ball entered one inch to the left of the middle line, and one inch above the sternal end of the clavicle. Exit on the posterior part of the right arm, at the middle line, two inches below the axilla. The ball passed in front of the trachea, broke the inner half of the right clavicle, went in front of the vessels of the neck and the subclavian artery, in front of the axillary artery, and below the humerus,—speaking of that bone as raised and abducted at the time. When hit, he thought his arm was shot off. It dropped, the gun fell, and, screaming that he was murdered, he staggered, bleeding freely, and soon fell unconscious. When a little later he revived and raised his head, a second ball struck him in the right temporal fossa, and emerged through the right eye. He jumped up, ran a little way, and fell once more. When hit, he lost all motion in the limb, which became numbed, but felt no pain. Two weeks later, feeble power to move returned gradually in the elbow, shoulder, and arm, and after two months in the wrist and hand.

Treatment.—Cold-water dressings and means to relieve burning, but all ineffectual. The joints became swollen early, and the arm bent at a right angle. The hand, dependent, lay across his chest during a long period. He made some attempts at passive motion as he found the hand becoming stiff, but no great good was thus gained; and, as the contractions took place and the joints grew worse, the wrist became moulded to the curve of the chest, on which it lay.

About the tenth day, burning pain began in the palm and fingers, especially in the cushions of the fingers and the knuckles. It was at its worst a month later, and remained thus another month, after which it grew less. When at its height, he suffered from sound louds, vibrations, and dry contact. The rubbing of his boots on the floor was the greatest annoyance, and this he relieved by wetting his stockings. Since October, four months after he was wounded, it has been unaltered. Sensation, little affected at the onset, has undergone no change of moment. Voluntary motion, which grew better for awhile, suffered anew and increasingly as the nutritive changes developed themselves. When they first arose we have been unable to determine.

Pain.—In August, 1864, he began to lose the violent pain. It was not gradual, but one day he noticed suddenly that his glove was dry, and yet he could use his hand well and without pain. It was not entirely gone, and he continued to wet his hand for some months; but it grew much better. Even now he feels dry rubbing in the palm of the hand and down to the finger-tips, and a loud noise, such as a wagon making a great noise in passing, or a sudden emotion, as seeing a person fall, etc., makes the same impression. In the left hand there is no pain.

XXVIII  Jacksonian Epilepsy

JOHN Hughlings Jackson (1835 to 1911), the father of British neurology, had a special interest in focal motor epilepsy. As a reporter for the Medical Times and Gazette and later as a physician at the London Hospital and the National Hospital in Queen Square, he studied patients with this disorder and continued to ponder its meaning. His interest was reinforced by the fact that his wife suffered from focal convulsions before her early death.1,4

Proceeding from his analysis of focal epilepsy, Jackson was then able to formulate a series of hypotheses about the organization and function of the entire central nervous system.4-14 These interpretations were based entirely on clinical observation and reasoning; he left the experimental proof of his assertions to others.

Epilepsy had been studied by physicians for more than 2,000 years before Jackson's time, and focal seizures had been described occasionally as medical curiosities.11-15 In 1827, J. F. Bravais published a description of focal motor seizures in his thesis for the doctoral degree from the Faculty of Medicine of Paris, but he offered no suitable explanation for this phenomenon.21,25-27 Subsequently, Richard Bright and Robert Bentley Todd observed similar cases in England,23 but at that time epilepsy was generally thought to be spiro-medullary in origin.15 The electrical excitability of the cerebral cortex was not established until the pioneering research of Gustav Fritsch and Eduard Hitzig in 1870.28,29

Bentley Todd, who was a distinguished physician in London,29,30 also described the occurrence of post-epileptic hemiplegia and monoplegia in some of his patients. This phenomenon, though previously mentioned by Bravais and later studied by Alexander Robertson,31 is now known as Todd's palsy.32,33,34

Because of analogies with apoplectic hemiplegia, Hughlings Jackson reasoned that the lesion responsible for focal motor epilepsy lay within the territory of the middle cerebral artery. He erred in assigning too much importance to the corpus striatum, but he correctly deduced that focal convulsions are the result of sudden discharges of partly damaged and unstable gray matter in the contralateral cerebral hemisphere. In addition, he inferred that the movements of muscle groups are represented on the brain's surface.

The accuracy of Jackson's reasoning has been proven during the past century by extensive evidence from the fields of pathology, neurophysiology, and electromyography.29,38 Although his writings show little interest in individual patients or in treatment, his insights into pathophysiology provided a rational basis for later advances in the understanding and control of epilepsy.

References

A STU dy of CONVULSIONS.*

By J. Huglihes Jackson, M.D., F.R.C.P.

A convulsion ismodel but a symptom, and implies only that an excessive, and a disorderly discharge of nerve tissue on muscles.

This discharge occurs in all degrees; it occurs with all sorts of conditions of ill health, at all ages, and under all sorts of circumstances. In this article I shall narrow my task to the description of one class of chronic convulsive seizures. The great majority of chronic convulsions may be arranged in two classes:

1. Those in which the spasm affects both sides of the body almost contemporaneously. In these cases there is either no warning, or a very general one, such as a sensation at or about the epigastrium, or an insensible feeling in the head. These cases I shall call "epileptic," and sometimes cases of "genuine" or "idiotical" epilepsy.

2. Those in which the fit begins by deliberate spasm on one side of the body, and in which parts of the body are affected one after another. It is widely held that both class only that I intend to deal in this article.

But although I thus limit myself to one class of cases, I contend that the title of my article is correct, and that I am studying the general subject of convulsion methodically when I work at the simplest varieties of occasional spasm I can find.

Cases of unilateral convulsions are unquestionably the most common, and of which I have principally present at a paroxysm, watch the march of the spasm. I have known a fit of this kind last ten minutes. . . . For instance, we may first see one muscle of the right hand, then the base of the whole arm, then of the face, leg, &c. Besides, patients can describe the onset and much of the march of such seizures. We can therefore compare and contrast these convulsions with hemiplegia—which form of palsy the convulsion not infrequently leaves. In some of these cases we find great disease of the brain . . . post-mortem, and thus we can infer the seat of the minute changes on which the discharge producing the spasm was dependent. This we have, . . . on the one hand a record of the events occurring in a certain kind of convulsion, and on the other hand knowledge of the internal part diseased. We are freed, therefore, from the great vagueness of the word "epilepsy." We do not care to say that a tumour of the brain (or minute changes near it) "causes" epilepsy, but that changes in a particular region of the nervous system—say in the region of the left middle cerebral artery—led to convulsions, in which the spasm began in the right hand, spread to the arm, attacked next the face, then the leg, &c.

I chiefly wish to show in this article that the most common variety of hemiplegia is a symptom of one of the same region of the brain as in the symptom hemiplegia; viz., the "region of the corpus striatum." The term loose region of the corpus striatum" is advisedly used. Hemiplegia shows damage (to the extent of destruction) of the motor tract; hemiplegia shows damage (equivalent to changes of instability) of the convolutions which discharge through it. Palsy depends on destruction* to the head of the pyramidal tract. As the convulsions are rich in grey matter I suppose them to be to blame in severe convulsions at all events; but as the corpus striatum also contains much grey matter I do not see how this can be the part to blame in so slighter convulsions. Indeed if the discharge does begin in convulsions, no doubt the grey matter of lower motor centres, even if these centres be healthy, will be discharged secondarily by the violent impulse received from the primary discharge. Nor does both the corpus striatum and many convolutions—are supplied by one artery, the middle cerebral or Sylvian, and this artery circumscribes the region I speak of. These muscles which suffer more easily are less likely to act independently of their fellows of the opposite side. Those which must act with their fellows of the other side—for instance the intercostals—do not suffer at all; and those which are, to speak, half way in their action—e.g., muscles which turn the two eyes and the head to one side—suffer only in very large lesions, and then but for a short time, a few hours or days. It is but putting these facts in another way to say that parts suffer directly as the actions they engage in are voluntary, and inversely as the action they engage in are automatic. This is seen in the order in which the muscles move in the more automatic actions recover first. Now just the same principle applies to cases of hemiplegia, so far as this at least, that the fit begins most frequently in sequence of the parts which suffer next it. This point is now to be considered in some detail.

First beginning unilaterally may double itself by movement in any part of the region which is implicated in hemiplegia, i.e. in the face, in the arm, or in the leg. But I know few cases of this class which begin other than in the side of the face, (usually the cheek), in the hand, or in the foot. They very rarely begin in the upper arm, or in the calf. The fit usually begins, it is to observed, in that part of the face, of the arm, and of the leg, which has the most used art. . . .

Fits beginning in the hand are common, fits beginning in the cheek and tongue are less frequent, fits beginning in the foot are rare. . . . the fits which begin in the hand begin usually in the index finger and thumb; fits which begin in the foot begin usually in the great toe.

*The word "destruction" is scarcely the correct word to use, for it is not meant that the nerves in the region are necessarily broken up, although they often are in palsy. But simply that there is a change in them which destroys their function. Thus, . . . palsy is supposed to follow a convulsion because the area of the inner root of the nerve is lost its function, from the effects of the excessive quantity of nerve force sent out from this region; or because the occupation the word destruction may conventionally be used.
Parts which have the most varied uses will be represented in the central nervous system by most ganglion cells. I say most varied movements, as it is not only a question of number of movements, but also of number of different movements.

We shall speak of three varieties of convulsions beginning unilaterally—
1. Those beginning in the hand.
2. Those beginning in the face and tongue.
3. Those beginning in the foot.

The seizures occur in all degrees. . . . There are not merely degrees of more or less severity of asepsis. The point of significance is that the spasmodic movements are not conterminous, but follow a distinct march, and a different march according as the asepsis begins in the hand or in the foot. The sequence is, however, not simple. The asepsis does not affect the arm, then cease, next affect the face, &c. It is a compound sequence. For instance, the face begins to be affected before the arm of the arm ceases.

When observing the paroxysms we have therefore to note two things.

First, the region affected; for instance, we see the face, arm, and leg of one side are in asepsis.

Secondly, the order in which past parts are involved; for instance, in the same manner in which the hand, then passed up the arm, then affected the face and lastly went down the leg—"out at the toes," one of my patients said.

REMARKS ON EPILEPTIC HEMIPLEGIA.

The cases I am describing are those cases of chronic convulsions which are so often followed by hemiplegia. It is the epileptic hemiplegia of Dr. Todd. I do not know how it is that some patients have no asepsis after these seizures, and some have. The same patient may have hemiplegia after some of his seizures, and not after others. The presumption is that the degree of asepsis depends on the severity of the convulsions, i.e. on the quantity of discharge. When the convolution is limited in range, the asepsis by it is limited in range. . . . I have recorded a case ("Medical Mirror," September 1869), in which asepsis of the arm only followed a convulsive seizure—the asepsis, according to the patient, being limited to that limb. In this case, there was a new growth in the hemisphere in the hinder part of the superior frontal convolution. . . . When the fit is severe, there may he hemiplegia complete in range, except perhaps for deviation of the head and eyes. But the hemiplegia, however complete in range, and however decided in degree, is transitory, and we may very safely tell our patient that it will pass off in a few days or weeks, and we may usually say so when we feel certain that the fibs are the result of organic disease in the head. The asepsis does not depend directly on the organic disease . . . but doubtless the result of the "overwork" of nerve fibres which pass from the part discharged to the muscles convulsed. The nerves and the muscles require time to recover from the effects of the sudden and excessive discharge.

But although we can assure our patient that his asepsis will pass off, we shall be obliged to confess that both his fits and the asepsis will probably return again and again.

It is not said that hemiplegia after a convolution is transitory, but that hemiplegia after a convolution deliberately beginning unilaterally is transitory. Hemiplegia after a convolution may signify large cerebral haemorrhage destroying the motor tract, and then the asepsis is permanent, or it may signify plugging of the middle cerebral artery.

ARREST OF FITS BY THE LIGATURE.

There can be no question that the ligature is a most valuable means of arresting such fits. I have known very great success from this procedure in Brown-Sequard's practice, and also from another plan he adopted, founded on the principle, viz, circular blisters—a garter of blister round the limb.* Probably the ligature, &c, merely put off the explosion; and the asepsis, or subjects of this, as of other varieties of fits, very often say that they feel better after a seizure—after a full discharge of that part of the nervous system which is unstable. It indeed occasionally happens that an epileptic complains more to his doctor when his fits are diminished in number. It may be that when his serious troubles are lessened, he thinks of the smaller ones. But I suppose that before the psychic explosion which constitutes the severe fit there are frequent minute discharges—too trivial to produce any visible effects, but enough to cause discomfort to the patient. Never-
one-sided spasm, or spasm beginning in one side, implies local change in the central nervous system as surely as one-sided palsy does . . .

Let us suppose that a square inch of convolution is diseased. If this part were destroyed, there need be no symptoms; but if it be not destroyed, but unstable, there must be symptoms—for it will discharge on muscles when its tension reaches an unstable equilibrium . . .

And even in those cases where we do find a lump in the brain . . . we do not discover the symptoms which the discharge depends. The lump does not discharge, but some ("softerened") part of the brain near it—which part cannot be destroyed or it would not discharge at all, but which part must be diseased or it would not discharge so much, nor in so disorderly a manner, nor on slight provocation . . .

It is held by some that the coarse disease, although it lies in the cerebral hemisphere, is quite as much an eccentric cause of a fit, as a worm in the duodenum; and that in both the medulla oblongata and pons are the centres which discharge. (I do not deny that grey matter in these parts is secondarily discharged.) When we consider that the hemiplegia left by the fit I describe is like that following destroying lesions in the Sylvian region, and is not like that following destroying lesions in the pons or medulla oblongata, it becomes, I submit, infinitely more probable that the primary discharge is of grey matter in the region (Sylvian) in which the coarse disease is dischargeable.

2.—The functional nature of the change in nerve tissue . . .

If I am told that hemispasm is "only a symptom," and may depend on "many causes," I admit it in the sense that various pathological processes may lead to that instability of nerve tissue which permits an occasional excessive discharge on muscles; but from the point of view of function there is but one cause of convolution, viz., instability of nerve tissue. Of course there will be varieties of range of convolution, degrees of instability, degrees of quantity of nerve tissue unstable, and, more important than all, degrees of evolution of the nervous processes (near to and further from the motor tract) which the pathological changes render possible.

3.—The pathological processes . . .

(a) Emolism.—It is not very uncommon to find when a patient has recovered or is recovering from hemiplegia, the result of emolism of the middle cerebral artery, or of some branch of this vessel, that he is attacked by convolution beginning in some part of the paralysed region, almost always, I believe, the face or the hand. I have, however, yet made a post-mortem examination on a patient whom I knew to have had fits of this kind after supposed emolism. It will be safer, then, to say that such seizures occur in patients who have recovered partially, or seemingly entirely, from hemiplegia occurring with heart disease, or with the parturient state . . .

(b) Coarse Disease.—It is admitted that it may be of any kind, but it so happens that, in nearly all the chronic cases on which I have had autopsies, the examination has revealed hypertensive disease of the hemisphere. The foreign body has been a phylactic nodule . . .

4.—Circumstances which determine the paroxysm.

Many things may discharge nerve tissue . . .

But I speak here only of chronic cases in which there is a persistent local lesion and an occasional discharge.

I think . . . that there are two factors in the production of a paroxysm—1st, Permanent local instability; 2ndly, Something which determines the discharge of the part unstable.

The part unstable "stores up" force, and when it reaches a certain degree of instability discharge of it is easily provoked. It may be that when by continuous nutrition it has risen to a certain degree of instability—it explodes, either "spontaneously," or in some normal periodical change in the body, or in some abnormal disturbance, the result for instance of fright . . . It falls then to a state of stable equilibrium, and once more by continuous nutrition rises to its former undue instability, when another explosion can occur. It is in short an exaltation of ordinary nutrition and function. I suppose that the provoking agents may be various—that many things will upset the equilibrium of the highly unstable nerve tissue . . .

All these general causes, I presume, act by altering the circulation in the head, during which alteration the equilibrium of the unstable patch is upset . . .

The usually accepted theory of the production of the paroxysm is that it is determined by contraction of arteries. (Brown-Séquard.) I have advanced the speculation that . . . the liability to the convulsions which I have described in this paper—those at least beginning in the hand—is due to persistent changes in the region of the middle cerebral artery, and that the paroxysm itself is owing to a local vascular contraction . . .

XXIX

Wernicke's Sensory Aphasia

IN THE Edwin Smith Surgical Papyrus, datting from the 17th century BC, an association was noted between temporal skull trauma and loss of speech. Nevertheless, before the work of Broca, the brain was generally thought to act as a whole, with no anatomical localization for the expressive aspects of speech.

In 1874, the German neurologist Carl Wernicke (1848-1905), published a short monograph in which he used simple anatomical diagrams to present a more comprehensive view of speech mechanism. Wernicke described five clinical syndromes that would be expected from lesions of (1) the afferent auditory pathways, (2) the speech reception center, (3) the association areas between the speech reception area, and (4) the speech expression center, and (5) the efferent speech pathways. However, he provided little pathological documentation of these theoretical possibilities. Of the ten cases he presented, only four had autopsy examinations, and the lesions involved relatively large areas. Furthermore, the illustrations in Wernicke's monograph depicted the right side of the brain; he did not stress the importance of the left cerebral hemisphere, though all four of his patients examined postmortem had left-sided lesions.

Despite these inadequacies, some of Wernicke's deductions have proven correct. Subsequent studies have demonstrated that lesions in the posterior half of the left superior temporal gyrus and the adjacent part of the middle temporal gyrus (Wernicke's area) may give rise to a receptive aphasia characterized by defective comprehension of spoken words, though less incorrect speech (jargon dysphasia), dysgraphia, and dyslexia.

The syndrome is now known as Wernicke's sensory aphasia, in honor of the man who first brought it to the attention of the medical world. Though the subject of aphasia is more complex than Wernicke envisioned, he shares with Broca the distinction of providing the first anatomical framework for our present knowledge of aphasia.

References