The Morison Lectures ON NERVOUS SEMEIOLOGY, WITH SPECIAL REFERENCE TO EPILEPSY.

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BY

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LECTURE III.—SYMPTOMS INDICATING INCREASE OF NEURAL FUNCTION (*continued*).

LOWEST LEVEL FITS.

As already explained, this term is taken in a wider sense than that given it by Hughlings Jackson, and made synonymous with "infracortical fits."

The question whether the neural site of general epileptic seizures is of necessity always the same has received some attention, and been examined by myself elsewhere (ref. 19, Lecture II). Briefly, it amounts to this: can their motor phenomena be produced at other (lower) levels than the cortical? The experimentalist has shown that in the decorticate or decerebrate animal fits of a general kind can in point of fact be made to develop by resort to convulsants such as absinthe or thujone; and the distinction sometimes still advanced by the clinico-anatomist, that the clonic element in fits is of cortical origin, the tonic of infracortical, cannot be upheld in its entirety. Both tonic and clonic convulsions have been produced by Pike and Elsberg³⁰ in animals deprived bilaterally of the cortical motor areas, an interval of time sufficient for the return of "good locomotor reactions" being allowed to elapse after operative intervention; Dandy,³¹ it is true, claims to have proved that in the dog the cortex is required for the clonic component. In full decerebration, with only pons, medulla, and cord left, convulsions of a more or less general kind may develop during the respiratory sinking that presages the death of the preparation. But whether these are identical in all respects with those occurring experimentally in the intact animal can be settled only by precise observation, and so far as I am aware this has not yet been forthcoming. In regard to man the evidence undoubtedly points to important differences between fits of higher and of lower origin, as we shall see.

Taking our stand on the position that fits of any kind are fundamentally expressive of the functions of the part or parts affected, we must at once allow for the greatest diversity and heterogeneity. A major attack ranges from a disturbance on the highest plane (the aura) to phenomena of a visceromotor kind, through the whole scale of semeiology; and fits ostensibly caused by disorder in infracortical mechanisms cannot do otherwise than reveal the functions which these possess. The clinician tries to deduce the localization of a given attack by scrutiny of its functional features, correlating what he observes with knowledge otherwise derived. Facts compel us to admit that the property of uncontrolled discharge is common to all nerve-cell groups on occasion, even though we draw a distinction between permanent instability in a diseased tissue and occasional instability in a healthy tissue; and it stands to reason that if this generalization is accepted we may expect such syndromes as are specific for mechanisms of a given level. But one of the difficulties immediately encountered is to decide whether the observed phenomena partake of the general characters of a "fit." I confess this interpretative question becomes almost impossible to answer in any completely satisfactory fashion. The expression "genuine" epilepsy is little short of meaningless; such epilepsies as grand and petit mal are usually recognizable—the Jacksonian variety too—but clinical manifestations of analogous release or excitation in other divisions of the nervous system do not always convince the observer of their right of admission to the general category. Sometimes I envy the man in the street, who can talk of his symptom as a "turn," a "go," a "do," non-committally yet graphically, whereas science

compels the neurologist at least to try to give it a local habitation and a name in conformity with its physiology and nosology. As neural semeiology consists simply of hyper- and hypo-activity it is open to the clinician to claim any temporary, disorderly, paroxysmal, recurrent, uncontrolled manifestation of either motor or sensory processes as in essence a "fit," and this on any physiological plane. Does not common parlance permit the phrases "a fit of the blues," "a fit of temper," "a fit of laughter," and so forth? Then why not a fit of neuralgia, of tachycardia, of polyuria? Unfamiliar and possibly bizarre though such conceptions may seem, they do not differ integrally from "a fit of asthma" or "a fit of sneezing," which some will concede are allowable. Provided the phenomena partake of the general nature of a "fit" as just mentioned, with a substratum of physiological escape, it will be difficult to deny their intrinsic likeness to each other however protean their external aspects.

Such general comments are apposite to the question now before us, which can be approached by discussion of various infracortical groups.

"Striatal Epilepsy."

Within recent years the term "striatal epilepsy" seems to be enjoying a certain vogue. In 1925 Professor August Wimmer³² of Copenhagen recorded the case of a patient whose symptoms commenced by attacks of unilateral tonic spasm, lasting a few seconds, unattended by loss of consciousness, and recurring many times a day. Later they became generalized, and an eventual diagnosis of epilepsy was made. At a subsequent period involuntary movements of an athetoid type made their appearance, and finally the clinical picture became one of typical torsion-spasm. Because of the possibility of the latter condition having a striatal basis, Wimmer designated the tonic attacks and (more or less tonic) generalized fits "striatal epilepsy," supporting his contention by reference to a case observed by Stertz,³³ in which generalized seizures preceded the development of torsion-spasm and the lesions discovered post mortem were confined to lenticular and caudate nuclei. He also found substantiation for his argument, as he maintained, in the occurrence of epileptic attacks along with post-encephalitic Parkinsonism. Preferring the expression "subcortical epilepsy," Spiller³⁴ published in 1927 two cases of attacks of brief tonic spasm—in one, affecting both hands and a lower limb; in the other, the arm and leg on the same side. These spasms (no clonic element was in evidence) were brought on by voluntary movement of the limbs concerned in both instances, and in one could also be elicited by passive manipulation; further, the muscular contractions were painful and accompanied by a thermal dysaesthesia of "heat" or "burning" in the lower limb unaffected by the spasm. Because of a concomitant cervical sympathetic palsy in the case that ended fatally (no necropsy being allowed) Spiller suggested a lesion in the regio subthalamica, possibly haemorrhagic (there had been evidence of meningeal bleeding in the case at an earlier stage). Still another term, "extrapyramidal epilepsy," has been proposed, by Sterling,³⁵ for cases of spastic contractions in the course of post-encephalitis or during a more acute period, distinguished by painful spasms of fingers or toes, limbs or segments of limbs, and inclusive of facial and torticollis movement; brief or prolonged, the muscular phenomena are essentially intermittent and often paroxysmal, and their frequently "tetanoid" or "tetaniform" character is stressed in the title of his paper.

This plausible association of tonic spasms or painful spasmodic contractions with destructive lesions of the corpus striatum must, however, be examined under very considerable reserve. Naturally I accept any view ascribing tonic spasm to the group of the epilepsies in a general way, provided it occurs in "bouts" or "fits," so to say; but when the element of pain enters prominently into the motor contractions the case is somewhat altered. Clinically speaking, not a few of the examples cited, more especially by Sterling, can be fairly assigned to that class of painful cramps, and it seems inadvisable to widen the horizon of the epilepsies, almost limitless though that may appear to

be, by inclusion of a clinical category of such distinctiveness; equally well might we place the phenomena of intermittent claudication in the same collection. By common consent tetanus and tetany remain outside the epileptic rails, yet one of Sterling's examples of "extrapyramidal epilepsy" presented contractions of the hands and fingers, occurring daily for nine days and lasting two to three hours, exactly like those of tetany. Similarly, in the peculiar cases we owe to Spiller the facts that the muscular contractions themselves were dolorous and (in particular) that they arose in consequence of active, voluntary movement, in my opinion render their ascription to any of the epilepsies, as ordinarily understood, more than a little dubious. Painful reflex spasmodic phenomena occur under a variety of different circumstances; the cramps of cholera are etiologically distinct from those of ballet dancers or swimmers, which ensue on voluntary movement, and each from those of encephalitis; but they are clinically alike in the elements of paroxysmal painful tetanic muscular contraction; and we should proceed warily in the matter of regional allocation. I share Spiller's own clearly expressed hesitation as to the precise nature of the symptom-complex in his cases and his unwillingness to accept a striatal origin for them.

Further, on previous occasions I have animadverted on the irrational attribution of motor phenomena to affection of the corpus striatum merely because of their occurrence in the course of epidemic encephalitis, and I take the opportunity of underlining again this *non sequitur*. Implication of the pyramidal system for spasmodic syndromes is not excluded because Parkinsonism and its rigidity may be indirectly due to striatal lesions. So far as I have noticed, the obvious comment has not been given any consideration, that such tonic spasms, being release or excitatory symptoms, cannot result from outfall of striatal cells. Neither the generalized fits nor the torsion-spasm of Stertz's case can have been caused by the destructive lesions of the caudate and lenticular nuclei found on microscopical examination by Spielmeyer. It is immediately plain that the actual mechanisms involved in the production of these tonic movements must have occupied an extrastriatal position.

There is no novelty whatever except its proposed name in this conception of "striatal epilepsy." Reference to the old descriptions of Ormerod and Gowers in respect of their cases of lenticular degeneration (quoted in my monograph²⁶ on the subject) shows that "tonic spasms" were even then recognized as forming a part of the clinical picture. Gowers employed the epithet "tetanoid" for the contractions in his cases, and said of the first that "the muscles of the neck, trunk, and limbs were all involved, in varying degree, in the generalized tonic spasm"; and, again, "the spasmodic contractions of the musculature became intensified," etc. The value of this old clinical evidence of the development of tonic contractions in the course of a striatal affection is in no sense diminished by our inability to attribute them directly to striatal cell-fibre systems. I have myself from time to time observed analogous cases, though they have never been reported. One may be selected for illustrative purposes; the patient came under my care in 1923, two years before the publication of Wimmer's interesting paper.

A boy, 5 years old, noticed to have been more or less "fidgety" since birth, but with no previous illness except German measles, began to suffer from indefinite involuntary movements with a propensity to affect the left limbs and side. They offered some resemblance to choreo-athetosis without being quite characteristic of either subgroup. After a year or two it was observed that, following any insignificant jar, knock, fright, or excitement, the boy lost power down the left side for two or three minutes; the left leg inverted and assumed an equinus position, while the left arm flexed at the elbow and was held in front. Unable to speak, he did not lose consciousness. The attack was very often (not, it appeared, invariably) preceded by a sensation of pain starting in the left foot and passing up the trunk to the shoulder and then coming down the left arm to the hand. When the sole of the left foot was stimulated (as in testing the plantar response) an inversion spasm of the limb took place, while pain was experienced and shot up as high as the left shoulder.

On minute examination, however, no definite objective sign of organic nervous disease was discovered.

The boy has been under my continuous observation now for more than seven years, and I have seen a number of the tonic attacks, which can be thus succinctly described: the left foot extends and becomes inverted, the left leg is in tonic extension, the head is bent over to the left and the trunk arched slightly, with concavity to that side; the arm is flexed at elbow, somewhat abducted at shoulder, and stiff throughout, while the fingers are commonly flexed. Some spasm of the left orbicularis oris is observable, but the mouth is not drawn over. Consciousness is preserved; the boy does not drop anything he holds in the affected hand, yet does not answer questions while the attack is on. Its duration has gradually become shorter, now lasting no more than thirty seconds. By 1929, at length, signs of change apart from the fits made their appearance, in the form of enduring rigidity and some weakness of the left leg; and at the present time the facies and ensemble are indicative of a mixed pyramidal and extrapyramidal affection—a slight unilateral paresis and equally slight suggestion of Parkinsonism.

On purely theoretical grounds this unusual case can be represented as one of *reflex unilateral tonic epilepsy*, and feebly be thought *infracortical* in origin, mainly, no doubt, because of its tonic type; but I do not myself consider the symptoms can be attributed to excitation of striatal arcs. The aura follows a physiological "march" corresponding to the arrangement of centres in the post-Rolandic cortex, while the extension and equino-varus postures of the left leg are of pyramidal type. Pain of cortical derivation is not unknown, and the reflex nature of many of the fits rather favours a similar site for them, as I think; at least, the element of conscious perception of the stimulus is not excluded from acoustico-motor and other reflex epilepsies adjudged by some to develop at lower levels. But still, the absence of the clonic element impresses, even if strict proof is wanting of the striatal or extrapyramidal locus of the spasms. In this connexion we do well to remember that when in Jacksonian epilepsy the "second side" becomes implicated, its contractions are sometimes tonic rather than phasic, and take the whole side much more quickly than in the case of the one first involved. I have had under care recently a little girl whose epileptic attacks throw the left limbs and face into clonic spasms, with head and eyes deviated to the left, but the right arm and leg remain in tonic extension, and the abdominal muscles of the two sides are boarded in strong tonic contraction equally, without twitches. Until further study enlightens us it is premature, in view of what has already been said by way of criticism of the "striatal" conception, to assign tonic manifestations to activity of the basal ganglia when we know they can also develop in other ways, as is now to be discussed.

Tonic ("Cerebellar") Fits.

The occurrence of general tonic seizures (leaving aside the mainly segmental and unilateral tonic spasms to which some attention has been given above) has long been a neurological commonplace. Described by Hughlings Jackson as "tetanus-like seizures," sometimes as "cerebellar fits," in their essence they consist of tonic contraction of extensor groups of trunk and limbs, with temporary head retraction and opisthotonos; occasionally the upper extremities are flexed rather than extended, and emprostotonos may take the place of the other. A full description of a number of such cases was published in my paper²⁷ entitled "Decerebrate rigidity in man and the occurrence of tonic fits" (1920), and various others gathered from a somewhat sparse literature were referred to. Since that time more have come under personal observation, while from his wide experience Foerster²⁸ has collected a series of the same kind; Martin,²⁹ too, has published an interesting quartette of cases of pure tonic fits in association with syringomyelia, attributing them with some plausibility to rise of intracranial pressure and internal hydrocephalus—no uncommon finding in the disease.

Speaking generally, haemorrhage, abscess or tumour of the cerebellum, or of pons, medulla, and cerebral peduncles, haemorrhage or rupture of an abscess into the ventricular system, meningitis, and other basal conditions calculated

to interfere with fluid circulation between that system and external arachnoid spaces, are among the pathological conditions apt to be causative of generalized tonic fits. Their association with a state of decerebrate rigidity is far from infrequent; in such cases they occur as temporary exacerbations, accentuating and exaggerating the background of tonic posture in extension. Again speaking generally, it can be shown that fits distinguished by the assumption of tonic attitudes and the appearance of tonic contractions are most likely to develop when the lesions of the case dissociate physiologically the cerebral hemispheres from cerebello-mesencephalo-pontine levels, and when, therefore, striatal function also is in complete abeyance. An instructive case which bears out this statement indirectly has been recorded by Reynolds⁴⁰; it is that of a girl of 15 whose grand mal attacks, before any operation, consisted of "(1) tonic emprostotonos, in which she remained rigidly anteflexed and silent for about twenty seconds; (2) as the trunk relaxed, the head and eyes commenced to jerk clonically to the right and the right hand entered into clonus. Then (3) the movements became slower and ceased, and she wept as if frightened," etc. He was able, by operating and draining the cerebral cortex above the tentorium, to dispel the clonic element altogether and leave only the basal tonic element; at this stage the fits continued as purely tonic and emprostotonic in character for two weeks; after some twenty of these, bilateral suboccipital drainage of the arachnoid space immediately and permanently dispelled this residuum, and neither type of fit has recurred since.

In their elaborated form the picture is unmistakable, especially when evidence of coexisting organic nervous disease of one or other type as alluded to above is at hand. But brief tonic fits, not much more than a rather elaborate petit mal seizure, also occur, and deserve mention because of their unusual features. A recent case is that of a girl of 14, whose so-called "menstrual epilepsy" is as follows: a sudden inspiratory cry is succeeded by stretching out rigid arms in front while neck and back are contracted in powerful opisthotonos and she falls back with a crash, legs extended and stiff; cyanosis at once sets in, and involuntary micturition takes place. With almost equal abruptness the musculature relaxes, consciousness returns, and violent vomiting ends the attack. No clonic movement of any kind has ever been seen.

The close resemblance of the fits in this case to those of the patient of Wimmer (mentioned above) should not be overlooked; the latter comprised initial cry, generalized tonic contractions with loss of consciousness, cyanosis and tongue-biting, without, however, sphincter relaxation. Yet Wimmer, who himself diagnosed ordinary epilepsy at first, was influenced by the subsequent development of torsion-spasm and called the condition "striate epilepsy" accordingly. Since variants of this kind are encountered by themselves, "idiopathically," with undiscoverable causation, it appears to me improper, and for the reasons already advanced unjustifiable, to allot them any such topical designation.

At this point, too, another aspect of the question of tonic fits may not be out of place. The neurologist is aware that convulsive hysterical seizures no longer constitute a clinical banality. Nothing should appear to any thoughtful mind more evident than that hysteria follows the scientific movements of the day and adapts itself to progress in medicine. Hysterical fits in the grand manner are out of fashion. Years have elapsed since last I saw a typical case of the *arc de cercle*, and I remember that Babinski showed at a medical congress a cinematograph record of all the phases of the attack because he considered it would ere long become of purely historical interest! Now while hystero-epilepsy may assume any form, as it were, undoubtedly one special class of fit is distinguished by the patient's musculature becoming contracted in rigid extension, head retracted, back curved, arms extended and hyperpronated, legs extended, and feet inverted. Here I am interested not in the possible psychogenesis of the attack, but in its physiology. For there can be no doubt that it offers intimate affinities from the standpoint of *form* with what obtains in fully developed tonic fits attended

by physiological decerebration. We commit ourselves to no theory of hysterical etiology in underlining the fact that the neurosis can have at its disposal only the existing sensorimotor processes of the individual patient; and it is legitimate to stress the symptomatological analogies between the two types when the *mechanism of production* of the symptoms is concerned. I well remember a hysterical patient who, when she entered the out-patient clinic, fell on her back in rigid extension, crushing her hat under the back of her head on the floor, toes down-pointed, arms straight out and pronated till the backs of her hands touched. Investigation revealed a wretched history of incest and psychical trauma, but the *form* of the attack is not explained thereby. The physiology of hysteria is one of many matters in neurology which call for serious research. I confess my ignorance of why convulsive hysterical manifestations partake so frequently of a tonic character; the once-debated question of the reality or otherwise of hysterical Jacksonian epilepsy seems now to be forgotten, presumably because it is thought to have been "decided" in the negative, but why then this mimicry of organic tonic fits?

Periventricular Epilepsy.

A final grouping of lowest level or infracortical fits includes those in which symptoms of an affective and visero-sensorimotor kind predominate. Everyone who has made a study of epilepsy knows with what frequency symptoms referable to the neuro-sympathetic system are in evidence; momentary arrest of the heart's action, pallor, sialorrhoea, sphincter relaxation, and other analogous phenomena occur to the mind in this connexion. As the "discharging lesion" sweeps over different physiological levels or implicates a succession of physiological centres it does not appear in major cases to exercise any selectivity. Whether this is through representation of visceral activities at the highest physiological level need not at present detain our discussion; anatomically speaking, we know of sympathetic centres in the brain representing lower nervous arrangements, but so far they have not been traced with certainty to the cortex itself. We shall therefore take the attacks to be now considered as indicative of functional activity on an infracortical plane.

In the spinal cord a general division of cell-fibre groups into four systems or columns is obvious—namely, somatic sensory, somatic motor, visero-sensory, and visero-motor respectively. These are readily recognizable at prespinal levels—namely, in medulla and pons—and the point of interest to which attention is directed resides in the relation of the visceral and somatic-sensory columns, particularly, to the ventricles of the cerebrum. At the level of both medulla and pons they fringe the margins of the fourth ventricle; the thalami are lined by the third; and researches of recent years have thrown a great deal of light on the various visceral ganglionic collections skirting the floor and lower sides of the same third ventricle, as far back as the commencement of the aqueduct. That the major portion of these centres, concerned with *la vie affective* and *la vie végétative*, is situated round the ventricular system cannot be without significance. It is therefore permissible to speak in general terms of "periventricular epilepsy," by which may be designated cases remarkable for localization of the disorder in neuro-sympathetic centres, at least in major degree, to the exclusion (absolute or relative) of sensorimotor phenomena of somatic type.

The actual nucleus or focus of the "nerve-storm"—the "storm centre"—cannot perhaps be determined with any nicety, for if we are to conceive of the sympathetic system as an anatomical continuum (in accordance with the conclusions of Philip Stöhr⁴¹ and others) it becomes difficult to ascertain whether peripheral or central cells form the starting-point; besides, as has been remarked in a previous lecture, we must postulate an open way between the focus and the percipient cortex, since in few of the seizures to be described is consciousness ever lost; on the contrary, the subject is only too acutely aware of some violent disorder referred to his viscera, and equally incapable of inhibiting it. The assumption must be that abnormal impulses ascend

to the cortex and are there interpreted as accurately or (for most instances) as imperfectly as may be, in accordance with previous experience or with want of it.

1. Prominent in the general category of affective or visceral epilepsies come the "fits" known (after Gowers) as "vaso-vagal attacks," or "pseudo-angina," or popularly as "nerve-storms"; in their physical concomitants they are akin to what the psychiatrist is nowadays in the habit of designating "anxiety neurosis." Cases exhibiting the syndrome to be described immediately have interested me for many years, and I have collected a large number from private and hospital practice; and in an article⁴² on "epileptic variants" the subject has been given attention and analysis.

Briefly, to quote from that paper, "the patient complains of attacks of palpitation at the heart, difficulty in breathing, a suffocating sensation in the throat, hot flushes, icy coldness, sweating, shivering and trembling, nausea, and other epigastric sensations, and often, also, an indescribably distressing feeling of impending trouble, of fear, even of imminent dissolution." The symptoms of the vaso-vagal attack point in a general way to temporary functional disturbance referred to organs supplied by the pneumogastric nerve, presumably initiated in the vagal nuclei of the floor of the fourth ventricle and in the neighbouring vasomotor nucleus. But radiation along neuro-sympathetic paths therefrom is usual enough; in particular, polyuria and pollakiuria are very common at the close. One or two hitherto unpublished clinical cases will serve for purposes of illustration.

A. C. B., male, aged 58, suffers from fits commencing with flatulence and cructations, followed after a time by a sinking sensation at the epigastrium, thudding heart's action, vertigo, nausea, clammy sweating, and trembling of the body and limbs; an agonizing dread of something unpleasant about to happen develops when the attack is in full swing. Its duration is perhaps from fifteen or twenty minutes to two hours, during which (if it is rather prolonged) he will pass water freely, quite six times or even more. It may amount to a pint of clear, limpid urine the first time, but subsequent quantities are on a smaller scale.

This patient has a persistently low body temperature, ranging from 95° to 97.4° F.; on none of the various occasions when it has been taken by a medical man has it been higher.

Interesting features distinguish another case of the same class.

G. N., male, aged 47, has attacks in series, four to six a day for five or six days, with an interval of weeks thereafter. Lasting for about thirty seconds, they begin with a sudden "jump" at the heart, "as though it were coming into my mouth," and to this succeed a peculiar feeling in the finger-tips, which seem "oily and smooth," icy coldness (even the inspired air seems somehow for the time colder than usual), giddiness, pallor, trembling, and inability to speak. As the attack finishes an overpowering "nightmare" of depression seizes him, making him feel frankly suicidal. Every hour (for hours at a time), when the serial fits develop, he voids clear watery urine in quantity.

Especially worthy of note is the fact that in this case the "turns," which have continued for some twelve years, were at first invariably heralded by an unpleasant, "rotten" taste in the mouth, bringing with it a strong feeling of familiarity ("I have tasted this somewhere before"), and a peculiar dreamy sensation (in itself not unpleasant) "exactly as if I were going under gas." For the last two years, however, this uncinete aura has not recurred. To the student of the subject its association with periventricular epilepsy must appear highly informative, for in the first place the fact that a "visceral" or "affective" attack follows stamps the latter with the epileptic seal and justifies its acceptance as an epileptic variant; and, in the second, the radiation from temporo-sphenoidal to ventricular locus exemplifies an unusual line taken by physiological excitation or release, and suggests the possibility of still others.

Another instructive association is illustrated by the next case.

Mrs. B., aged 59, exhibits an old left juvenile cerebral hemiplegia, dating from the age of 12. For the last year she has suffered almost daily from attacks which commence with a sensation of "something moving or fluttering in the stomach"; this

"something" passes up the body into the left arm, where it becomes a "burning crawling feeling," "as if my arm were being gripped round with red-hot wires"; the sensation extends to the tips of the left fingers, and is described as if "something stinging were trying to get out through the skin." At the same time palpitation starts, linked to a vague fear of impending evil; the patient feels sick and giddy, shivers all over ("I can't keep a nerve still"), and perspires clammy, but solely in the left upper limb, arm, and hand, never on the right. Immediately after the fit she passes a large amount of clear urine every half-hour for usually three or four times.

The interest of this case consists mainly in the peculiar connexion of visceral and somatic sensory auras; the initial vagal aura (stomach and heart) radiates over to a cortical sensory area—presumably a *locus minoris resistentie*—whence in consciousness is experienced a definite Jacksonian sensory aura confined to the affected left arm. The fact of the sweating being restricted to that somatically involved limb is equally noteworthy. Within the neuraxis impulses can spread by unexpected and unsuspected routes in particular circumstances. Several clinical examples of the association of migraine, for instance, which is not supposed to be of periventricular origin, with these visceral attacks have come under observation, and one may be cited.

D. B., female, aged 35, unmarried, has suffered from sick headaches for years. These take a more or less classical form, with persistent vomiting, visual phenomena (not, however, teichopsic), unbearable headache, tenderness of scalp, photophobia, etc. They last usually for forty-eight hours. In the course of the migraine (which is always preceded by a day or two of pronounced physical and mental eutonia) the patient's hands and feet become icy cold and the head hot; palpitation and anxiety develop, and a mild degree of sweating. At this stage she invariably keeps passing water at short intervals, up to as many as twelve times in succession.

Though just an episodic syndrome, an epiphenomenon of the migraine, its interest resides in the linking together of cerebral centres by paths not commonly open to traverse.

Finally, in this connexion, I must allude to a recent case which illustrates still another of these combinations, and that along distinctly unusual lines.

C. C., male, aged 23, unmarried, has suffered from peculiar attacks for six years, at irregular intervals. They commence by a sensation at the heart ("as if it were moving round in a circle" to the accompaniment of "painful pricks") and in the stomach (the organ seems to "turn right over"); and the usual sequence of giddiness, shaking, hot waves, etc., is experienced in rather severe degree. Thereafter the patient feels extremely sleepy, and sometimes actually falls fast asleep. He has often observed that the fits are brought on by unrestrained laughter, and sometimes, too, by his being tickled; at such a moment he is suddenly seized by the pain at the heart, and the syndrome forthwith unrolls itself.

To my mind the case is deeply significant; no similar instance of the provocation of the vaso-vagal fit by the emotion which causes laughter has hitherto come under my notice. Clearly it furnishes a link on the one hand with narcoleptic semeiology, in the association of laughing and sleep; but while there is no evidence of cataplexy on the other, yet the production of a periventricular fit by a stimulus which in other circumstances gives rise to the cataplectic attack undoubtedly narrows the gap between narcolepsy and epilepsy. I have seen two cases of epilepsy in which the aura was one of motiveless and compulsive laughter, and there are instances on record of epileptic fits having been induced by that act, itself resulting from tickling the soles of the feet (Féré⁴³ quotes those of van Swieten, Esquirol, and Reynolds). Since in by far the greater number of cases of vaso-vagal fits the manifestations are attended by a feeling of apprehension, anxiety, or even of terror, it is curious to observe their origination in an emotion of a pleasurable kind. The mechanisms "let go" by the act of laughter deserve physiological study; its visceral concomitants include dilatation of capillaries (the skin reddens and glows); glandular secretion (the eyes sparkle; we laugh "till we cry"); vulgarly, also, we laugh until sphincter relaxation is imminent. We nearly "die of laughing"; we are "tickled to death." Is this seeming clinical novelty, then, akin to the last of these? The patient laughs or is tickled into laughter, till suddenly affected by a paralyzing disorder of his cardiac

and respiratory mechanisms. I confess I find his case of peculiar scientific interest, but am content at present to sketch it in outline.

2. Less definite in constituent features, but equally suggestive of periventricular dysfunction, are cases distinguished, *inter alia*, by yawning. One may be taken from a previous communication¹² by way of example.

C. L., male, aged 25, has had peculiar fits for about one year, recurring on an average at fortnightly intervals. They are constituted by continual yawning, with constant voiding of urine, and are followed by violent giddiness, headache, and occasional vomiting. A family history of epilepsy has been obtained, but no organic signs have been seen on repeated examination.

One may perhaps hazard the speculation that these phenomena represent some disorder of periventricular visceral centres concerned in the regulation of sleep and of fluid interchange, with radiation to other visceral centres situated in the floor of the fourth ventricle.

3. Another rather unusual but interesting visceral association is provided by those cases of ordinary epilepsy which appear to have as a regular accompaniment *rise of temperature*. I do not here refer to the fever which develops when repeated severe general fits occur, as in status epilepticus, but to individual attacks, separated it may be by long intervals, in which elevation of temperature is found. Doubtless some such cases are simply explained by the temporary occurrence of some intestinal toxæmia accountable for both the fit and the fever, yet in other instances the question arises whether the latter may not be of neural origin. I have in mind the case of a schoolboy of 16, who for two years had had an occasional epileptic fit; in each instance associated with muscular effort (wrestling, swimming, running, and in the gymnasium), the attack consists of brief loss of consciousness, pallor, and involuntary micturition, without any convulsive movements (tonic or clonic) being in evidence. On each occasion the temperature has shot up after the attack to 103° or 104° F., coming down the same day, or the next, as suddenly as it has run up. No ready explanation for this occurrence has been discovered, even with the most careful search over the somatic systems. At the most, a severe generalized fit will not raise body heat more than half a degree. For the above and one or two somewhat similar instances it seems conceivable that a neural (neuro-sympathetic) mechanism is implicated.

4. When the semeiology of still other epileptic varieties is carefully scrutinized the fact that they appear to consist of little else than *isolated neuro-sympathetic disturbance* must be allowed. The remark is clearly apposite in respect of many examples of petit mal, so-called. Cases which the clinician has great difficulty in classifying, the choice lying between "faint" and "fit," belong here. Syncopal attacks are in essence visceral, and often indistinguishable from forms of fit; if of circulatory etiology they are considered "faints"; if of neural, they are "fits." But here, too, we may be labouring a distinction without a difference. An elderly gentleman has suffered from rare major epileptic fits for many years; since he has become slightly arterio-sclerotic they have ceased, but what he calls "sensations" have taken their place. These recur nearly every day. He was seated in my consulting room and my blood pressure instrument was on his left arm; asked a question about his old school, to my surprise he vouchsafed no reply; grasping the table with his left hand, and the arm of the chair with his right, he sat motionless, turned a greyish colour, and his respiration immediately accelerated in a rapidly rising series of ever shorter and quicker breaths, becoming slower again in about half a minute and the arrhythmia ending with a deep sigh. He immediately remarked, "Oh, I was at such and such a school," naming it as though he had not been aware of the seconds that had thus passed. Petit mal, of course, is merely a descriptive epithet; but, neurally considered, the symptom-complex in the case was surely limited to the visceral ganglia of the floor of the fourth ventricle, a disturbance there (whether vascular or neural I know not) bringing in its train that transient clouding of consciousness known as an "absence épileptique." Isolated escape of this and

analogous kinds represents on occasion all that there is of epileptic semeiology.

If we could but discover the secret of inhibition and of de-inhibition, if we had some scientific means of reinforcing the former at the moment when escape from control is imminent, could we but determine that switching off of the current which in some patients can be effected by potent stimuli aroused through other channels at the crucial moment, our treatment of epileptic episodes would not be so nugatory as it often proves to be.

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STREPTOCOCCAL SEPTICAEMIA AND APLASTIC ANAEMIA.

CASE REPORT.

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THE patient whose case we record was a girl aged 18; she had never been robust, though she had enjoyed moderately good health. The tonsils had been under suspicion owing to a tendency to sore throat. On the evening of November 13th, 1929, there occurred pain over the left costal margin. The temperature was 104°. This kept up and was of the high remittent type when seen by two of us (A. W. R. and A. G. G.) on November 16th. Nose bleeding and ulceration of both tonsils had occurred on the 15th. She was a well-made girl, well nourished, coughing up blood, which apparently came from the nose. She was not unduly anaemic. The left cheek had a slightly bluish tinge. The pulse was 118, respirations 20; the fingers were not clubbed; the skin was normal in texture and clear of blemish or petechiae; there were no glands to be felt in the neck, axillae, or groins. In the chest there was a slight diminution of breath sounds towards the left base behind, but no interference with movement and no signs of fluid. There was nothing definite on examination of the abdomen. On November 19th the urine contained albumin; blood, and a few casts, with a moderate number of long streptococci. The signs in the left side of the chest were unaltered, but the left lumbar region was tender, and, though it was difficult to feel, the kidney appeared to be enlarged. The epistaxis continued until November 21st, and the albumin in the urine persisted.

On November 25th the temperature was of the high remittent type at the 102° level, and the patient was markedly anaemic. The sclerotics were greenish, as was the tint of the skin. The left lumbar and hypogastric region was tender, and one small petechial haemorrhage was noticed under the left breast. There were no signs of infective endocarditis nor of severe renal disease, and the blood in the urine had disappeared, leaving a trace of albumin only. The fever persisted; she became extremely ill with low muttering delirium at times, restlessness, and a return of the epistaxis on November 27th and 30th. She was treated by iron (Blaud's pill) and antistreptococcal serum (seven injections each of 10 c.cm., four of which were concentrated serums) on every second day and adrenaline chloride 10m daily. On December 5th she was given a