INHIBITORY EPILEPSY.

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The fact that many of the phenomena of what is ordinarily understood as epilepsy are inhibitory and not excitomotor in character has scarcely received the attention it deserves. Yet the clinical neurologist who gives some consideration to the question cannot fail to be impressed with the comparative frequency, in epileptic cases, of symptoms not to be explained physiologically except by some process of inhibition. Loss of consciousness—what else is it than arrest of function, indeterminate though its mechanism may still to some extent be? Perhaps the most typical feature of the average case of petit mal is cessation of function of one or other form. The patient stops what he is doing, stares, drops what is in the hand, ceases speaking, falls, sits still and motionless, ignores questions or does not hear them, and so on—whatever the precise formula in a given case the major portion of the syndrome consists patently in the expression of some variety of inhibition. Similarly, in many severe fits the initial stages illustrate the action of the same physiological mechanism, and so do certain phenomena to be noted occasionally at a later period. For example, loss of sight—darkness, blindness—is not infrequent as an aura; loss of hearing, though less common, occurs similarly now and then; loss of speech, too, may develop at the stage of prelude. Initial muscular relaxation also deserves mention. Not a few post-convulsive symptoms usually attributed to exhaustion are better regarded as exemplifying inhibition.

Of no less interest is the question of stimuli of one or other sensory kind being succeeded not by motor excitation but by motor inhibition. Gowers is one of the very few writers who have alluded to this possibility. He was of the opinion that weakness associated with epileptoid seizures may in minor attacks be due to inhibition of the same centres as are discharged in severer attacks, and mentioned a case of Jacksonian epilepsy (typical, right-sided) in which minor fits consisted of inability to speak or move the right arm or leg, there being no convulsion. More significant, for the purpose of this communication, are two other cases referred to briefly by the same author. The first is that of a woman of 40, probably the subject of organic disease, whose attacks are thus described.

> "They begin with 'ticking in the right ear,' and this is followed by a painful sensation 'like hot needles running into the skin,' which passes down the side to the leg and foot and, after reaching the toes, returns up the leg and side and is felt in the arm, hand, and tongue. She mumbles and cannot speak for half an hour. There is no motor spasm, but as

soon as the sensation is felt in the leg and arm the side becomes extremely weak, so that, although ordinarily able to walk well, she becomes unable to stand and scarcely able to raise the arm."

The second case, reported with greater brevity, is that of a patient of 48; the attacks consisted in "pins and needles" in the left thumb, which passed up the arm to the shoulder and left scapula, with a sense of contraction in the arm. This became powerless, so that she dropped anything that was in the hand.

These and analogous cases may be taken to illustrate restraint or inhibition of motor centres by epileptic sensory discharges, a phenomenon which, as will be shown later, can be substantiated by other data well known to the physiologist.

A remarkable example of the same epileptic syndrome was under my close observation for a number of weeks, and is here narrated in detail.

PERSONAL CASE.

W.C., male, age 24, unmarried. Occupation, barman.

Family History: Mother, who died at age of 39, from heart disease, suffered from "fainting fits." No record of epilepsy or insanity in near or distant relatives.

Previous history: Always has had good health. Denies venereal infection. Is abstemious in regard to alcohol and has never been the worse of liquor. No rheumatism or rheumatic fever.

History of present illness: Six months ago, without any apparent cause, the patient began to suffer from curious attacks of tingling in the left hand and arm, and left side of the body, which have continued steadily and are now becoming more frequent. At first these occurred perhaps one or two in a fortnight, but now every two or three days. They may come at any time, but as far as he knows not during the night. In the attacks he never loses consciousness but he feels queer and "regular knocked."

Aura: Of this a very definite description is given, and it has always been the same. It begins by his eyes becoming "misty," not one eye more than the other, or before the other. He can see his way about but he cannot distinguish the forms of objects. He has noticed that the mistiness is chiefly on looking downwards; on looking to either side he sees constant "bright flashing stars" in the outer parts of the field. They are not coloured.

This mistiness of vision and these subjective visual phenomena continue for about 15 to 20 minutes before the attack proper commences.

Character: The attack commences by a sensation of tingling, of formication, of "wires" in the left fingers and hand. This is variously described by patient—it is as if "he had his hand in a stocking"; as if "it were entangled in string." In the earliest attacks the sensation did not pass the wrist, and patient was able to abate it by putting his hand into water, rubbing it, etc. But all the other attacks have been wider in range. The sensation passes up the left arm in a few seconds, then down the left side of trunk rapidly into leg and foot and toes; it then appears (in the more recent attacks) in the left side of the face (lower division). It plays along the lower jaw on the left side, and makes the left half of the tongue tingle.

The duration of the sensation varies from only a few seconds to some minutes. When it is over, it leaves arm, leg and face quite numb and "dead."

At the same time as the numbness appears patient loses all power in his arm and leg. Anything he has in his hand he will let drop; he will be unable to pick anything up with

the fingers. In a second or two the loss of power in the left arm and hand is absolute and he is quite unable to move it. Similarly with the leg, he is completely unable to move it at any joint.

The duration of this combined numbness and loss of power may be merely a minute or two, or as long as 20 minutes.

When the attack passes off recovery of power is usually simultaneous in arm and leg. He thinks there is little if any loss of power in the left face; it feels as though it were being stretched or dragged up, but it is not so powerless as the limbs.

Sequelæ: Usually has headache (temporal and frontal) after the fit, and this may last a whole day. There is no sickness or vomiting.

Patient thinks that the left side, arm especially, is becoming rather weak, but in any case the degree is slight. Otherwise, feels perfectly well.

State on admission: No indication of abnormality in circulatory, respiratory, or alimentary systems.

Cerebral: Attention, memory, intelligence, excellent. Is not conscious of any mental change. Not emotional. Apart from fits, no headache or giddiness.

Speech, taste, smell, hearing, unaffected.

Cranial: No cranial tenderness.

2. V.A. 6/6 R. and L. No restriction of fields, rough test.

O.D.: L, appears normal, though rather high-coloured. R, definitely high-coloured; veins full.

3.4.6. No diplopia, no ptosis, strabismus or nystagmus. No ophthalmoplegia.

Pupils regular, equal, medium size; reactions normal.

5-12. No impairment observed in the function of these nerves. No facial asymmetry or weakness. No defect in phonation, articulation, or deglutition. No defect of palatal movement. Pharyngeal reflex present. No facial hypæthesia or anæsthesia. Tongue protruded straight.

Motor System: General muscular nutrition and development good.

There is some general flabbiness of the muscles more marked in the left arm than in the right. No particular difference in legs.

No incoordination of any kind. No involuntary movements. Patient is right-handed.

There is perhaps just a little relative weakness of the left arm as a whole. No difference in power of legs, which is good.

All voluntary movements good in range.

Sensory System: No objective changes of any kind detected. No astereognosis. No atopognosis. No loss or diminution in sense of position or passive movement. No diminution of muscle pain sense.

Reflexes: Abdominal and epigastric present and equal; brisk. Knee jerks, arm jerks, brisk; no apparent difference between the two sides. No ankle clonus. Double flexor response. No organic reflex impairment.

Description of Fits Observed by Myself.

(1) 12.40 p.m.—When I saw the patient he was lying on his right side, with his legs drawn up, right not so much as left, and with his left arm flexed at the elbow and extended at the wrist. The left foot was distinctly inverted and toes were pointing downwards. He said that at that moment the whole of his left side felt numb, including the lower part of his face and round the nose on the left side. He further declared that the sensation had come on when his legs happened to be drawn up, and that he could not move his left leg. When I tested him, he was unable to move the leg at all, and could move the arm only at the shoulder and elbow, and that very slightly.

Patient had no appearance of distress, no difficulty in speaking, no twitching, and was absolutely conscious and coherent the whole time.

Testing him quickly, it was seen that his arm was rather rigid at the shoulder and elbow, and that the wrist and fingers were quite flaccid. The resistance to passive movement seemed to be due to defective inhibition of antagonists, a phenomenon well seen in the left leg. The leg was resistant to passive movement, more proximally than distally, and immobile. His face on the left side was distinctly affected, especially in the lower division, for both volitional and emotional movements. His lower maxilla was " chattering " and he was unable to control this. He remarked then that his tongue on the left side, and the parts underneath his tongue, were numb.

Tested quickly for sensation, there was anæsthesia of the hand and wrist, and of the leg. The limits were undefined. There was astereognosis in the left hand, and also very considerable diminution of the sense of passive movement and of position. There was very little if any diminution in appreciation of painful stimuli. All the tendon jerks on the left side were exaggerated, but I failed to elicit an extensor response on the left. The abdominal reflexes were diminished on the left.

12.46.—By this time there was some return of movement in the arm, and also in the leg. He could slowly push his leg down, though he could not lift it off the bed. He was able to flex and extend at wrist and could move the fingers slightly. The position of the foot was maintained. In the attempt to move the leg, defective inhibition of antagonists was particularly well seen. Thus as he slowly extended, the hamstrings were seen and felt to contract spasmodically. The same thing was noticed in the gastrocnemius when he tried to dorsiflex the foot.

Spontaneous left patellar clonus commenced at this point, and when the left knee jerk was elicited there was prolonged patellar clonus. Ankle clonus was likewise obtained, but it was not so well sustained. There was a left flexor response at this stage. The numbness was gradually disappearing from leg and face, but it persisted in the arm and hand. When I was testing his sensation for touch, he remarked that the touch of my finger on his leg and arm and hand gave rise to a feeling of pins and needles at the spot touched.

The facial asymmetry was absent now, and his tongue was protruded straight, although it was tremulous. There was no defect of jaw movement. The jaw clonus had disappeared.

The loss of sensation to touch remained, and stereognosis was still inaccurate.

12.55.—In the attempt to close his hand, there was some irregular twitching of the forearm muscles, chiefly of the antagonists to the movement. Diminution of muscle sense was not now so marked. By this time the movements of his legs were normal in range but not of normal strength. He complained that where his limbs had been numb they were now cold, but to my hand there was no actual difference in the temperature of the two sides.

1.02.—Asked to hold his arms out it was seen that the left arm was oscillating very slightly and irregularly, but he was able to hold it as well as the right.

The reflexes on the left side were now distinctly less brisk than a few minutes ago, although patellar clonus was still obtained. On stimulating the sole of the left foot, there was flexion—though very slight—of the great toe, and extension of the small ones. Left abdominal reflex less marked than right.

There was still slight persisting anæsthesia of the fingers, left, but not elsewhere.

1.08.—All movements on the left were now good in range, but there was slight weakness generally at all joints. The anæsthesia had disappeared.

(2) *Patient again seen in a fit one week later.*—When I saw him, he was lying on his bed with his clothes on. He was perfectly conscious, a little pale, and his lower jaw was chattering incessantly. His left arm and leg were helpless. The arm was flexed at the elbow, and lying across his body. On request, he was unable to lift the hand or arm off his body. Neither limb was at all stiff or rigid; on the contrary they were completely flaccid. He

complained that all the feeling had left the limbs on that side, and that the numbness was extending to the left side of his face. The numbness and coldness began in the fingers and passed up the arm and down the trunk to the leg.

On the left side the jerks were unusually brisk; there was definite though not well-sustained ankle clonus. Also there was apparently a left extensor response, though this was difficult to be sure of; sometimes there was no movement of the toe in response to the stimulus. The left arm jerks were also exaggerated. Both abdominal reflexes were active at this stage.

A minute or so later there was slight return of power in the limbs, distal more than proximal, and more in arm than leg. At this point sensation was rapidly tested, when it was found there was no diminution of appreciation of pain on the left side, but considerable diminution of tactile acuity.

Topognosis also was tested, when there was seen to be definite preaxial localisation.

Patient then remarked that the numbness had spread to the inside of the left cheek, and under the tongue on the same side. The chattering of the jaw was less, but he said that he had a disagreeable feeling in his throat, as though he were "swallowing a sob."

His sense of position and of passive movement was then tested in the left hand, and it was found that when his third finger was moved he referred it to the first, his fourth to his third, his second to his second. The diminution in sensation continued.

When I was testing his sense of pain again, in the left hand, he said that a prick felt hot as well as sharp. His fingers still felt numb, but the numbness was passing from the rest of him. From this point his left grasp began rapidly to improve, but it was noticed that he could not sustain it owing to defective innervation of the synergic and defective inhibition of the antergic muscles.

A few minutes later the subjective sensations had almost disappeared, except in the fingers. The jerks remained very brisk. The power of the limbs was returning.

(3) *Six days later, a third fit was observed in part.* It had begun with a sensation "like a battery" starting in the left hand and running quickly up the left arm and down the left side into the leg and foot, as well as into the tongue and face (left), and this was succeeded by numbness in the same left-sided distribution. When I saw him, he said he had lost all power over the left limbs. I found that the left side of the face moved less well than the right, and that the tongue was protruded distinctly to the left. The left limbs were limp and useless; voluntary power was practically nil, though there were just the slightest movements possible of finger flexion.

While I was conducting this brief examination, the clinical condition changed before my eyes (as on previous occasions). The left limbs became rather rigid, the arm moved (involuntarily) into a position of slight flexion at elbow and adduction at shoulder while the fingers partially closed. At the same time the left leg flexed slightly at hip and knee, extended at ankle, and assumed a position of adduction.

Within a minute or two power commenced to return; movements of the left arm increased in strength and were associated with a sort of coarse tremor or oscillation of the left arm at the shoulder—an internal and external rotation of the upper arm in succession. Passive movement of the left leg evoked a kind of spasmodic alternation of extension and flexion at hip and knee through a very limited range.

The deep reflexes were exaggerated on the left side, but both plantars were in flexion, and both abdominals were present and active. Sustained ankle clonus was readily elicited on the left side.

Some ten minutes after the commencement of the attack the patient was rapidly recovering both movement and sensibility, and in a quarter of an hour altogether the above-described symptoms and signs had vanished.

The hyperæmic condition of the optic discs eventually changed into a slight papillœdema, and the question of operation was broached. The patient refused to consider this, and passed from my observation.

DISCUSSION.

The case illustrates excellently the occurrence of akinetic or inhibitory phenomena in succession to pronounced sensory discharge, and offers features of considerable importance in respect of cortical physiology.

In the first place, we note that the sensory aura follows strictly the march that is to be expected from cortical excitation, viz., from the fingers up the arm to the shoulder and neck, thence down the trunk to the leg and toes, and at the same time into the tongue and face. This aura by itself is sufficiently indicative of the cortical site of the physiological disturbance. Secondly, the outfall of motor function is found to vary according to the stage reached in the course of the seizure, the affected limbs being flaccid at the outset, and passing through a period of relative rigidity on the way to recovery. When the arm and leg were completely flaccid they were also powerless, and as tone returned so did a degree of volitional innervation, though the two processes did not advance entirely *pari passu*. It would seem that some degree of return of muscular tonus preceded the other. Of especial physiological interest is the fact, determined in each of the three personally observed fits, that during the process of recovery from the stage of absolute flaccid akinesia the limbs for a time exhibited the phenomenon of defective inhibition of the antagonists in a striking fashion. In other words, disarray of the normal interaction of synergists and antergists for the time being existed. This clinical manifestation has long been recognised as liable to occur in cases of hysteria with motor symptoms; physiologically speaking, hysterical disorders are initiated at cortical or transcortical levels. But defective antagonist inhibition is not confined to cases of hysteria. Considerable attention was given in my Croonian lectures to personal observations made in cases of chorea and of athetosis, which have proved that in these states Sherrington's law of reciprocal innervation is often in abeyance and that interruption of the orderly sequence of agonist and antagonist innervation is frequently found. In the same series of papers arguments were advanced in support of the view that such interference with reciprocal innervation is commonly of cortical site and is not observable at lower physiological levels, or only exceptionally so.

If this hypothesis is well-grounded then the appearance of the sign of defective antagonist inhibition in the course of akinetic Jacksonian epilepsy is particularly informative, for it throws light on cortical motor function from the clinical side, and incidentally substantiates the general contention. It cannot be seriously maintained that the motor symptoms of this case of mine have any other origin than the motor region of the cortex.

Actual loss, or diminution, of the deep reflexes was not observed at any point in the course of the attacks in this case. Possibly, had I tested them at the period of maximum flaccidity, it might have been otherwise, but as motor and sensory states were on each occasion first submitted to examination the

reflexes were reached only after the lapse of several minutes, and this may have been sufficient for a stage of diminution to have passed. However this may be, their exaggeration, the presence of ankle clonus and, on one occasion, of a probable extensor response, clearly point to infracortical release synchronous with cortical inhibition In this connexion the appearance of an extensor response during a phase of the ordinary major epileptic fit may be recalled. I have not myself been fortunate enough to observe definite loss of the knee-jerk in the course of general fits, but Gowers states it is sometimes abolished immediately at the close of the stage of convulsions.

The development of temporary motor paralysis, without spasmodic discharge, in the course of attacks of the above description, must be assigned to lowered activity of the motor centres involved, that is, to their inhibition, and it is legitimate to assume that the discharge in the corresponding sensory centres has somehow been the cause of this. The likelihood of this being the correct explanation is by no means remote. Under a variety of circumstances powerful stimulation or excitation leads to arrest of function. When a person is " thunderstruck," is " struck all of a heap," in consequence of some violent sensory stimulus, he may be for the moment incapable of movement—he is " paralysed." Reflex action can be interrupted or inhibited by strong painful cutaneous impressions. The only satisfactory way of explaining the cessation of the motor phenomena of a Jacksonian fit by ligature of the limb concerned—a procedure known to the ancients—is by assuming an action on sensory centres and inhibition of corresponding motor centres. In a recent paper on epileptic variants (see this JOURNAL, vol. viii, p. 223) I have cited a case in which a strong sensory discharge (olfactory) was succeeded by a trance-like state of motionlessness. It is known that a negative phase or refractory state of a given physiological mechanism or reflex arc follows excitation. Too loud a sound deafens ; too bright a light blinds. We may readily conceive of a refractory or inhibitory phase developing in a motor mechanism if it is suddenly or rapidly assailed with violence by afferent stimulations of an exaggerated character. However antagonistic and mutually exclusive processes of excitation and of inhibition respectively appear to be, they must without doubt be held to be at the same time closely interrelated. The most recent evidence pointing in this direction is furnished by the elaborate investigations of Pawlow, who has conclusively proved how readily inhibition follows stimulation, and how, in respect of cortical function, the two processes are constantly and continuously interacting and superimposing, one on the other.

This being so, it would be curious indeed were phenomena of inhibition not to characterise one or other of the phases, one or other of the manifestations, of what for want of a better term we call epilepsy. Epilepsy cannot call into being physiological processes that do not exist ; at the most it can only distort or caricature the processes of the individual concerned. As Hughlings

Jackson said, "the convulsion is a brutal development of the man's own movements." Analogously, an epileptic development may be a sudden inhibition of the man's own movements.

My chief purpose in discussing the inhibitory side of epileptic semiology has been to bring the latter into line with other recognised clinical states of which akinesia is a, perhaps the, prominent feature. Elsewhere I have recently examined at length the clinical symptoms and pathogenesis of so-called narcolepsy and cataplexy, and have stressed the fact that "sleep" is somewhat of a misnomer in view of the observed nature of the clinical syndromes. These are constituted, rather, in numerous instances, by a state of immobility without loss of consciousness, and in my opinion are often allied to catalepsy on the one hand, and to epilepsy on the other. Instead of rigidly enforcing such criteria as may appear to differentiate all of these, it has been my aim to emphasise their parallels and resemblances.