JACKSONIAN EPILEPSY.
A background and a post-script.

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In the year 1827 there appeared a thesis for the degree of Doctor of Medicine in the Faculty of Medicine of Paris. It was written by L-F. Bravais of Annonsay in the Department of Ardèche and was entitled "Researches on the symptoms and treatment of Hemiplegic Epilepsy"; it was based on the observations of the author on certain epileptic subjects in the Hôpital Bicêtre. This thesis proved to be, for those days, a close clinical study and record of a series of cases in which the convulsions were localized to one side of the body or even to one limb. Localized epilepsy was no new observation for it had been mentioned by Hippocrates, whilst Galen had found that the tying of a ligature round a limb served to prevent an epileptic attack provided that that limb was the site of origin of a seizure. These were early but isolated observations, and the immense literature of epilepsy gathering volume throughout the ages has little or no message for us because it was concerned with bare clinical records and with empirical methods of treatment.

Neuro-pathology had not yet been born, and so it comes about that many interestingly described cases are robbed of interest because no attempt was made to connect pathological cause and clinical effect. It was to be left to the magisterial brain of Hughlings Jackson to found not merely a natural philosophy for epilepsy, but to help to unravel the apparently insoluble mystery of the workings of the cerebral hemispheres. How he came to do this requires a statement of the state of knowledge, so far as we can reconstruct it to-day, at the time when he commenced his work. It is for this reason that reference has been made already to Bravais, and indeed Bravais' work calls for more than passing comment. for French authors in particular are apt to designate focal epilepsy as "épilepsie Bravais-Jacksonienne." We ought, therefore, to see what proper claim there is to the linking of one name with the other, how far the views of the one were held by the other, what new thing Jackson had to add to the observations of a young man who antedated him by nearly forty years.

The reader who turns to Bravais' thesis in the hope of finding gold (and who does not turn to some old thesis without keen anticipation of discovery) will be only partly rewarded. This paper is none the less an important one; it is the first serious study of a particular type of epilepsy, and the recognition of one variety as worthy of a special memoir marks the beginning of modern conceptions of convulsive attacks. He commences well. "Having lived some time amidst epileptics we are compelled to know well their attacks, to study the march, to distinguish the symptoms, and especially to examine if there do not exist varieties more easy to cure. Dare we support this opinion, that indeed there exists and that one frequently meets a kind of epilepsy distinct by its phenomena and susceptible of being subjected to a useful method of treatment? Is this assertion going to expire, as so many others in the last pages of a thesis?" Thus writes Bravais in his introduction. Note that he has used the word "march," one that we are apt to attribute to Jackson—the march of the epileptic seizure. He goes on to say that he will treat of the principal varieties of epilepsy limited to one of the halves of the body and that he will call this hemiplegic epilepsy. He defends the name hemiplegic by saying this: "Now, epilepsy strikes the
members with convulsions, as apoplexy strikes it with paralysis." This again sounds prophetic, for Jackson was later to make great use of this comparison. Bravais goes on to accept the classification of epilepsy into idiopathic and sympathetic (reflex) varieties and finally to describe focal epilepsies under five headings, those that commence: (1) in the head, (2) in the arm, (3) those that irradiate from the foot, (4) those that commence in the abdomen or thorax, and lastly (5) those that depend on a local disease of the nerves. He observes that many seizures localized to one limb are sometimes followed by paralyses that may last for variable times—the post-epileptic palsy that we, in this country, attribute to R. B. Todd, of Guy’s Hospital, and to Alexander Robertson. Bravais further states that epilepsy may affect a paralysed limb, and makes another astute observation, that if one limb of an epileptic is paralysed one may be sure that it was in that limb that the attacks originally commenced.

But when one has selected these particular and undoubtedly very important passages one has done with Bravais, for it is clear that he has no conception of any physiological basis for the epileptic seizure even when most localized, and that his mind has not really been engaged on any profound interpretation of what he has observed. His intention, indeed, was to define a variety of epilepsy which he thought to be more curable, using the pharmacology of his day, than the generalized sort, and his method of cure was by the use of vesiculatory applications (especially cantharides) circularly around the distal portion of the limb that led in the attack.

Bravais’ statement that hemiplegic epilepsy is easier to cure than the generalized variety opened up avenues of thought that his time was not ready to traverse. We might to-day accept Bravais’ view, but only after profound modification. We should say that most cases of localized epilepsy carry a decipherable message, and that it is amongst these that we shall look for the cases most amenable to surgery. We should particularly underline the possible origin of such a fit in an often benign cerebral tumour.

Enough has been said to indicate that Bravais was close to important discoveries, but that he did not make them, that certain of his observations are acute, but that they are largely sterilized because his intention was in no way to unravel the pathogenesis of epilepsy, in no way to discover the reason why epilepsy might take the form which he had observed. It is most important that the limitations of his work should be fully understood. And how, after all, should Bravais press on to views which were revolutionary enough when Hughlings Jackson propounded them years later. For had not Flourens, the dominant physiologist of his age, only four years before the publication of Bravais’ thesis, propounded his faith on the activities, or lack of them motorily, of the cerebral hemispheres? His experiments showed, or seemed to show, beyond all question and all doubt, that the hemispheres had nothing to do with movement. One or two extracts must be quoted from Flourens’ famous Mémoires given before the Académie Royale des Sciences of the Institute of Paris in 1822 and 1823. "I removed," he writes, "on a small rabbit the two frontal bones: the animal lost little blood, and he was as well after the operation as before. I opened the dura mater of both sides; I opened the arachnoid also and separated the hemispheres; I then punctured the cerebral hemispheres in their whole extent, without producing anywhere the least sign of muscular contraction." Again: "I removed the cranial wall of the left side of a young dog; I punctured, I tore the cerebral lobes and the cerebellum of that side; the animal was neither disturbed nor agitated. I pricked, on an older dog, the quadrigeminal tubercles: as long as I injured only the superficial layers, that is to say only the tubercles,
I saw no convulsions; when I touched, on the contrary, the cerebral peduncles, on which the tubercles rest, feeble convulsions appeared. I pricked the medulla oblongata: violent convulsions followed."

Flourens had in previous experiments demonstrated that convulsions followed gross stimulations of the spinal cord and medulla. Here is an example: "I uncovered in a young dog the spinal cord in all its extent from the sacrum to the skull. Then I irritated, successively, all the points of this cord thus exposed, beginning at the caudal extremity; and I provoked at all these points phenomena of muscular contractions. I then opened the skull and I continued my irritations on the cerebral mass, and I soon found a point where these phenomena of muscular contraction ceased." A similar experiment showed the like results on a pigeon, on a frog, and on a rabbit: "Everywhere in the whole extent of these regions (spinal) on all these animals punctures and compressions were followed by convulsions." It is in these papers that Flourens gives his classical description of the behaviour of the animal deprived of its cerebral hemispheres, the blind and deaf animal that seemed to dream, but which responded by movement to all stimuli. He records the fact that contralateral weakness of the limbs sometimes follows on removal of a cerebral hemisphere, but that it is variable and always recovered from. Flourens arrived at these conclusions because scarcely any other was possible on the evidence. He failed, of course, because he used the wrong method of stimulation. Electricity was just available, but it had not yet come to the physiological laboratory. His extirpation experiments might have suggested that there were flaws in his argument. Nor did he realize the immense differences in the functions of the brains of the very various animals that he used. But others made the same mistake and thought, apparently, that what was true of the frog and hen was equally applicable to man. Flourens must have been a very expert operator, and we cannot withhold admiration from his work, nor from the terse paragraphs in which he records his results.

In face of these experimental findings it is small wonder that the spinal theory of epilepsy should have arisen and should have held premier place until Jackson came to propound other views. Bravais writing only four or five years after Flourens' lectures could hardly have held a different opinion, and the reader will search his thesis in vain for any theory of epileptic convulsion. Indeed, the treatment of unilateral or hemiplegic epilepsy laid down in his work is a peripheral one, as has been shown. Not for him a conception of its central origin. More recently Brown-Séquard in his lectures at the Royal College of Surgeons between 1838 and 1858 brought forward evidence of the spinal and reflex origin of fits, though this great investigator did not believe that all epilepsy was caused by spinal or peripheral nervous lesions. Jackson himself recognized "Lowest level Fits" (i.e. spinal or medullary fits) but they were the laryngeal crises of tabes dorsalis the respiratory fits of toxic substance, and his conception of these will survive scrutiny to-day.

**Hughlings Jackson on Epilepsy.**

Soon after Hughlings Jackson began his work at Queen Square he became interested, as Bravais and later Charcot, Buzzard, Broadbent and no doubt others had been, in local fits. And above all he was impressed, again as Bravais had been, by the phenomena of post-epileptic paralysis. He observed that the majority of localized epileptic seizures affected the face, hand or arm, and rarely the leg, and that after an attack the limb might be weak or even useless. He was stimulated in his scrutiny of these cases by the publications of Todd, and
could see no difference between the post-epileptic hemiplegic and the post-apoplectic hemiplegic. The reactions and lack of power in these limbs was identical, and the observer, Jackson stated, who did not know the history of the case, who did not know by what stages the patient had arrived in his present paralysed state, would be entirely unable to distinguish the one condition from the other. But in apoplexy the site and nature of the lesion was known. Was it not proper to assign the level of the lesion in local epilepsy roughly to the same region? Of these epilepsies he writes "The fact that the symptoms are local implies, I hold, that there is of necessity, a local lesion. I submit that one-sided spasm, or spasm beginning in one side, implies local change in the nervous system as surely as one-sided palsy does..........I assert, on the contrary, that there must of necessity be some place where the nervous system is diseased, or the spasm determined by causes acting generally would not be local. The fact that palsy after convulsive seizures beginning unilaterally disappears quickly is certainly no proof that there is no permanent local lesion." Of the exact seat of the lesion he was not yet quite certain. "Hemiplegia shows damage (equivalent to destruction) of the motor tract, hemispasm shows damage (equivalent to changes of instability) of the convolutions which discharge through it. Palsy depends on destruction of fibres, and convulsion on instability of grey matter. As the convolutions are rich in grey matter I suppose them to be to blame, in severe convulsions at all events; as the corpus striatum also contains much grey matter I cannot deny that it may be sometimes the part to blame in slighter convulsions........Now both these parts—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 passages appear in his famous "Study of Convulsions" (1870) but without excessively long quotations it is impossible to give a correct impression of his philosophic outlook. Not for nothing was it that he had saturated himself in Herbert Spencer. He uses great language and sets out fine shades of meaning by frequently re-expressing himself in different ways.

We may enquire why Jackson spoke of the local epileptogenic area (in his own words, "the motor province of the middle level") as being particularly within the territory supplied by an artery, the middle cerebral. Probably there were two reasons for this: One because he was using the analogy of apoplexy to illustrate his contention, and secondly, perhaps more importantly, because Brown-Séquard had advanced the view that convulsions were due to spasm of cerebral vessels. This last view has been revived by Spielmeyer, by Foerster, by Penfield, by Leriche and others in recent years.

In 1871 came the publication of the work of Fritsch and Hitzig proving that electrical stimulation of the animal cortex resulted in contralateral movement, observations confirmed and extended by David Ferrier almost immediately. Jackson was conscious that he had not precisely stated that the cortex was the site of local convulsions. Writing in 1875 he says that long before the appearance of the new experimental work that was, of course, to revolutionize conceptions of cerebral function, he had published paper after paper to illustrate "that the facts of convulsive seizures should be used for purposes of localization ......At that time, however, I believed the corpus striatum to be the part discharged in convulsions beginning unilaterally, although then and several years before I believed the convolutions also to contain processes representing movements. What at this time interested me most was, not so much the localization of movements in the cerebral hemispheres, in the sense that, for example, the movements
of the foot are localized here and those of the arm in another place, but the facts of the cases as they bore on a broad principle of localization. "I considered them as part of the evidence that the most special or most voluntary movements have the leading representation."

It was many years before the work of Sherrington and the better directed studies of clinicians were able to give the final proof of doctrines elaborated when Jackson was in his early thirties. He had slowly pieced together by an observation here, a minute study there, a conception which would satisfy the censorship of his own mind. With the wisdom that comes after the event we may wonder why he was so cautious in accepting the testimony of his own eyes, for he followed his cases to the post-mortem room and saw there, as he had expected, cerebral lesions that had caused the fits. But Hughlings Jackson was no jumper to conclusions, there had been enough of that before his day in conceptions and in interpretations of convulsive phenomena. Probably the dead weight of unconverted medical opinion made him slower in his advance, for none are more difficult to convince of a new doctrine than men already satisfied with existing ones. Perhaps it was the very majesty of Jackson's deliberate progression that has built up for him his unassailable position as the neurological philosopher, the Newton of neurology.

The Jacksonian Convulsion.

It was, of course, Charcot—alert, active, hard-working, lucid—who first used the term Jacksonian epilepsy as a tribute to his colleague across the Channel. Jackson did not refer to local seizures under their new title nor did he give an account of what he would regard as a classical example. This omission had good reason, for he would not regard anything as typical, knowing that some small dissimilarities would always be there to make one case a little different from the next, and indeed from any other. In our own time we have loved to simplify, to codify everything, in a word, to standardize disease phenomena, to create syndromes. It is all part of the life around us which influences scientific thought, except that of the highest order. But we are undoubtedly beginning to realise that this simplification comes of the limitations of our own minds (see Sherrington 1934), of a certain restlessness and impatience of the thought that goes on and on, weaving new and ever more complex patterns. Jackson himself thought, as we might say, in the grand manner, and we can imagine that though he might have looked on to-day in benevolent indulgence, he would have found himself unable to accept a standardized description of any physiological action. For though the bare outline of a great conception may very well be written on a post-card, its full expansion may be incapable of complete expression; it will expand and still further expand in the minds of great recipients. So it was with Jackson's views on epilepsy. Definitions are however essential and we may define a local fit by saying that in its perfect form it represents an experiment by disease, that the more closely it resembles what we either imagine, or know, to be the effects of localized stimulation of the motor cortex, the more surely can we postulate some local cause for it. Further, in its purest types the patient retains consciousness throughout. We know that the muscular contractions of the onset, whilst often of the same group, may vary somewhat, just as stimulation of exactly the same point on the motor cortex may not always give precisely the same response even in the same subject. Sherrington has demonstrated the subtle changes in response that repeated stimulation of the motor cortex may elicit, how the effect may be different if the point has been stimulated before and especially if nearby areas have been excited in the meanwhile. He has shown even that the responsive motor cortex grows in extent in a long experiment as stimulation proceeds.
Jackson almost forecasted these findings in his writings. He recognized epilepsy at the three levels which he theoretically created—lower, middle, and highest level fits. The Jacksonian seizure as we know it belonged to the middle level group; those of the highest level were those in which consciousness was early lost and will be mentioned again later. In the local fit he laid stress on the march of the myoclonus, its passage up the arm, say, to the face and down the body to the foot. He had observed that a fit beginning thus might often pass off without loss of consciousness, but that from such a beginning both sides of the body might come to be implicated, and that then consciousness was always lost. It has been mentioned that Bravais spoke of the "march" of the convulsive movements from limb to limb. The word "march" itself is not particularly important to Hughlings Jackson. He uses "spread" just as often; it has a meaning for him, whilst it had none for Bravais. "The mode of onset is the most important matter in the anatomical investigation of any case of epilepsy" a fact as true to-day as it was when made in 1873. It points, says he, to the part of the brain which discharges or where the discharge begins. But he was well aware that in many cases of localized convulsions there was no gross change in the brain, that the local fit could exist as a variety of idiopathic epilepsy. Whether that can be regarded as true of the very localized fits of relatively unchanging pattern is open to doubt. Syphilis seems to have furnished him with most of his cases.

Sensory Epilepsy.

In its common interpretation Jacksonian epilepsy connotes the myoclonic variety only. This is an incomplete statement of his teaching for he was the first to observe epilepsy of the special senses (olfactory or uncinate fits) in addition to subjective or "crude sensations" in the limbs. These crude sensations are the subject of long dissertations by him. He never believed that any part of the cortex was purely motor or purely sensory. Even in a reception so apparently pure as vision he accepted the Spencerian doctrine that the complete perception of objects depended partly on ocular movement. And we realize well to-day that a great part of tactile sensation requires movement for its complete fulfilment (cp. Wilfred Trotter). Hughlings Jackson was not therefore likely to postulate a purely sensory type of epilepsy, nor did he except for the special senses. The problem is not without interest for us to-day. Does an aura commencing very locally mean that the exciting focus is post-central? Or are minute twitchings already occurring in the muscles felt by the patient, but misconstrued?

It will generally be accepted to-day that true sensory epilepsy occurs. Jackson says "It is worth mentioning that some patients have a feeling as if a part were convulsed when it does not really move; one of my patients subject to veritable convulsions beginning in his left thumb had sometimes what he called 'convulsions not to be seen' of that part. Another patient had the feeling of convulsion of one side of his face, but looking in a glass he saw that it did not move." In a footnote to this passage he makes a comment in which it is clear that he foresaw the case which is immediately to be described: "No doubt if a man subject to fits beginning in his right thumb were to lose the right arm by amputation his fits would still seem to him to begin in this thumb—to begin in his spectral thumb, for some time at least." And so it is, as I can illustrate through the medium of a fortunate case which amounts to an experiment, for the patient lost the arm in which focal sensory fits commenced yet she did not cease to have fits nor to have an aura which brought back into her consciousness the "phantom limb" which was at other times absent.
CASE I.—Generalized convulsions commencing with aura in right hand.

Miss A. B. aged 40. At the age of 20 whilst playing the piano a tingling sensation began in the fingers of the hand and slowly spread up the arm, becoming more painful and more unpleasant as it ascended. Shortly afterwards the index finger began to twitch and movement and sensation spread upwards to the shoulder and face, and she became unconscious in a generalized convulsion. From that day onwards she was subject to epileptic attacks, always of the same kind and with the same sensory aura. Although the feeling in the fingers began as a mild one of pins and needles, it very rapidly developed into a dead numbness, with a very painful quality super-added. So much was this the case that she sought various treatments and, after some years, was advised to have the nerves of the arm injected with alcohol. The patient accepted the suggestion, being convinced that if her hand was rendered anaesthetic she would be freed from her attacks. Exactly what happened is not known, but the arm is said to have become black after the injection and was amputated a few days later above the elbow. The patient felt that now indeed she would be free, but to her intense disappointment the attacks shortly returned, and returned in their old form. Ordinarily she was unconscious of her lost hand, but when a fit was developing the phantom limb came to life. Tingling developed in the phantom fingers and in a few seconds the phantom digits began to twitch and the familiar orderly march of the convulsion was inaugurated. The sensations experienced in the hand were entirely and completely unchanged, and had precisely the same qualities as before the amputation.

This case, therefore, supports Jackson's view but it illustrates not only his point about a subjective feeling of twitching but demonstrates also a premonitory aura which was purely sensory. The work of Harvey Cushing, of Van Fleetberg, of Penfield, and of Offried Foerster leaves us in no doubt that stimulation of the post-central area in conscious human subjects is capable of calling up sensory impressions. I can support that, and on one occasion had an unusual experience as a by-product.

CASE II.

The details of the case history are not important. It concerned a man admitted to the National Hospital under the care of Dr. Macdonald Critchley with a long history of right sided fits. A benign type of glioma was diagnosed and a flap turned to expose it. No definite tumour margin could be detected, although a subcortical astrocytoma was readily discovered. The motor area had necessarily to be mapped out before embarking on a resection, and in process of doing this the left sensory cortex was stimulated. Towards its lower end and rather behind it I happened to stimulate the angular gyrus, when the patient at once lost his speech. He had been conversing and recording his impressions, when he suddenly fell silent. After three or four minutes he was able to speak again and said that he suddenly became unable to express himself, although he was not in the least unconscious. It is noteworthy that he had had no fit of that kind before.

Thus was produced experimentally precisely what happens in a local fit originating in the left motor area—aphasia usually accompanied by focal myoclonus on the right side. This aphasia must be an inhibition, and it raises the question whether we are really correct in speaking of explosive discharges of cells as if they were the primary actors. Are we right in thinking that cells gather up a store of energy which they expend in a convulsive seizure? Or may it not be that an inhibition is removed (as, for parallel, the unstopping of an organ pipe) and a violent movement follows? The problem of epilepsy in the speech centre, as everyone concerned with external expression, presents difficulties. Certainly there is never a pouring out of words in the attack, though post-epileptic verbal phenomena may take curious turns.
Traumatic Epilepsy.

A few of Hughlings Jackson's early cases (those that no doubt impressed themselves most deeply on him) were traumatic, with Rolandic lesions. And since injury produces the most simple examples of localised epilepsy, it is but natural that a vast number of such cases have been recorded. The Great War provided a legion of experimental subjects of this class. We know to-day that the great factor in the production of epilepsy after a head injury is injury, not to bone and dura, but to the pia-arachnoid and cortex. We further know, particularly from Penfield's work, that damage to the last two named results in adhesion whilst injury to the dura alone does not. It is doubtful whether the adhesion is itself important; there can be no doubt that the cortical injury is. It is likely that the incidence of epilepsy after such an injury is high, 20 per cent. at least, whilst it is ten times less common in ordinary closed head injuries. We know that injury may lead to fits whose character, and particularly the mode of onset, is determined by the function of the cortex underlying the wound. The fits may thus be motor, sensory, or special sense. They may be truly locally convulsive, the classical Jacksonian fit, "a brutal attack by that man's own movements (p.43)". They may equally become generalized after an introduction proper to the site of the epileptogenic focus. But also they may be generalized, and have no distinguishing quality about them. Thus after a frontal penetrating injury, the fit may commence without warning, the patient dropping in his tracks as if shot, commencing a fit quite indistinguishable from the generalized idiopathic variety. The question arises, is this still a Jacksonian fit or should we beg the question by calling it a post-traumatic fit? Put in another way the question would be, is such a fit the result of an epileptogenic focus, as active as any in more demonstrative regions, but unable to express itself except by means of a generalized fit or by means of abrupt loss of consciousness, a highest level fit? Jackson does not give a precise indication on the actual point, but there can be little doubt that fits even of this kind should still be called Jacksonian. For he says "Fits of epilepsy proper ("genuine epilepsy" of some nosologists) are "highest level fits" and that many of them, not all, are produced by excessive discharges beginning in parts of the prefrontal lobes, highest level (motor province) of the cerebral system. Although the prefrontal lobes are not experimentally excitable, I suppose it will not be denied that their cells katabolise and liberate energy in their normal activities, and it is not unreasonable to suppose that cells of parts of them may, by pathological changes, become highly explosive, so that they occasionally discharge excessively." (p.414). That, I think, answers the questions raised.

Apart from abrupt loss of consciousness it is easy to conceive of other possibilities of prefrontal discharges by the explosive action or release of cells in the highest physiological centres. A difficulty that often arises is to know what time limit can be set beyond which a fit can no longer be said to be of traumatic origin. Various terms have been laid down by different authors, one year, two years, three years. All are empirical; there is no time limit. But we shall be well advised to examine the credentials of fits of very tardy development with great care. The following case is of the utmost importance in illustrating how long the time-lag may be, in a case of undoubted validity.

CASE III.

H. L., aged 22, was admitted to the Manchester Royal Infirmary having commenced seven days before to take fits. On admission the fits were occurring at the rate of one every twenty minutes, were confined to the head and left arm, lasted for two minutes, and at the close consciousness was rapidly regained.
Twelve years before he had been lying asleep on the sands at Southport when someone threw a heavy pointed flint at him. This penetrated the skull close to the middle-line on the right side and slightly in front of the motor area. He was taken to Hospital immediately and in the course of the ensuing ten weeks three operations were performed. The wound healed, a temporary weakness of the left arm cleared up, and he remained quite well for ten years.

During a night in February, 1932, he had his first fit. The fits increased in frequency and severity until they were occurring every twenty minutes, then more and more often until they were separated only by three or four minutes. Scarcely had he recovered, indeed, from one when the next recurred (status epilepticus circumscriptus). The fits began with turning of the eyes completely to the left accompanied by turning of the head in the same direction. The left arm then flexed, and face, arm and hand became involved in myoclonic shocks. Paraldehyde, chloral and bromide had no effect in checking the convulsions. In the intervals between the fits the patient's intelligence was unimpaired and he was able to give a full account of the original injury.

On admission, paralysis of the left face and hand was already fully developed, the leg being unaffected. Tingling and numbness were subjectively experienced in the

**Fig. 1.**

Jacksonian seizures commencing ten years after injury.

Excision of damaged cortex with endotherm needle.
whole left upper limb and two point discrimination was defective, as was positional sense. His skull presented a large defect (3in. × ½in.) immediately behind the coronal suture and near the mid-line. An osteoplastic flap was turned down, cut to enclose the original wound. The skin was fixed to the fibrous layer which filled in an old dural defect, and beneath that was found an area of damaged brain adherent to the overlying tissue (Fig. 1). The dura was reflected back to expose normal convolutions and after securing the vessels at the edge of the damaged area this was excised with the endotherm. A small muscle graft was applied to the superior longitudinal sinus which had been exposed. Flap replaced. Usual closure. The paralysis of the arm rapidly recovered and twenty days later he left the Hospital having had no further convulsion. Nor has he had one since.

If so unquestionable a case as this may develop epilepsy ten years later, we must obviously be extremely cautious in denying cause and effect in other cases where the sequence is more disputable. A point of interest here is the correlation between the pattern of the fit and the site of the injury. This last was so high that one would have expected epilepsy in the leg, but it is no uncommon thing to find that the arm or face will seize the opportunity, as it were, for convulsion much more readily than will the leg. Hughlings Jackson’s observations on this point have already been referred to.

Status epilepticus circumscriptus (a term which best describes the condition) is uncommon after head injuries. Here is another case in which the convulsions stopped abruptly after a very limited cortical excision, although he had had over one thousand fits in rapid succession.

CASE IV.

T. W., age 18, was admitted to Hope Hospital, Manchester, and transferred to Salford Royal Hospital 13/4/34 with frequently repeated epileptic seizures. He had suffered a head injury seven months previously and was unconscious afterwards for five days. On the seventh day he had a fit presenting clonic spasms of the face and both sides of the body. Afterwards there was a flaccid paralysis of the right arm and of the right face. For a few days afterwards he had several fits confined to the right side of the face, and gradually became unconscious after a large number of attacks which occurred frequently and lasted about three minutes. All began in the right side of the face and spread down to the right arm and leg. He was conscious between the fits but was becoming mentally dull.

Nineteen days after admission he was operated upon in the Stockport Infirmary, where he then lay under the care of Mr. G. P. Henderson, but no clot was found either within or without the dura. An area of cerebral softening was noticed in the cortex over the face area. The brain appeared to be under pressure. Following on this intervention he remained well for six months, when the fits once more started quite suddenly. They were of the same character as before, affecting chiefly the head and face. The leading movement was a turning of the head and eyes to the right, and he said it appeared as if something caught his eye and compelled his eyes to turn to that side. What the object was, or whether light or coloured, he could not particularize. The conjugate deviation was followed by twitching of the face, but there was no movement of the arms or legs. Next day the arm began to twitch, too, and the fits continued with great frequency until he had developed a monoplegia of his right arm and an expressional facial palsy on that side. He was able to speak but it was difficult to understand him. So far as could be judged he was well orientated and knew what he wanted to say, in some degree at least. For the first week the fits were confined to the right side and then became more or less generalized.

When I saw him he had had 168 fits in 24 hours, and I transferred him to the Salford Royal Hospital for further observation. He had 300 fits in the next 48 hours, and altogether must have had between one and two thousand since the onset of his second attack of convulsions. An osteoplastic flap was turned above the site of the previous operation, the dura was found to be adherent at four points to the cortex.
Head injury seven months previously. Upwards of 1000 fits in a few days. Reflection of dura demonstrating points of cortical damage.

(Fig. 2), the areas of adhesion were small, and that in the face area was excised with the endotherm needle. Following on this his fits ceased abruptly and he remained well for a year, when he again had a rather similar series that lasted for three days, but stopped spontaneously. It may be that further cortical excision is going to be necessary.

Focal fits of this kind are exceedingly uncommon in the ordinary closed cranio-cerebral injury for local cortical damage in the motor area is rather the exception in this type of case, the basal and polar regions of the brain being those most commonly lacerated.

**Jacksonian Fits and Intra-cranial Tumour.**

Several of Hughlings Jackson's best cases of localised convulsions were due to intra-cranial tumour, but the pathological and histological descriptions leave a good deal to be desired. In his 1870 paper "A study of convulsions" (p. 8) there is a section on epilepsy beginning in the foot, and at that time he had had no
case to enlighten him as to the cause of this most uncommon type of local convulsion. But in 1882 he was able to record a case of epilepsy in the right foot, the attacks covering a period of twelve years, with post-mortem examination revealing a calcifying glioma in the left motor area. We should say to-day that epilepsy of the leg area pointed to a cerebral tumour more certainly than any other of the focal epilepsies (Fig. 3). But we should know, also, that a tumour is not invariably present. Jackson said that autopsies on cases with very local epileptiform seizures might show nothing post-mortem or else such uniformly generalized cortical atrophy that no special distinction could be made of excess at any single point. There is one tumour which can, classically, produce the most perfectly localised fits commencing in the foot—the para-sagittal meningioma indenting the leg area of the cortex. But para-sagittal gliomas can do the same thing, as Jackson found. (In his day the dural endothelioma or meningioma was not recognised as such, and many examples of gumma, "tubercles," and fibrous tumours were probably of this nature). The very fascinating problem of intra-cranial tumour and convulsive states would take us too far afield and too many fascinating personal experiences come to mind.
Suffice it to add that tumour or abscess furnishes another example of Jacksonian epilepsy, the special sense discharge of olfaction, the uncinate fit.

**Hughlings Jackson and Cerebral Localisation.**

It must be remembered that the modern doctrine of cerebral localisation came to full fruition only during the last years of his long life. The Jacksonian fit has been for half a century the most perfect, as well as the most dramatic, clinical evidence of localised function. (Hemianopia, maybe, is an older and as good a testimony). But there is very little in Jackson's writings to support the view that he was an exponent of the patterned cortex. He was, no doubt, interested in the findings of Brodmann, Flechsig, C. and O. Vogt, Campbell, and Elliot Smith but his view of nervous mechanisms would not be confined by any narrow boundaries, by the limits of, say, areas 8, or 35, or 50. Long after precise limits had been laid down to the excitable areas for the face, hand, leg, we find him insisting that function did not reside only in one minute spot; the whole nervous system was a sensori-motor mechanism. A convulsion was for him "a contention in which many movements are 'run up' into spasms. There is a sudden and excessive discharge of many nervous arrangements representing movements, at once or nearly together, because the cells of those arrangements have by some pathological process become highly unstable." It will be observed that although he speaks sometimes of outbursts of normal movements he thinks actually they are disordered and contending. We have had to wait for proof of that observation fifty years; the fascinating researches of Denny Brown and Graeme Robertson have put it beyond doubt. It is worth noting, furthermore, that weakness on the same side of the body as the lesion in hemiplegics attracted Jackson's attention, as well as the anatomy of the direct pyramidal tracts and degenerations in both crossed tracts after a unilateral lesion. Thus do we get a faint foreshadowing of the work that Fulton and his co-workers were to do so ably at Yale.

Hughlings Jackson's conception of cortical representation was essentially a philosopher's rather than an anatomist's. He saw and analysed the workings of the system as a whole, and would not be bothered with too precise an anatomical statement. How far, then, was he a physiologist? Certainly he was that, but not one of the laboratory type. He used the culture of the years before him, and it happened that that which he could use best by temperament was the philosophy of Herbert Spencer, whose "Psychology" impressed him deeply. Contemporaneously with Hughlings Jackson sprang up great researches which illuminated his ideas, and he was content to use the results of animal research offered him. But one feels that he did not need them, and that he was capable by his own more than astute clinical judgment, and by his powers of reasoning, of founding unaided the neurological concept which is his legacy to posterity.

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The quotations from the writings of Hughlings Jackson are taken from Vol. 1 of "Selected Writings of John Hughlings Jackson," admirably edited by Dr. James Taylor (Oxford Medical Publications, 1931).

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**Correction.**

We regret that in the bibliography of Mr. Williamson-Noble’s article on "Hughlings Jackson and the Ophthalmoscope," which appeared on p. 162 of our April issue, the publication of Hughlings Jackson’s "Selected Writings" was inadvertently attributed to the Oxford Medical Press instead of to the actual publishers, Messrs. Hodder & Stoughton, Ltd.

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**General Post-Graduate News.**

It should be noted that Courses arranged by the Fellowship of Medicine are open only to Members and Associates unless otherwise stated. A copy of each detailed syllabus is sent to every Member and Associate.

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**Chest Diseases:** May 27 to June 1. Victoria Park Hospital. All day. Fee £3. 3s.

**M.R.C.P. (Evening Course):** June 11 to 27. National Temperance Hospital. Tuesday and Thursday evenings at 8.0 p.m. Clinical and Pathological. Fee £6. 6s. *(Maximum of 24).*

**Urology:** June 17 to 29. St. Peter’s Hospital. All day. Fee £5. 5s. *(Maximum of 8.)*

**Cardiology:** June 24 to July 6. National Hospital for Diseases of the Heart. All day. Fee £7. 7s. *(Maximum of 20.)*

**OTHER COURSES.**

**Cardiology (Week-end Course):** May 4 and 5. Victoria Park Hospital. All day. Saturday and Sunday. Fee £1. 11s. 6d.

**Chest Diseases (Week-end Course):** May 11 and 12. Brompton Hospital. All day. Saturday and Sunday. Fee £1. 11s. 6d.

**Obstetrics (Week-end Course):** May 25 and 26. City of London Maternity Hospital. All day. Saturday and Sunday. Fee £2. 2s.

**Venereal Disease:** May 27 to June 22. London Lock Hospital. Afternoons. Fee £2. 2s.

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