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Some Observations on Epilepsy.

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I FEEL that I cannot adequately express my appreciation of the honour of being elected to a chair which in the past has been occupied not only by many distinguished men of science, but also by some whose names are indelibly imprinted upon the history of medicine. That a surgeon should be chosen to fill this position is something more than a personal honour; it is a compliment to that branch of practice in which he is engaged, implying as it does a recognition of the fact that surgery has come to occupy a not unimportant place in neurology.

In those affections of the nervous system to which surgical measures are applicable, the physician is chiefly concerned with diagnosis, whilst the surgeon must pay particular attention to the technical problems which operative treatment presents. Neither is fitted by training or experience to perform in these respects the functions of the other, and I do not doubt that if a combined physician-surgeon could be evolved, he would evince the sterility common to hybrids.

I take the opportunity of touching upon this subject in view of the well-known opinion held by Harvey Cushing that the surgeon who concerns himself with operating upon the nervous system should be his own neurologist. However attractive this may be in theory, it can never, to my mind, attain practical realization, for just as a wide knowledge of general medicine is essential to the neurologist, so is an intimate acquaintance with general surgery desirable for one who

takes seriously that part of general surgery which concerns itself with the nervous system. The acquisition of this familiarity with all branches of surgery leaves but scant time for the laborious and exacting work, both at the bedside and in the laboratory, which the training of a neurologist demands. I hold that the nervous system is not a suitable field for surgical specialism in the more restricted sense of the term.

The co-operation of neurologist and surgeon is not only of value in the diagnosis and treatment of certain diseases of the nervous system, but it also affords opportunities for the study of what Moynihan has called the Pathology of the Living. Neurology has already gained much from observations made in the operating theatre, and far more may be confidently expected in the future. The mere exposure of the cortex, the removal of tumours, section of nerve roots and other manipulations reveal phenomena which can be observed neither at the bedside nor by post-mortem examination. In more senses than one the operating theatre may be made a link between the ward and the post-mortem room. When a physician fails to witness the operations which are performed upon his patients, he not only neglects a fruitful source of information for his own future guidance, but he also deprives the surgeon of the very great advantages which accrue from consultation upon questions which may arise during the course of an operation.

In his Presidential Address to the Neurological Society in 1902, Herbert Page said: "I venture to take up the position that surgery may be even bolder than it has hitherto been in endeavouring to minimize and remove some of the causes which seem to be at the root of the later consequences of severe head injury." Among these later consequences the occurrence of epileptiform seizures stands out prominently, more so than ever before by reason of the large number of sufferers from epilepsy resulting from cranio-cerebral wounds.

Having had to deal with many such patients during the past three or four years, I have been increasingly impressed by the feeling that surgical procedures for the treatment of epilepsy are greatly hampered by the obscurity which still exists as to the essential factors that underlie convulsive attacks in general.

James Taylor has recently reminded us that in regarding epilepsy as a disease, there is some danger of losing sight of its purely symptomatic aspect, and has emphasized the desirability of searching in every case for the physical basis which forms the starting point of the convulsive seizures. He contends that the attempt to enmesh epilepsy in the net of psycho-analysis represents a definitely retrograde move-

ment, whilst any real step forward in the elucidation of the nature of "ordinary epilepsy" is to be made from a study of Jacksonian attacks.

Gowers said that a tumour or spicule of bone could "so train the whole brain into a habit of discharge that the attacks differ little from those of idiopathic epilepsy." Instead, however, of merely seeking to reconcile the convulsive seizures of known focal origin with those of idiopathic epilepsy, we should rather approach the problem from the opposite point of view, realizing that in idiopathic epilepsy we have phenomena unassociated with any obvious cause, which may yet be in all respects identical with those which occur in connexion with gross lesions, from the damp squib effort of *petit mal* to the full pyrotechnic display of a major attack. Further, we must remember that between ordinary epileptic fits and focal fits associated with visible lesions there exists a bridge in the form of attacks of a Jacksonian character in which no gross lesion appears to exist.

Surgery would be in a better position to play its part in the treatment of epilepsy, whether or not of focal origin, if we knew more of the factors underlying the phenomena of a fit; and if we could recognize any essential precipitating cause common to all forms.

I would not presume to criticize the various views as to the pathogenesis of epilepsy which have been advanced in the past, or which are at present entertained. I should like, however, very briefly to examine what is known as the vascular hypothesis, because during the frequent opportunities which I have had of viewing the living brain, under both normal and abnormal conditions, I have been greatly impressed by the striking rapidity with which visible circulatory changes may occur in response to various disturbing causes. I cannot help feeling that sufficient importance is not attached to the possible effects upon cerebral function of what may perhaps be called minor and transitory circulatory disturbances. It is not upon the congestion or anæmia of the brain as a whole that I would lay stress, but upon the local variations and abnormalities which can be observed in connection with tumours and various traumatic lesions. It seems to me that the only local influence which an operation can have upon an epileptogenous area, short of excising that portion of the cortex, must be exercised through the medium of the local circulation. If there is a fundamental precipitating cause for all convulsive attacks, any hypothesis which does not take into account the pathogenesis of fits originating in the neighbourhood of a gross lesion must fail to touch that essential point.

Gowers, discussing the pathology of idiopathic epilepsy in 1900,

wrote: "The conceptions of epilepsy which have been current during the last forty or fifty years . . . are not much more instructive than the old demoniacal pathology which gave the disease its name." One such conception he specified, namely, that "the epileptic fit was the result of a spasm of the arteries of the brain," and went on to remark that this notion had disappeared absolutely before the simple but conclusive facts which connected the manifestations of the disease with a morbid state of the cortex. It is noteworthy, however, that, some years later, in the "Borderland of Epilepsy," he admitted the possibility of cerebral vaso-constriction in relation to vaso-vagal attacks. But the view that the phenomena of epilepsy result from disordered cortical function does not by any means necessitate relinquishing all belief in an underlying vascular disturbance.

On the contrary, both the "morbid state of the cortex" postulated by Gowers, and the "spasm of the arteries of the brain" denied by him, may conceivably be concerned together.

The whole subject of the relation of disorders of the cerebral circulation to convulsive attacks, as well as to loss of consciousness and other clinical manifestations, was fully discussed by A. E. Russell in his Goulstonian Lectures in 1909. To these lectures I have long been indebted for suggestions and references bearing upon the very interesting and important question of the relationship between gross cerebral lesions and epilepsy. For it is obvious that in operating upon epileptics, and in choosing cases for operation, we should like to have a clear idea of the purpose of the operation, based upon some reasonable conception of that relationship. The surgeon who merely removes a disc of bone from the cranium of an epileptic patient places himself on a level with the practitioner of the Stone Age, the marks of whose handiwork upon the skulls, probably of the epileptic or the insane, are to be seen in museums of ethnology.

The thesis which Russell developed from a mass of clinical evidence was that the common factor underlying many disturbances of cerebral function is to be found in some disorder of the cerebral circulation. It is interesting to note, in view of the trend of recent research, that as long as twelve years ago he hinted that, so far as the causation of epilepsy at least is concerned, inquiry into the processes, both nervous and chemical, by which the cardiovascular apparatus is governed, might afford a valuable clue. He further drew attention to a similarity between experimental decerebrate rigidity and the tonic spasms occasionally observed in cases of sudden complete arrest of the cerebral circulation.

In most writings upon epilepsy such expressions as "sudden discharge of nerve force," "sudden liberation of energy," and "explosion of nervous energy" are commonly found. Such terms, if they relate to the cortex alone, can only mean that the fit is to be looked upon as a manifestation of excessive cortical activity. It is impossible, however, to regard loss of consciousness, one of the most sudden and striking events, and often the only recognizable event in an epileptic fit, as a manifestation of the discharge of nervous energy.

A sudden failure of the cerebral circulation is inevitably followed by loss of consciousness, and we know that it can also produce convulsive movements, as seen in the attacks associated with the condition known as "heart block." It is inconceivable that a sudden arrest of the cortical circulation can, at one and the same time, bring about both a suppression and an augmentation of function. When loss of consciousness and convulsive movements result from the same cause, we can only believe that those movements point to a suspension and not a stimulation of cortical activity. The tonic stage of muscular contraction appears to indicate, not a discharge of nervous energy from the cortex, but the liberation of a lower level motor mechanism from cortical control. In a word, the unconscious rigid epileptic is, for the moment, a decerebrate man.

In a recent paper Kinnier Wilson has brought forward clinical evidence to show that decerebrate rigidity, both with and without the occurrence of tonic fits, occurs in man as the result of lesions which interrupt the cortico-spinal path, and that such rigidity is strictly comparable with that which obtains in animals when the mesencephalon has been transected. He points out that lesions which have not the completeness of the experimental section find expression in rigidities of limited scope, and that even functional interruption of cortical control may be manifested by a similar sequence of events. It requires no great effort of imagination to suppose that cortical control may be suddenly and momentarily cut off by a sudden and transient disorder of the cortical circulation, and cause, according to the extent and duration of the circulatory disturbance, varying degrees of decerebrate rigidity.

Whilst a state of momentary decerebration may be held to explain the tonic stage of an epileptic fit, it is not so easy to account for the clonic movements. Kinnier Wilson, in the paper alluded to, having characterized the tonic fits observed in the cases which he was describing as "attacks of decerebration," remarks that the absence of

any clonic movement is of much significance. He takes it to indicate that tonic and clonic movements have different origins, and are the expression of the activity of different motor mechanism, the one being characterized by static, and the other by phasic activity.

In a few recorded cases an epileptic fit has occurred during actual examination of the heart or radial pulse. The tonic stage has coincided with the sudden arrest of the circulation, whilst the clonic stage has commenced with the return of the circulation. In cases such as Russell's where sudden and permanent arrest of the circulation was immediately followed by tonic spasm, no clonic movements were observed.

It can hardly be doubted that the clonic movements which follow the tonic stage of a general epileptic fit are of the same character and depend upon the same motor mechanism as those of a Jacksonian fit which is accompanied neither by a tonic stage nor by an initial loss of consciousness. They exhibit the phasic character of movements associated with cortical activity, and resemble both those which can be evoked by faradic stimulation of the cortex, and those of a Jacksonian fit dependent upon a cortical lesion.

Many years ago, Long Fox, who attributed the tonic spasm of an epileptic fit to cerebral vaso-constriction, ascribed the clonic movements to a "gradual yielding of the vasomotor constriction, allowing at first more blood to enter the arteries than during the period of tonic spasm, but yet far less than is necessary for controlled movement or for rest."

That the cerebral circulation is under direct vasomotor control was long denied, chiefly owing to the work of Leonard Hill, who, believing the variations to be entirely passive, summed up his views in the paradox that "the cerebral vasomotor nerves lie in the splanchnic area." Although the cerebral circulation differs from the circulation elsewhere in many ways, and particularly in its passive dependence upon external circulatory changes, it is now generally admitted to be also subject to active vasomotor control, although the final demonstration of this by direct physiological experiment is still lacking.

Harvey Cushing noted that epinephrin will blanch the pial vessels over the area of its application, and that faradic stimulation will do the same. Brodie and Ferrier found that on injecting adrenalin into the basilar artery of the removed brain the outflow from the torn sinuses was diminished or completely stopped according to the quantity injected.

During the course of an experiment which Professor Sherrington kindly gave me the opportunity of witnessing, I was struck by the fact that when a faradic stimulus of sufficient strength to evoke epileptiform movements was applied to the cortex, the pia-mater could be seen to blanch, and it naturally occurred to me to wonder whether epileptiform attacks starting from a traumatic lesion may not be accompanied or even initiated by a similar blanching of the cortex.

The evidence in favour of vasomotor activity in the brain is abundant, and it seems incredible that pathological variations in the cerebral circulation should not be associated with definite clinical manifestations. The characteristic rapidity and suddenness of vascular changes, and their characteristic tendency to recurrence, would allow of recurrent phases of disordered cerebral function, and on this hypothesis many of the phenomena of epilepsy can be fully explained.

The cerebral cortex has been raked over and over again by successive generations of neuro-histologists without yielding up the secret of the cause of idiopathic epilepsy, and it may be that the search, transferred from this barren field to that of the autonomic nervous system, will prove more fruitful.

Before discussing the part played by gross lesions in focal epilepsy, it is necessary to refer briefly to the effects of electrical stimulation of the cortex.

In man the effects of such stimulation appear to resemble closely those obtained from anthropoids, but as the recorded observations are comparatively few, and not primarily experimental, this need not be referred to further, except for one important point. Fedor Krause, in 1911, had the opportunity of stimulating the cortex of a patient who was the subject of Jacksonian epilepsy without gross visible lesion. He found that faradization of the epileptic zone elicited fits, whereas from the rest of the motor cortex only the corresponding movements were obtained, identical currents being used, and the duration of the stimuli being the same in each case.

In this connection an interesting and probably unique experience recorded by Sherrington is of considerable importance. Having by chance found amongst his laboratory animals a monkey which was the subject of epileptiform attacks, he took the opportunity of investigating the effect of cortical stimulation. The spontaneous attacks had begun in the left angle of the mouth (or perhaps in the tongue), and had spread to the rest of the face, neck, left arm, left leg, and then to the right limbs. Attacks could be induced by the taking of a large morsel

of food into the mouth. A minimal faradic stimulus applied to the tongue area of the right hemisphere provoked a tongue movement which almost immediately became clonic and epileptoid, meaning by the latter that it continued as a series of movements after the faradic stimulus of the cortex had been withdrawn. If the stimulus were continued for a few seconds the epileptoid movement spread from the tongue, and would occasionally involve all the facial muscles on the left side, but it never spread beyond the face. In the left hemisphere the tongue area yielded epileptic discharges easily, though not so readily as did the tongue area of the right cortex, nor did the movements spread. Sherrington remarks that it must be remembered that limited epileptiform discharge is, in the monkey, usually obtained by prolonged or quickly repeated faradic stimulations of almost any point in the motor cortex, and that therefore the result observed with the tongue area of this animal was of a quantitative rather than a qualitative nature. Further, that the exceptional readiness with which tongue epilepsy could be evoked was remarkable because, in his experience, the tongue area is not one from which epilepsy is usually easily elicited. The results of cortical stimulation in this epileptic monkey fall into line with those obtained by Krause in his epileptic human subject.

We require, however, an explanation of the fact that at least in the highest apes (and probably in man, to judge from Krause's observations) electrical stimulation is incapable of provoking a Jacksonian fit of any magnitude. Even in Sherrington's epileptic monkey: "By no persistence of the faradization could the epileptoid movement be made to extend beyond the face. It never, unlike the natural seizures observed in the animal, spread to the neck or limbs even of the same side, let alone the opposite." We know that in experimental work the depth of the narcosis affects the results in a marked degree, and it is possible that it is an important factor in determining the differences noted. It may be, too, that higher control, whether consciously exerted or not, accounts for differences in the extent to which the cortex responds to the stimulus of a focal lesion. It is a matter of common observation that patients can often arrest the spread of clonic spasms by holding firmly the affected limb at the commencement of a fit, by which manœuvre controlling influences are doubtless directed to the cortex involved, so as to neutralize the effect of the abnormal stimulation.

Horsley and Schäfer many years ago demonstrated the fact that the excitability of the cortex is increased by repeated stimulation, so that a weaker current will evoke a spread. This was confirmed and ampli-

fied by Leyton and Sherrington, who, in the course of their work upon the cerebral cortex, elicited many additional facts bearing upon focal epilepsy. Great variations were observed among individual animals in the ease with which a Jacksonian "march" could be provoked by the faradization of a cortical motor point. Not only is the threshold of excitability lowered by faradization, but the area from which a given movement can be obtained may be gradually extended. Thus after provoking movements of the lips, for example, by stimulating a cortical point just in front of the central sulcus, the same movement may be elicited by faradization of a previously inexcitable point more anteriorly situated. Further, faradization of the post-central cortex may facilitate the elicitation of movement from certain points in the precentral cortex at about the same horizontal level.

In this connection the following experience is of interest. Working with Dr. Gordon Holmes I had the opportunity of observing the effect of faradic stimulation of the cortex in a patient who for several years had been the subject of frequent focal fits resulting from a traumatic lesion. This lesion involved an area of the cortex, some 4 cm. in diameter, both behind and in front of the Rolandic sulcus, at about the level of the temporal crest. The stimulation was carried out in order if possible to identify the point of origin of the fits, and the technique employed was the same as that used by Sherrington. For obvious reasons the observations could not be made with the completeness of a physiological experiment, but there was no doubt as to the readiness with which epileptoid movements could be evoked from many points, some of which were far distant from the cortical area of normal representation.

Leyton and Sherrington have named the movements obtained by electrical excitation of the cortex "fractional" movements, and remark that they are in themselves co-ordinate and seem to form parts of complex purposive acts.

Electrical stimulation of the cortex, even when carried out with every possible refinement of technique, must be the grossest caricature of the natural stimuli which provoke purposive movements. Yet such electrical stimuli applied to the precentral cortex in man can be made to evoke definite movements, limited in extent and bearing a striking resemblance to simple natural movements. Although, as Ferrier first pointed out, these movements exhibit purposive co-ordination, they are not purposive movements, and in this respect resemble those of mild epileptiform attacks.

A Jacksonian fit is merely a series of fractional movements, devoid of purpose, and determined in point of sequence only by the relative anatomical position of the cortical areas in which they are represented. In other words, a Jacksonian fit is made up of a series of fractional acts, which, although in themselves co-ordinate, lack the mutual co-ordination of natural purposive acts. This mutual incoordination of fractional acts is illustrated by the biting of the tongue, due to the unnatural sequence of tongue protrusion and jaw closure. In this respect the movements of epilepsy resemble the effects provoked by electrical stimulation, which even when so applied as to evoke a "march" or sequence of movements, does not appear ever to call into action the peculiar synthetizing function of the cortex defined by Sherrington. No series of fractional movements so combined as to bring about a recognizable purposive act, even of a simple character, has, so far as I am able to ascertain, been recorded as resulting from electrical stimulation of any part of the cortex. In fact no more intelligent movements can be obtained from the cortex than from the mid-brain; they differ in character rather than in purpose, the one set taking the rhythmical form characteristic of movements obtained from the cortex, and the other lacking that particular nature. It seems to be not impossible therefore that the disturbance of cortical function, whether caused by experimental stimulation or by a gross pathological lesion, may, in part at least, be due to a lowering of the normal inhibition, and that the movements which result may be of the nature of release phenomena. On such a conception the epilepsy resulting from pathological lesions can be brought into line with that which can be induced experimentally.

The association between gunshot injuries of the brain and the development of fits is so obviously a case of cause and effect that I will refer to them before touching on the subject of fits associated with morbid lesions, and on those of idiopathic epilepsy.

The local morbid conditions underlying the fits which result from gunshot wounds of the head fall into three main groups.—

(1) Recent lesions such as local contusion associated with small hæmorrhages and œdema; or the more gross disruptive effects of a penetrating wound.

(2) Inflammatory lesions due to recrudescent sepsis, where a latent infection becomes active and gives rise to an area of softening, or to an actual abscess.

(3) Cicatrices binding the scalp to the brain and membranes through a cranial defect.

As regards the first two groups, the early fits, namely those which occur in the first few days after the injury, as well as those connected with recrudescent sepsis, are clearly associated with vascular disturbances due to direct damage to blood-vessels, thrombosis, inflammation and œdema; they continue only during the period of acute circulatory disorder, and cease when that period comes to an end.

The third group, with cicatrices binding the scalp to the damaged brain, is by far the most important, and it is to the cases included in this category that I wish to draw particular attention.

A year ago the Re-survey Boards of the Ministry of Pensions had, in the previous twelve months, examined more than 25,000 old cases of gunshot wound of the head. Excluding those examined more than once during that period, the number of individuals was approximately 18,000, amongst whom nearly 800 (or 4½ per cent.) were the subjects of epileptic fits. A very large number of such patients present a cranial defect through which the scalp adheres to the membranes and the damaged brain. Penetration of the dura at the time of the wound entailed not only gross direct damage to the underlying brain and its vessels, but also invasion of the injured tissues by micro-organisms. The consequent inflammation resulted in further destruction of nerve tissue as well as a greater disturbance of the vascular supply, both from thrombosis at the time and from strangulation of vessels later by fibrous tissue, the amount of which would to a large extent be proportional to the intensity and duration of the preceding inflammation. Further, the penetrating wound is inevitably followed by adhesions between the surface of the brain and the scalp.

It may reasonably be assumed that the rich blood supply of the normal cortex provides a wide margin of safety against accidental variations, whilst the relatively avascular scarred cortex possesses a smaller margin of nutritional safety. In these circumstances local circulatory disturbances would be likely to arise from accidental causes. One such cause which I believe to be of the very greatest importance, depends upon the fixation of the brain at the point of damage to the overlying membranes and scalp, and is occasioned by the cerebral movements.

There is no doubt that the normal brain obeys the law of gravity, and alters its position with varying positions of the head. Every organ of the body possesses a degree of mobility proportional to the extent to which it is covered by a serous membrane separating it from the wall of the cavity which contains it. The serous membranes of the thoracic

and abdominal cavities, as well as those which line the joints and the tendon sheaths, result from and facilitate movement. The brain is no exception; the serous cavity lying between the dura and the arachnoid can fulfil no other purpose; the intense pain occasioned by any movement of the head in meningitis is analogous to that of pleurisy. Whenever the skull and dura are widely opened during an operation, movements of the brain can be demonstrated by altering the position of the head, and although their extent is probably exaggerated by reason of the different physical conditions which obtain in the open as compared with the closed skull, there is no reason to suppose that all movement is absent when the skull is whole. Indeed J. Luys has demonstrated the fact of cerebral mobility by means of frozen sections, and Gavoy has, by means of a specially designed cerebral kinesiometer, been able to measure excursions of from three to nine millimetres.

When a brain, attached to the scalp by adhesions, attempts to move in response to a change of posture, it is prevented from doing so from a pull at the point of anchorage; this pull either mechanically or, possibly, by causing a reflex vasoconstriction, may well produce a momentary local anæmia in the damaged brain, and so initiate a fit.

Seeing, however, that only a very small number of patients with anchored brain develop epilepsy, it is obvious that the local lesion is but one of the links in the ætiological chain. Clearly a given stimulus is not effective for all brains, and it is necessary to assume in those patients who do develop fits a tendency to epilepsy resulting from what is vaguely called a relatively low degree of stability of the nervous tissue.

I have been fortunate enough to encounter a case which illustrates this important point remarkably well. A man, 35 years old, had at the age of 15 been accidentally wounded in the forehead by a revolver bullet. He was operated upon shortly after the accident, and was in hospital about fifteen weeks. He afterwards remained perfectly well in every way, joined the Motor Transport Service in 1915, and was sent to France where he served until 1919. He received no wound or other injury, but was often under shell fire and bombing. His first fit occurred after six months' service, and between that time and the date of his admission to hospital in 1920 he had about two dozen fits, all of which occurred during sleep, except two. The fits associated with frontal injuries of this character not infrequently occur during sleep, and they are, if the explanation which I have suggested is correct, to be attributed to the sudden changes of posture which may occur during sleep. This patient, in the two attacks which occurred in the daytime, fell unconscious,

without warning, and remained so for some minutes. He did not bite the tongue nor urinate; he suffered neither from headaches nor giddiness. There was a small irregular circular opening in the right frontal bone, into which the brain bulged on stooping forward. No abnormal neurological signs were detected.

The interpretation of this case seems to be that, although for twenty years this man had had a cranial defect through which the brain was anchored to the scalp, the local lesion had been insufficient to cause a fit until the nervous stability had been lowered by the stress of warfare. In May, 1920, an operation was performed. The scalp was dissected free from the dura, and the dura was separated from the bone for an inch or more beyond the edge of the opening. A thin sheet of celluloid was placed over the dura, extending some distance between it and the inner surface of the skull. A thicker sheet of celluloid was used to close the bony opening. Some ten weeks later he had a fit, whilst asleep, and since then no further fit has occurred.

In cases of this class the aim of an operation is twofold, first to ensure a permanent separation of the brain from the surface at the point of damage, so as to restore as far as possible its mobility; and secondly to close the bony opening so as to restore the physical conditions under which the cerebral circulation normally works. Both of these objects can be attained by the use of celluloid, the technique of which procedure will I hope form the subject of a future communication.

I and my assistants at the Tooting Pensions Hospital have now operated upon more than 200 cases by this method. About 120 of these patients suffered from fits. The time has not yet arrived for passing a judgment upon the results, but I may say that so far they are remarkably promising, even in some cases where the fits had occurred over a long period. Nor is it possible to estimate fairly the extent to which the beneficial results are due to the other treatment carried out at the same time, for in all cases the patients have been kept in hospital for from two to six months after the operation, and treated regularly with small doses of bromide.

It seems probable, to judge from our experience, that better results may be expected in cases where the fits have been infrequent, and have not occurred over a long period of time. Nevertheless, satisfactory results are not impossible even in long-standing cases, as shown by the following example:—

A man of 21 had, when 7 years of age, received a severe cranio-

cerebral injury in the left parietal region, which resulted in gross hemiparesis. A large area of bone had been removed, and the damaged brain had become adherent to the scalp. The brain pulled upon the scalp, which became concave when the patient lay upon his right side; the brain bulged when the head was inclined to the left.

Eleven years after the accident he began to suffer from fits, the origin of which was attributed to a blow upon the head in the region of the cranial defect. When admitted to St. Thomas's Hospital he had had fits for three years, for the first year about once a fortnight, for the second year about three times a week, and for the third year about once a month. On one occasion he had twenty-five fits in a single day. He was operated upon by the celluloid method and carefully treated for several months afterwards. Two years have now elapsed since the operation, during which time he has had only two fits. He has taken no drugs for a year, has improved greatly in general health, and is able to do some light work.

The claim of surgery to participate in the treatment of fits resulting from injuries to the skull and brain may fairly be regarded as well established, not only on the ground that a reasonable explanation exists of the mechanism by which they are evoked, but also because we now possess a good deal of evidence to show that the fits can in many cases be either abolished, or very considerably reduced in number and severity, by an operation which succeeds in removing or at least modifying, the local exciting cause. This evidence, now in course of preparation, I purpose bringing forward in the near future.

Leaving now the subject of epileptiform attacks due to gross injuries, we must approach the question as to what contribution surgery has to offer towards the treatment of fits other than those of traumatic origin, whether associated or not with local lesions of the brain. For the purpose of examining this question, I would divide these cases into three groups:—

The *first group* comprises all the cases in which there exists a lesion both lending itself to accurate localization and allowing of a reasonable surmise as to its nature. Many tumours, both innocent and malignant, as well as abscesses, cysts, tuberculomata and gummata, can be included in this category. Whilst any of these lesions may constitute the starting point of an epileptic attack, it must not be forgotton that there is no gross lesion, whether traumatic or otherwise, which necessarily causes fits. On the contrary, the proportion of patients with any such lesion who develop fits is small. As already mentioned, the incidence of

epilepsy amongst pensioners with old gunshot wounds of the brain does not at the present time reach 5 per cent.

The frequency, however, with which fits are associated with tumour of the brain is perhaps somewhat striking. I have recently looked through the notes of 270 cases of brain tumour, exclusive of cerebellar and pituitary cases, upon which I have operated. I find that no fewer than eighty-two patients, or 30 per cent., exhibited fits of a focal character. Grouping regionally the tumours associated with fits, I find that exactly half were situated precentrally, and of the remaining 41 22 were post-central, 12 temporal and 7 occipital. Of the whole 82 tumours, 65 were malignant and 17 were endotheliomata.

In no fewer than 40 per cent. the fit was the first symptom of which the patient complained. The fit was the first symptom in a rather larger proportion of the endotheliomata than of the gliomata, but the difference is not, in my opinion, sufficiently striking to be of any great value in diagnosis.

There can be no doubt, I think, that the nearer the tumour, or the surrounding area of cerebral softening, approaches the central sulcus, the more likely are fits to occur.

The exact nature of the stimulus by which a tumour or other gross lesion elicits evidence of cortical disturbance, whether of a motor or of a sensory character, is not easy to define. To say that a lesion "irritates" the brain merely creates fresh difficulties by raising the question of the exact meaning of irritation. Used in its usual sense irritation implies activity, but a lesion such as a bony boss resulting from a depressed fracture cannot be regarded as an active agent. If the brain from time to time accidentally, or continually on account of its respiratory and cardiac movements, impinges upon such a bony boss, changes conducive to epilepsy might occur. A tumour might be in a sense "active" from alterations in size or tension due to variations in its blood content, or to such sudden gross change as that caused by a hæmorrhage into it. Active inflammatory changes also occur from time to time in the membranes bounding a cortical tumour, whilst naturally vascular disturbances are a prominent feature where frankly inflammatory lesions are concerned. In the immediate neighbourhood of many of these lesions the appearances are such as to suggest that rapid or sudden alterations of vascularity could readily take place, and such changes seem to me to constitute the one common factor which is capable of explaining the associated epileptiform movements, and so bringing their causation into line with that of other forms of epilepsy, whether traumatic or apparently spontaneous.

In the surgical treatment of the cases included in this first category, namely of recognizable and localizable gross lesions, the operation is naturally directed primarily to dealing with the tumour or other lesion, and the result as regards the fits, as well as other symptoms, will depend upon its nature and the completeness with which it can be removed. When a simple tumour can be completely removed, or an abscess shelled out entire with its capsule, the outlook is good. When an abscess has to be drained, fits are not so likely to be abolished, since the drainage necessitates anchorage of the brain to the surface, which is in itself, as I have endeavoured to show, provocative of epilepsy.

The *second group* comprises those cases in which fits of a definitely focal character occur, but in which the nature of the lesion, if any, cannot be ascertained without actual inspection of the brain. Operation, which is fully justified in many of these cases, must necessarily be primarily of an exploratory character. In spite of the fact, however, that the technique of cranial operations has been so greatly improved, we must not fall into the error of putting a cerebral exploration quite on a level with an abdominal exploration, or undertake it with the same confidence that, even if nothing is found, no harm will result. By such exploratory operations I have found tumours, meningeal cysts, on one occasion a nævoid angeioma, and on another occasion a small abscess, all of which were capable of being satisfactorily dealt with.

On the other hand, a wide exposure of the cortex may reveal no visible lesion of any kind; the brain may appear to be absolutely normal both to inspection and palpation. In such a case the question naturally arises as to whether that portion of the cortex in which the initial movement is represented, having been defined by electrical excitation, may be excised. The effects of cortical excision upon function are, thanks to Horsley, Sherrington, Marinesco and others, well known; but the results of such excisions in patients suffering from focal fits without visible lesion have not, even in the hands of the most expert operators, proved very encouraging.

Cushing, from an experience of some fifteen cases, writes as follows: "Some had unexpectedly resulted in cures; one or two which promised to be especially favourable owing to the circumscribed nature of the initial movement (thumb and corner of mouth) were not benefited."

Krause gives details of three cases of focal epilepsy without visible lesion in which he excised what he calls the "primary spasming centre." One patient had had no fit some months later; one eight years later only had occasional twitchings; and one died shortly after the operation in status epilepticus.

Krause attributes the failure of cortical excisions in the hands of others to the localization having been made on anatomical grounds and not by electrical excitation. He believed the resultant scars to be of no importance, and not provocative of any spasm or symptoms whatever.

It is clear that in determining the advisability of performing cortical excision the resultant disabilities would be of less account in dealing with focal epilepsy starting in parts already deprived of their function. The published cases are too few and too insufficiently recorded to afford good ground for any definite conclusions. But the subject cannot be altogether dismissed without further investigation.

The *third group*, namely that of the so-called idiopathic epilepsies, includes all the cases in which the fits can neither be ascribed to any ascertainable organic lesion, nor possess any features pointing to a definite focal origin. I have searched in vain amongst the many records of operations for epilepsy, some purely fantastic, some based upon a more or less reasonable conception of its causation, for any encouragement to pursue further the quest for an operative procedure which holds out any prospect of benefiting the sufferer from idiopathic epilepsy. It may be unwise to say that the future will reveal nothing fresh in this direction, but it is at any rate certain that, at the present time, surgery has nothing to offer in the treatment of general idiopathic epilepsy.

In some other forms of epilepsy surgery may, as I have indicated, be made to play a useful part, but no operation can alone be expected to effect a cure. I am, however, convinced that, in properly selected cases, surgical treatment can be of great value in lessening the task demanded of medicinal and other therapeutic measures. If these measures cannot be carried out rigorously over a prolonged period, then, for the good of the patient and the credit of surgery, it were better to refrain from operating.

Cynical friends have told me that operations upon the nervous system do but illustrate the truth that hope ever triumphs over experience. The surgeon must be an optimist, but his optimism need not be that of the ostrich. It should be based upon the expectation that inquiry into the causation of those diseases which he seeks to remedy will eventually furnish the clue to their successful treatment. The search may lead him into deep waters, and this must be my excuse for venturing so far out of my depth as to address a gathering of neurologists on such a subject as epilepsy.