prevent subinvolution and the development of displacements, which not only impair the pelvic musculature, but lead to defective uterine action in subsequent labours.

Under such a regime, the pathological phenomena of old age will become rare, but the old multipara will continue to bear children and to require the special attention of the midwife. Much can be done by simple conservative methods to mitigate the evil effects which have already been described; the exhibition of iron and calcium combined with a suitable aperient has a most favourable effect on the muscle tone, and it may usefully be combined with 0.5 c.cm. of pituitrin twice a day during the last week or so of pregnancy; this not only improves the tone of the uterus, but in many cases brings on labour, to the benefit of the patient. Uterine distortion and dislocation must be controlled, and the resulting malpresentations and malpositions corrected by manipulation or the application of padded binders. The induction of premature labour before or at term is a useful prophylactic measure for women who habitually produce big children; in this connexion it is well to remember that, from the functional point of view, the pelvis gets smaller as the patient grows older.

During labour the application of forceps or version in certain cases will relieve the efforts of the uterus and reduce the work of the myocardium, and the third stage must always be conducted with the greatest possible care. A good deal can be done by these conservative measures to reduce the maternal risk, but the obstetrician should not hesitate to employ more heroic treatment if he believes surgical intervention to be necessary. It may require a certain amount of moral courage to suggest operative delivery to a woman who has already been delivered many times *per vias naturales*, but a careful review of the situation will show the wisdom of such advice for carefully chosen cases; indeed, it is safe to predict that in a more advanced epoch the majority of 8-paras and all women of 40 will be delivered by Caesarean section.

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The Morison Lectures

ON

NERVOUS SEMEIOLOGY, WITH SPECIAL REFERENCE TO EPILEPSY.

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BY

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LECTURE II.—SYMPTOMS INDICATING INCREASE OF NEURAL FUNCTION.

PASSING attention has already been directed to the general division of nervous symptoms, whatever their order, into those expressive of increase and decrease, respectively, of nervous function. The classification is applicable as well to those of the neuro-sympathetic system as of the psychical series. Possibly the term "increase" is not the most satisfactory that might be selected; it is taken to include symptoms and syndromes indicative of escape or release of function, in addition to such as presumably come into existence through stimulation or excitation.

Clinical experience affirms that an extremely large part of neural semeiology is included in this first main division, but the two sets are not mutually exclusive by any means; convulsions and paralysis can occur in the same subject, not, of course, simultaneously in the same part, yet in immediate sequence, while a limb already paralysed may become convulsed; the phenomenon of anaesthesia dolorosa is constituted by the synchronous combination of subjective pains or neuralgia referred to a region which is objectively anaesthetic; involuntary flexor spasms can take place in paraplegic extremities that the patient cannot voluntarily innervate. Further, not a few of the semeiological concomitants of epileptic fits are substantially indicative of functional inhibition; and some varieties of seizure are considerably more inhibitory or akinetic than kinetic or excitatory. Thus the question may arise whether in point of fact any symptom group is not accompanied by evidence of both one and the other physiological actions; but this is probably true only of the higher levels of the nervous system, and in the special sense that if an excitatory process is to exteriorize itself it must succeed in overcoming inhibition. No escape-phenomena can accompany paralysis at the level of the lowest motor neurones, but the same affection of the highest almost inevitably entails the likelihood of release below. Whether inhibition is constantly present is doubtless matter for discussion; if I become conscious of the aching of a carious tooth it is always allowable to assert that inhibitory control must be in abeyance ere impulses from dental end-organs reach the cortex to give rise to (I do not say "to be changed into") sensations; yet of the actual existence of such a mechanism, still more of its intimate nature, we are likely to remain in considerable ignorance. As regards the general sensory system data suggesting the possibility of sensory inhibition have been marshalled recently by Foerster (loc. cit.), while in respect of the visual system some experimental work by Brouwer[18] has seemed to prove the existence of an *efferent* system accompanying the optic tract from external geniculate to occipital cortex, conceivably inhibitory in function. Thus some basis is provided for the hypothesis; at any rate, we know that different functional levels of the nervous system enjoy comparatively independent life, and that those of higher are, as it were, protected against those of lower grade. When we speak of centres "representing" lower units in more complex ways and of their constituting also reservoirs of energy, we should not overlook the possibility of their having inhibitory functions too. Hughlings Jackson has somewhere spoken of these upper centres as forming "resisting positions"; owing thereto, activities of the highest levels can proceed without interference from the environment (the life of thought would be intolerable otherwise) on the one hand, and without effecting reactions on it on the other. It is matter of common knowledge that impulses from the periphery must in numerous instances never penetrate to the cortical level, being damped down or turned off and shunted to the efferent side through infracortical reflex arcs. Muscular tone maintains the subject's erect position by impulses derived from the sensory periphery and passing over a mesencephalo-spinal circuit of which he is completely unaware. Similar comment applies to the abundant activities of the neuro-sympathetic system; on rare occasions only are we consciously aware of them. Protection may be justifiably assumed in respect of cortical action; and in consequence thereof it is permissible to argue that symptoms whose character partakes of increase or release of neural function must break down resistance and neutralize inhibition ere they come into being. But with this proviso much in neurological semeiology consists essentially of "positive" symptoms, signifying enhanced or exalted activity on the part of the mechanisms involved.

We may now consider one or two specially selected aspects of the most obvious type of "positive" syndrome—namely, that described in semipopular phraseology as "attacks," "seizures," or "fits."

On various previous occasions I have dealt with one or other side of this subject, though not before in the fashion now to be adopted. Most neurologists are familiar with the Jacksonian conception of three physiological levels or grades in the central nervous system, spoken of by him as the highest, the middle, and the lowest respectively. Of these the first he took to have its anatomical substratum in the frontal lobes; the second or middle level corresponded to that part of the cortex where are situated the Rolandic centres; the lowest was ponto-bulbar in position. We need not follow Jacksonian conceptions slavishly in the present connexion, seeing that he himself was ever willing to discard unsubstantiated hypotheses; at the same time, and in spite of its schematic character, I shall for my present purpose utilize his theory in modified form; the highest

level I shall assume to correspond to the whole cortex outside and above anatomical projection-centres; the latter constitute the middle level; while the lowest is taken to include what may properly be regarded as the old nervous system—namely, basal ganglia, brain-stem and cerebellum, and spinal cord. With this tripartite division of the neuraxis we may examine the relation of fits, however diverse and heterogeneous, to increase or escape of function in respect of each of these regions in turn.

It is, of course, necessary at this juncture at once to underline the fact that radiation of release or excitatory phenomena characterizes numerous attacks or seizures, whose manifestations are not in their totality confined to one level only; hence differentiation to facilitate description can scarcely avoid becoming schematic also, though not in any sense arbitrary.

FITS AT DIFFERENT PHYSIOLOGICAL LEVELS.

In accordance with what has just been suggested a theoretical division into highest, middle, and lowest level fits respectively is conceivable; but can these be distinguished clinically, and, if so, is the fact of any clinical value? I am of the opinion that both questions may be answered affirmatively.

HIGHEST LEVEL FITS.

These must depend for their intrinsic characters on the functions of the highest level, naturally. Discarding all such question-begging terms as psycholepsy, psychical equivalents of epilepsy, and analogous expressions, we may assert that any kind of attack signalized by phenomena of a psychical type is *pro tanto* a highest level fit. From this standpoint every fit preceded by a definite aura must be placed in the class so far at least as that symptom is concerned, for the aura is nothing more or less than a sensation or hallucination, and therefore belongs intrinsically to the psychical series. It must be regarded as the earliest component of the fit, its prelude, but not (as was formerly supposed) its cause. By no means invariably present, it practically always reproduces itself in the same form for each of the attacks of the person concerned. The aura ranges over the whole field of sensation, inclusive of the special senses, and indeed of feeling, memory, and other psychical elements; it may be simple and crude, or complex and elaborate; or these two opposite features may occur in the same case with reference to different sensory modalities. Sometimes clear and impressive, at others it is elusive, vague, fleeting, and difficult both to recall and to express. The question arises whether distinctions can be drawn, for any given sensory system, between auras of lower and of higher grade; and whether those of the former category are initiated in projection- as opposed to association-centres—that is, start in regions belonging *ex hypothesi* to the second or middle group. For that matter, fits of lowest level type, if an aura is present at all, must somehow be able at the outset to influence the cortex—assuming that to be the seat of consciousness. Evidently the way must be open for impressions to ascend to the percipient region in the latter case; part at least of the fit, therefore, must be of *ascending* type (a significant consideration); and when the aura seemingly starts at the middle level there must be neuronic integrity again in an ascending (transcortical) direction for its underlying impulse to reach the perceiving cortex (whatever and wherever exactly that may be taken to be). For instance, I recall the case of a child whose visual aura consisted of what she described as "twinkles" in the right visual field—crude, flashing medleys more or less like coloured stars—and I contrast it with that of another patient who, as he sat in a summer-house in his garden, saw a white-robed figure pass over his garden wall on the left, with back towards him, and move slowly towards the french window of the room which opened on to the lawn; as the figure reached the window he lost his senses. Clinically, again, an auditory aura constituted by a loud bang seems far removed from that of the patient who declared, "I seem to hear everything that has ever been said to me in all my life," or a sudden stench in the nostrils from "a series of odours, as if I were passing a perfume-shop"—to quote the words of another. Are we to consider the simple crudities of the one class to represent excitation in projection-centres, the complexities of the other to stand for associational or highest level excitation? Possibly we should, but it is questionable whether such distinctions can be drawn with precision; nor from the viewpoint of cerebral physiology is it clear that projection- and association-centres, for a given sense modality, are separable with any exactitude, although for anatomical reasons it may seem justifiable to consider them apart. A coarse and powerful sensation of "pins and needles" in the fingers perhaps points to excitation of projection-centre or -system, and it appears to belong to an entirely different category from the aura of the "dreamy state"—that condition in which the patient, at the oncoming of his fit, experiences a series of panoramic pictures, or lives again some incident or incidents in his past life, or feels as though he were in some other place than the actual one, or is conscious of a *déjà vu* feeling, and so forth. These peculiarly interesting auras [1][9] have recently analysed and illustrated, and shall not here say more of them. Often enough the elaborateness of the experience will not permit of its being explained by reference to one sense avenue only; thus a patient's aura consisted in his taking the end position of a row of twelve men in military formation; a superior in rank stepped forward and boxed the ears of each in turn, knocking him down, while our man stood in fearfulness awaiting the inevitable termination; as his ears were boxed he lost his senses.

From the features of the aura it is proper for the clinician to endeavour to determine its approximate localization, since this may be of immediate practical value; at the same time we are not always in a position to state whether it arises in a lower centre, or in a higher centre representing the other. This applies in particular to auras presumably of a neuro-sympathetic kind; when, for instance, in what for other reasons we take to be a lowest level fit the initial symptom is "pain round the heart, like knives" (as in a recently observed case), does the excitation arise in the ganglionic collections of the organ itself, or in viscero-sensory centres of paravertebral chain or spinal cord, or in the dorsal nucleus of the vagus, or at some conjectural higher spot still, re-representing the heart? The "dreamy state," whether it occurs in connexion with olfactory and gustatory hallucinations or not, suggests a disorder of function whose starting point is the temporal cortex of one or other side, spreading probably by definite transcortical paths to the occipital lobe or occipito-parietal cortex, for the visual component. We can conceive of the aura as ranging from excitation of a small portion of a sensory centre to implication of wide areas of the associational cortex; the psychical stuff it is made of is one and the same, whether we are dealing with tingling in the great toe or a feeling of being back in a former existence.

Thus regarded, I seek no differentiation between the auras of hysterical fits and of organic epileptic seizures. I cannot detect any physiological difference between one and the other. At the sound of Zeppelin bomb-dropping in London during the war a soldier patient hopped out of bed and pushed his pillow before him as he crawled on his stomach along the polished floor of the ward, re-living graphically his experience of the trenches when seeking cover; at the end of the room the hysterical fit came. Elaborate the aura was, but not different in kind from that of some temporal fits. However unfamiliar the conception may perhaps be to some, it is surely proper to consider hysterical seizures as highest level fits *par excellence*—that is, fits of which the phenomena are largely confined to that level. To my mind such divergencies as exist between hysterical and epileptic seizures are explicable with facility in terms of the functions of the physiological level at which the phenomena are occurring. This is true, in particular, of the motor display. Every clinician who has witnessed hysterical attacks is impressed by the quasi-purposive character of the movements; they have a meaning, they appear to be devoted towards an end; the cruciform posture, *arc de cercle*, *attitudes passionnelles*, and so forth, of the hysteric reveal what is in the subconscious mind. But are movements of analogous

nature foreign to the ordinary major epileptic fit? By no means. It is most important and significant to note that normal, co-ordinated movements frequently occur apart from those that are termed "convulsive"; during one or other part of the fit the epileptic patient may make champing movements of his jaws, smack his lips, spit, or clutch at his throat; after the convulsive period, and while still unconscious, he may exhibit other motor phenomena such as plucking at his clothes, undressing, running, and what not. A young man once under my care, suffering from a left frontal abscess, invariably waved his right arm round and round in circles ere overwhelmed by unconsciousness. The movement was co-ordinated, and as such clearly referable to the highest motor level—that is, to a cortical region distinct from the Rolandic. It is possible that the head and eyes deviation which is a common first sign of the generalized epileptic fit is caused by implication of the corresponding centre which the experimentalist has shown is situated in the frontal lobe, separate anatomically from middle level centres in the Rolandic cortex.

Moreover, the whole motor content of some attacks of the epileptic class is not convulsive at all, but strictly co-ordinated. A patient of mine suffers from fits in which he turns very pale and loses touch with his surroundings, but never falls; he raises his closed fist and shakes it, blinks with his eyes, then looks sideways as though to see round a corner. The fit is over in about a minute. His maternal uncle suffered from major epilepsy, also a cousin. The motor phenomena of petit mal are often co-ordinated rather than convulsive, as any close observer can convince himself. In this connexion it is of interest to recall Jackson's contention that the anatomical substratum of petit mal attacks is situated in the frontal lobes.

We see, then, that among the symptoms referable to excitation or transcortical release on the highest physiological level are (1) an aura in consciousness, and (2) types of movement co-ordinated and quasi-purposive, occurring either in association with convulsive movements or by themselves. To these I now add (3) post-convulsion movements and actions of the same high level class. Known for years under the term "post-epileptic automatism," the manifestations of this phase clearly belong to the group of escape-phenomena; purposive in appearance, none the less in the strict sense they are completely involuntary, and, being such, the patient is not responsible for them in a medical or forensic sense. Their duration ranges from a disorder of a few minutes to prolonged fugues, indistinguishable from those of hysteria. Recently a patient, living at Catford in South-East London, went for an afternoon stroll to a neighbouring pond; he was next seen taking a motor bus to Croydon; from there apparently he walked right across London to Paddington station, took a ticket to Penzance in Cornwall, and "came to himself" about 4 o'clock in the morning in a Cornish express far in the West. His mind was a complete blank after he had entered the motor bus. A sufferer from inveterate petit mal, there can be no reasonable doubt that his fugue succeeded a brief attack unrecognized by any passer-by. The medico-legal import of such states is at once apparent, for the subject is to all outward appearance normal. During the war an epileptic patient boarded the London train at Brighton; it stopped on the Grosvenor bridge over the Thames, just outside Victoria station; here he opened the carriage door and walked over the metals, where he was challenged by a sentry guarding the bridge. Unable to furnish a clear account of himself, he was arrested and given six months' imprisonment. Only on appeal was the evidence of those who knew his case accepted—namely, that he must have had a fit in the train and been in a state of post-epileptic automatism when he came in contact with war regulations. An even more impressive case, because of its criminal aspect, was that of a man in early middle life who sustained a severe accident in the frontal region and thereafter on at least three occasions developed fugues of greater or less duration (in one of which he came from Wales to London consciously unaware of the fact) preceded by "attacks" in which he turned pale, his eyes became fixed and staring, and for the time he was oblivious of his surroundings and did not hear or could not answer questions addressed to him. In one of these turns he, unfortunately for himself and for the child, threw, or let drop, his infant girl, 6 months old, out of a bedroom window on to a stone-flagged yard, where it fractured its skull and was killed. After his arrest on the capital charge, I gave evidence on his behalf to the effect that he had been in a state of fugue and was irresponsible for his actions, since unconscious of them, medically speaking; and I had the satisfaction of contributing to the favourable finish of the case.

Conditions of this kind seem far removed from the crudities of an ordinary generalized epileptic seizure, but I urge the irrationality of delimiting epilepsy by its middle level semeiology and insist that for clinical and scientific purposes both ante- and post-epileptic phenomena form an integral part of the whole. These, as must clearly emerge from any adequate consideration of the matter, include symptoms on the highest physiological level of activity, frequently if not invariably. In themselves they present close resemblances to symptoms which we call "hysterical," and if this view commends itself then it seems otiose to try and formulate distinctions, for they can scarcely amount to differences. The name we give to a symptom or syndrome is of purely secondary significance; its characters and pathogenesis must guide us in coming to any conclusions as to its nature.

Finally, in this connexion, I should like to direct attention to an aspect of epileptic consciousness which provides food for thought. Now and then one comes across a case where the subject declares that his attacks have been actually pleasant, or at least have been associated with, or left in his mind the impression of exhibiting, some element of a distinctly pleasurable kind. An intelligent lady of 32 suffers from attacks which belong to the uncinate variety of so-called idiopathic epilepsy, and are characterized by a "dreamy state" aura with a strong feeling of familiarity or *déjà vu*; in her own words, "I feel as if my mind and my body are coming apart"; or again, "I can see myself walking in the garden as if my mind were looking at myself from afar." This peculiar mental state is accompanied by a definite feeling of pleasure, so much so that at first the patient used to welcome the sensation as a "dream of delight"; the precise pleasurable element, however, she has never been able to specify. At a later stage the fits became few and far between, and confined largely to the briefest of *déjà vu* auras without any pleasurable component; for nine years now she has had none at all. A more recent case is that of a young woman who has been epileptic for some ten years; up to the age of 18 the attacks seem to have been little more than a kind of prolonged petit mal, and were accompanied by a highly pleasurable feeling; "I felt that I had been away somewhere in a pleasant dream, which I was enjoying to the full." Looking back on it, she was convinced it had been delightful while it lasted, and that this was due not so much to a physical as to a mental state. Later, the fits became more severe and typically those of grand mal, and since their development (with severe post-epileptic headache and myalgia) the pleasurable element has vanished and its place been taken by a vague and ill-defined dread or apprehensiveness, experienced now as a prodrome to the attack. I may also cite the words of another patient, whose case is given in some detail elsewhere[19]: "I used to feel I had had a previous existence, and that in these sensations I stepped out of myself into the other existence. I felt as if my brain were leaving my body for the moment. I used rather to welcome it, as a not unpleasant feeling."

The explanation of the rare appearance of this pleasurable emotion in connexion with epileptic auras and states is far from easy. The common epigastric aura not infrequently brings in its train a feeling or emotion of fear or terror, as of something impending, something dreadful about to happen, although seldom does it embody any specific ideational element with which the fear is linked. From over 3,000 cases of epilepsy some 92 were collected by Gowers[20] into a group distinguished by "psychical auras" (since all auras are psychical the term is imprecise), and

of these about one-third were characterized by a feeling of alarm or fright; but no allusion is made to the opposite variant. Perhaps it is not a coincidence that in each of the three personal cases just quoted the pleasurable emotion occurred in connexion with "dreamy states," though I do not know how to account for it except on the speculation that by some temporo-thalamic path a thalamic element entered into the composition of the whole. Nor do I understand why, if such be the case, the condition should not occur with greater frequency.

Middle Level Fits.

In accordance with long-established usage the term "epileptiform" convulsions should be confined rigorously to cases of Jacksonian epilepsy—that is, fits of the middle physiological level. I shall limit attention at this time to one or two problems presented by what may be termed "reflex Jacksonian epilepsy."

Reflex epilepsy is an expression which has never been defined with any precision. It has been employed in a general way to connote the development of fits as a sequel to peripheral irritation of one or other kind, and older treatises enumerate as examples those which have been thought consecutive to adenoids, errors of refraction, carious teeth, dyspepsia, helminthiasis, tight prepuce, and what not. The phrase "secondary epilepsy" was proposed long ago by Nothnagel for rare cases in which actual injuries of peripheral nerves were followed after an interval by epileptic seizures, with or without an aura referable to the part affected. It seems curious that with the wealth of peripheral nerve lesions produced by the war no such cases appear to have been recorded, so far as I am aware. We may recall also in this connexion the experiments of Brown-Séquard,[21] who after various operative procedures (hemisection of cord, section of sciatic) on animals (especially guinea-pigs) found it possible to develop in them convulsive attacks by excitation of an "epileptogenous zone" over face and neck (corresponding in distribution to part of the fifth and to upper cervical nerves). His results were confirmed by some observers and criticized by others; in 10 of 41 guinea-pigs with cut sciatic Bramwell and Graham Brown[22] produced "fully developed" fits by stimulating the zone after some seven weeks had elapsed. We may note, however, that from descriptions recorded generalized and not unilateral convulsive movements were commonly obtained. It is a precarious proceeding to apply to the case of man conclusions derived from experiments on much lower animals, but from time to time instances have been published of reflex epilepsy of this kind. As long ago as 1886 Hughlings Jackson[23] showed at the Medical Society of London a boy of 7 who exhibited epileptic fits whenever his head or face was touched; express mention is made of the fact that when he was aware he was going to be touched none occurred. In the discussion following this paper somewhat analogous cases were alluded to by various physicians, of which one is of considerable interest, that of an epileptic with disease of the femur. "When dressings were applied, or when the sinus was probed, the man had severe epileptiform seizures. Amputation was performed, and the man had only one epileptic attack afterwards, and that was at the first dressing, when the 'epileptogenous' area was touched." More than ten years previously, in 1874 to be exact, Dr. James Dunsmure,[24] physician to the Royal Hospital for Sick Children, read before the Medico-Chirurgical Society of Edinburgh a paper on the case of a boy of 5, who suffered from attacks of what was described as "temporary loss of voluntary power" whenever anything touched his head without his foreknowledge. The details of the fits suggest that they were mainly akinetic or inhibitory, although on one occasion the muscles of the right arm and leg were "slightly convulsed." Later information about the case was sent to Jackson by Dunsmure, and will be found in one of the former's Neurological Fragments[25] (No. 13, 1895). From it we gather that the youth eventually died of peritonitis, and that his brain was examined by the late Dr. Alexander Bruce, who in 1892 was pathologist to the Royal Infirmary. No changes were found in pons or medulla (Jackson had believed the fits to be of lowest level type), but many of the cells of the grey matter of the cortex were vacuolated.

In a paper on "Epileptic variants"[26] I have mentioned the case of a little girl whose first fit occurred when the hat-elastic under her chin slipped up and hit her nose, and allude also to other kinds of reflex attack which have come under notice, inclusive of so-called acoustico-motor epilepsy.

Recent practice has brought under my care two cases of what seems to be genuine *Jacksonian epilepsy of reflex origin*. The first is that of a woman of 51, whose attacks began eight months before her admission to hospital, towards the close of 1929. I shall quote from the records of the case made by my house-physician, Dr. Denny Brown.

She began to have attacks of unconsciousness whenever anything touched her right ear, or her head just behind the ear (for instance, putting her spectacles on). These began with a twitching and tingling pain in the right side of the face, and a clicking noise in the head. She remained conscious, but could not say what she wanted to. Weakness and numbness of the right side of the body lasted from one to twelve hours after each attack. Six weeks ago she began to have similar attacks brought on by some stimulus to the right foot or the right leg below the knee (for example, pulling a stocking over the foot). These also began with clicking in the head and a tingling pain and twitching, starting in the right foot and passing up the right leg and down the right arm, while also involving the face on that side. They were followed by unconsciousness for a few minutes, and by residual aphasia, numbness, and increased weakness of the right limbs for an hour or two.

Two attacks have been observed, the first produced when eliciting the knee-jerk on the right, the second by pulling the bedclothes away from the right leg. Both began by tonic extension, followed by twitching, of the right ankle, knee, and hip, and then of the right arm and face; immediately after this the whole left side at once became involved. The duration of unconsciousness was about four minutes, followed by aphasia and then marked perseveration, with weakness of the right arm and leg and extensor plantar response on the side, flexor on the left. Complete recovery ensued three hours after the onset, with return of all signs to those found on admission.

An exploratory operation over the left Rolandic cortex showed nothing else than a milky appearance of the arachnoid in the vicinity of the superior cerebral veins, with no evidence of neoplasm. There seems little doubt that the pathological basis of the condition is one of cerebral arterio-sclerosis, a very common antecedent to Jacksonian epilepsy, but the peculiar features of the case may best be examined after a note on the second.

It is that of a young man who, at the age of 30, some seven years ago, began to suffer from Jacksonian attacks, beginning in the right thumb, or in the fingers of the right hand. These were usually brought on by a "start," on occasion spread typically over the right side, and ended in unconsciousness and generalization, with tongue-biting. Examination yielded a negative Wassermann reaction in the blood; and since nystagmus, moderate in degree, was occasionally present on lateral deviation in either direction, while the optic discs were distinctly hyperaemic, with slight blurring along the inner edges, a tentative diagnosis of intracranial tumour was made, or, alternatively, of disseminated sclerosis.

The case has been followed with the greatest closeness since 1923; during that time no material change has occurred; very occasionally a right extensor plantar response has been obtained; the tendon reflexes on that side are brisker than the left, and the discs remain as before, without atrophy.

To illustrate the phenomena in this unusual case a series of incidents which have started the fits may now be described, as narrated by the patient himself.

1. When he was walking along in rain with his umbrella up his right hand, which was some little way up the shaft, was struck by the catch slipping and the frame coming down on it.

2. While he was carrying a rather heavy parcel in the same hand the string broke and gave the limb a slight "shock," aggravated by his making a jerky attempt to catch the parcel with that hand as it was falling.

3. As he was pressing rather heavily with a file in his right hand on an object he was working at the file suddenly broke.

4. When he was holding two small articles in his hands and endeavouring to screw them together one slipped out of his grasp with some little force.

5. On two occasions, lying in bed half asleep, with his right arm underneath him, "pins and needles," presumably from pressure, have commenced in the periphery of the limb.

In all of these (and other) instances a direct "start" or "jolt" of the right upper limb, distally or throughout, has been the immediate precursor of the Jacksonian attack.

6. He once tripped his right foot over an accumulator which he did not notice, and fell.

7. On a station platform, stepping forward to take a train, his right foot caught in an unevenness of the ground and he stumbled.

In these instances it was the right leg or foot which received the shock.

8. On several occasions someone has brushed past him or knocked into him unexpectedly, although not specially on the right side.

9. A sudden and loud noise will initiate the sensation in the right hand; it has also developed when a door was unexpectedly opened in his face, as he approached it, without touching him. Walking with a friend, the latter stumbled over a rut in the road and gave the patient a "fright." Again, in similar circumstances the friend scraped a piece of paper along the ground noisily with his stick, and this proved an excitant. Sitting one day on a seat in his garden he was gazing at his cat, which suddenly jumped on to a high seat opposite, missed its footing, and fell, giving him a "start."

From these latter illustrations we observe that stimuli reaching the sensorium by the avenue of sight or hearing must be added to the possible excitants of the Jacksonian attacks, and that these are not restricted to direct stimulation of either the limb or the side by which the fits invariably commence.

As already remarked, some only of the seizures go so far as to become generalized and cause unconsciousness; others are of an intermediate class. One which was observed was of the following nature.

Whenever the aura spread up the right arm from the ball of the thumb (on this occasion) the right arm exhibited characteristic clonic twitching, breathing became laboured, and the face congested almost to cyanosis, while the legs gave way and the patient sank to the ground. Twitching ceased at the shoulder, though facial grimacing on the right side was in evidence, and thereafter the right arm, and indeed the musculature generally, was completely relaxed. He began to talk "nonsense," while his eyes wandered irregularly from side to side. A quarter of an hour later recovery ensued. At no point in this attack was loss of consciousness absolute.

The exact mechanism by which reflex Jacksonian epilepsy comes into being is by no means clear. That in these two instances we are dealing with hyperexcitability of cortical reflex arcs seems obvious, and the presumption is that sensorimotor centres in the Rolandic cortex are in a state of irritability, with the corollary that transcortical inhibition is at the same time defective. To my way of thinking the existence of cortical reflexes is of much significance for the understanding of many of the symptoms of neurology—reflexes on a high physiological level over which, in abnormal circumstances (such as the presence of actual local cortical lesions), what we call in our ignorance "volition" can exercise no inhibitory effect. I am tempted in this connexion to quote the words of a famous teacher of the old Edinburgh school, of whom I have heard Sir Byrom Bramwell, as well as the late Sir David Ferrier, speak with deep appreciation; I refer to Professor Laycock,[27] and cite from an article of his dated eighty-five years ago.

"Four years have elapsed since I published my opinion, supported by such facts as I could then state, that the brain, although the organ of consciousness, is subject to the laws of reflex action; and that in this respect it does not differ from the other ganglia of the nervous system. I was led to this conclusion by the general principle that the ganglia within the cranium, being a continuation of the spinal cord, must necessarily be regulated as to their reaction on external agencies by laws identical with those governing the spinal ganglia and their analogues in the lower animals. . . . Observations and arguments like those satisfactorily adduced in proof of the existence of the reflex function of the spinal ganglia may be brought forward in proof that the cerebral ganglia have similar endowments."

The motor area of the neuro-physiologist is nothing more or less than a motor or sensorimotor ganglion pushed up into the neopallium, and it would of a truth be disconcerting to our ideas of neural continuity were that ganglion to prove of a completely different physiological order from those situated at a lower level. Nature cannot surely make any abrupt "jump" from one class to another; for example, it would be a curious thing if what we call "mental states" were *suddenly* to make their appearance, and only at a final stage. Lloyd Morgan, Parsons, McDougall, and others take the view (adumbrated long before by G. H. Lewes) that consciousness exists on different physiological planes; in its simplest form, according to Parsons,[28] it is a mere sentiency—the emergence of an "awareness of a change in the environment." This awareness is consciousness on the reflex plane; it is tinged with affective tone and with a minimum of cognition. McDougall[29] says: "It seems probable that the actions of even the lowest animals imply a vague awareness of something, together with some vague forward reference, some vague anticipation of a change in this something." Carrying the idea further, and to its logical conclusion, we may affirm that at the cortical level in man reflex action may be, must be, accompanied by full awareness, by consciousness on the plane either of instinct or of intelligence, yet in itself that reflex activity may none the less exhibit all the qualities of the same when expressed through lower mechanisms.

The clinical phenomena of the two cases which we have been considering demonstrate the occurrence of pure reflex response to extrinsic stimuli through arcs whose nodal point is certainly situated in the cortical grey matter; in the second example the readiness of these cells to discharge is proved by their doing so as a sequel to afferent impulses reaching them not merely along the paths belonging to the arc in a topographical sense, but also by routes from sense-organs of another series. What seems to be an essential in the case is that the arc should be seized unawares; the patient remarked, apropos the incident of the paper scraped along the ground, "Had I seen it happen the fit would never have occurred." The comment suggests his capacity for inhibiting the reflex is still in being provided he is warned and therefore prepared; but in the case of the other patient the element of surprise does not appear to be a necessary factor, for she informed me when I first saw her that fits had taken place though she knew that her leg or foot was about to be handled. Absence of awareness, however, also was essential in the case of the youthful patients mentioned above who suffered from attacks whenever the head was touched. Be this as it may, the intimate mechanism in respect of facilitation of reflex activity owing to the immediate presence of a pathological condition (tumour, arteriopathic change, an area of sclerosis) remains obscure; and this is particularly so where no organic change is in evidence. Impairment of transcortical inhibition, which must also in my opinion be postulated, can be nothing else physiologically than failure of one cortical element to inhibit another—a state of affairs the "extreme likelihood" of the occurrence of which is assumed by Sherrington for various fields of cortical action. When we speak of the "involuntary" movements of Jacksonian epilepsy we can mean little else than that they cannot be transcortically prevented. In accordance with principles suggested for consideration in my previous lecture, it is not necessary to seek an objective lesion actually in or at the "centre" for the reflex arcs concerned; the defective inhibition may conceivably be represented by change at some wide region or in some cellular layer remote enough from the spot where they are in action.

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