneously occurring epileptic discharges can be interpreted with any confidence, but their variety provides many arresting glimpses into the complexity of cerebral function.

Considering only marked and clearly defined differences in type of aura, it would seem (Table X) that at least five motor and seven sensory varieties can occur. On the motor side, although adversive attacks are to some extent only special examples of tonic movements, they form such a clear cut clinical entity, as well as often having a distinctive anatomical background, that they are considered as a separate group. On the sensory side, tingling and

pins and needles are considered together because their distinction was often difficult for the patient. However, in some cases their different qualities were clearly appreciated, and it is possible that they should be regarded as two varieties of response. Indeed, if we consider the minor differences in both the sensory and motor content and in the whole setting and sequence of the attacks—features which do, in fact, give them an individual " flavour "—then the impression of great variety of response from the sensori-motor cortex is enhanced. This is in keeping with Penfield and Kristiansen's (1951) findings from stimulation of the human cortex in which they mention no less than 21 different kinds of sensory aura. The occurrence of auras of pain, heat, or cold, in cases of small cortical wounds, is of special interest from the point of view of localization of function in the brain. The rarity of painful auras in our series is also reflected in results from cortical stimulation (Penfield and Kristiansen). Only two were recorded in all their series, and as these were usually associated with motor responses, they tentatively explained them as simply secondary results of muscle spasm. We have given reasons above for suggesting that this cannot be the explanation in all our own cases. Moreover, Lewin and Phillips (1952), among others, have recently reproduced the severe pain of an epileptic aura by stimulating electrically the post-central gyrus under local anaesthesia (Case 20). They have also abolished or greatly modified spontaneously occurring pain of an intractable type by excising the area of post-central gyrus from which the pain was produced on stimulation. So far as thermal auras are concerned, although they occurred in four cases as part of a spontaneous fit in Penfield's series, in no case were they reproduced by cortical stimulation. This might be taken as an argument in favour of their non-cortical origin. However, in all our cases showing these auras, the evidence for a cortical origin of the fits is quite as strong as in the examples of the many other forms of sensory and motor attacks which we have recorded. The patients with a thermal aura had often been wounded near the lateral fissure and the possible significance of this has already been considered.

Excitation and inhibition both on the motor and sensory side appear to be clearly represented in the cortical responses we have studied, and the way in which the very various auras may spread one to another has proved of special interest.

Although broad anatomical divisions of the sensori-motor cortex do appear to be maintained for the different qualities and bodily sites of epileptic response, our findings do not suggest that these can be rigidly determined by this form of study. The impression is one of flexibility of cortical response. Such a view is in agreement both with the general trend of present neurophysiological work, and with the results of cortical stimulation in man where pre- or post-central sites may each give rise to sensory or motor responses.

SUMMARY

Eighty-five cases of focal motor and somatic sensory fits are studied.

Five motor and seven sensory varieties are distinguished.

The relation of the site of injury, and the sensori-motor deficit to these auras is considered.

The direction of spread from one type of aura to another is considered.

The anatomical and physiological implications are discussed.

Acknowledgments will be made in a later section of this study.

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