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Focal epilepsy

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FOCAL EPILEPSY

By

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PREFACE

The purpose of this thesis on Focal Epilepsy is to compile, as nearly as possible, into one article, the development through to present day concepts concerning a subject but vaguely mentioned in the literature as such.

I have attempted to trace the development of the subject from the initial, brilliant works of Hughlings Jackson to modern authorities' views.

It is a hard subject to organize into a logical form without straying into a too long discussion of the factors underlying this symptom complex.

I have stressed the historical development; the more or less minute description of the actual phenomena; and the differential diagnosis from the standpoint of brain tumor.

It is far from a complete article, most of the literature being in foreign languages.

INTRODUCTION AND DEFINITION

The subject of this thesis, "Focal Epilepsy," will be considered here as synonymous with "Jacksonian Epilepsy;" using that term here in a broad sense as meaning a symptomatic monospasm or hemispasm due to cortical or sub-cortical discharge; a spasm which usually exhibits, if closely studied, an initial symptom and a serial order progress.

As originally and commonly employed today, the mere use of the term is supposed to predicate the existence of a focal lesion of the motor cortex, and it is sometimes defined as a spasm limited to a single group of muscles, or at least to a few muscle groups. Irritation or instability of portions of the cerebral motor area, in by far the largest number of cases of focal, or Jacksonian spasm, as here defined, is a direct or indirect cause of this form of epilepsy. But it is by no means true that it is always or even nearly always due to gross lesions of the motor zone. (80) Collier, in fact goes so far as to state that the commonest cause of Jacksonian epilepsy is idiopathic epilepsy itself. (19)

Most writers at the present time believe that the term Jacksonian epilepsy should be extened to include any epileptiform seizures with localizing features; be they of motor or sensory nature. (26) The sensory disturbances may accompany the spasm or be the only manifestation of the cortical explosive action. (83) Dr. Taylor (108) thinks the term should be limited to focal attacks not associated with loss of consciousness. This was Jackson's original idea, according to Dr. Taylor, and it seems desirable to him not to extend it to include local attacks which may be accompanied by loss of consciousness.

Almost seventy years have passed since the pioneer work of Jackson and others was done without materially altering the facts. It is recognized that cortical irritation or possibly the removal of inhibitions may lead to a local discharge which, if not too extreme, results in sensory or motor response without loss of consciousness...the type of attack to which the term Jacksonian has been applied. That this, however, is a matter of degree, rather than kind, is shown by the fact that there are various gradations between focal attacks without loss of consciousness and others beginning in the same way which ultimately become

generalized in character with loss of consciousness. (108)

Penfield (89) has recently said, "when one studies the cases of so-called idiopathic epilepsy or generalized epilepsy, a large percentage of them prove to belong to the group of focal epilepsies; and it seems to me that Jackson's original conception should be expaned now to include focal epilepsy."

Leopold and Auer, (72) however, hasten to mention that many generalized convulsions may appear as local convulsions owing to the fact that part or the whole of one hemisphere is thrown out of action due to another cerebral lesion. This is frequently seen in cases of infectious hemiplegia associated with epilepsy where the paralyzed limbs are in a state of spastic contractions; and in which the generalized convulsion makes itself more apparent on the sound side.

This apparent Jacksonian phenomena is in reality an abortive generalized convulsion and must be distinguished from true focal epilepsy. They (Leopold and Auer) would place this type with the pseudo-Jacksonian epilepsy distinct from the interpretation of the term used by Ballet and Crespin, (4) who applied the term to the unilateral seizures of idiopathic epilepsy.

In this paper then, the term focal epilepsy will denote a symptomatic epilepsy due to an irritative lesion affecting part of the motor area, not necessarily a gross lesion of the motor zone alone; presenting localizing features of motor or sensory character, and synonymous with a broad interpretation of the term Jacksonian epilepsy as above defined. It seems wise to me to avoid a categorical definition as to whether or not consciousness is lost or retained in this conception; although modern opinion favors both. As to classifying many of the idiopathic epilepsies as focal or not, I believe the matter too controversial to strictly delimit the tow; and the following discussion will, in main, avoid more than a presentation of modern views.

HISTORICAL SURVEY

Perhaps too much space is given to a rather complete account of the historical development and theory underlying the theme of this paper; but I found it of particular interest. Especially when one considers that the pioneer work begun in the development of this subject is basically the foundation upon which are builded many of the present day theories of brain function and localization of function. Much space is given to the works of Dr. Hughlings Jackson in particular, the outstand- figure in this field...many of whose theories are still keeping pace with present day knowledge on many subjects of neurological import.

The majority of the original works listed below by Leopold and Auer I was unable to find even with diligent search of the indices. They did not list their reference sources, and so the majority are merely quoted.

While Bravais in 1827 (11) was probably the first to fully describe and appreciate unilateral convulsions, it was in reality the work of Hughlings Jackson that showed the association of this phenomena with lesions of the motor cortex and laid

a long-enduring foundation for present day knowledge and views of localized or focal epilepsy. (38)(40)(41)(42)(45)

The importance of Jackson's efforts may be more clearly understood when one considers that Flouren's theory of the physiological equality of all parts of the brain was still in vogue. (35) Numerous observations were reported thereafter until the firm conviction was established that a sharp differentiation could be made between the generalized epileptic convulsion and the Jacksonian type as a convulsive type significant of a cortical lesion. (40)

Pritchard in 1822 first described local convulsions and in 1827 Bravais (11) called the condition "hemiplegic epilepsy" because of the paralysis following the attacks. Abercrombie described in 1828 certain cases distinguished by convulsions "without any affection of the senses," where he said, "the convulsions may be confined to one side of the body," and he noted that in one case there was a tumor on the opposite cerebral hemisphere; and in another case a portion of the opposite hemisphere was indurated. This was the first report of pathological findings associated with local convulsions but the author did not ascribe the convulsions as due to his findings.

Elliotson in 1830, designated the condition as partial epilepsy. Hughlings Jackson, by his remarkable works on the subject set on foot a train of investigation by different workers which culminated in the localization of the motor areas of the brain. (67) (68) (72)

Hughlings Jackson

The origin of his special investigations on epilepsy began in a case reported by Broadbent in 1866 (12) in which right unilateral convulsions were followed by a temporary defect of speech. During the following years he pondered more and more over the significance of these unilateral convulsions and he published several detached notes on his observations of them. (66)(63)

He was the first to suggest the possibility of the existence of motor centers for various movements within the area supplied by the middle cerebral artery; and that unilateral convulsions resulted from stimulation or irritation of these centers. (59)

Early in 1871 his speculations form a series of brilliant hypotheses corresponding entirely to clinical observations but lacking final confirmation. At that time the physiologists were teaching that the

brain could be sliced away without material loss of function; and Jackson's views that local convulsions were produced by a local lesion which discharged the nervous centers of the cerebrum were passed over in contemptuous silence. (68)

Later in the year 1871, however, Hitzig and Ferrier showed that the cortex of the brain could be stimulated and Ferrier, in 1872, in a series of most elaborate experiments, confirmed Jackson's observations almost exactly. (86)

Jackson commenced his arguments by suggesting that a unilateral convulsion represents the converse of unilateral paralysis, or hemiplegia, and both are the result of disease in closely related areas of the brain. "Hemiplegia shows damage (equivalent to destruction) of the motor tract; hemispasm shows damage (equivalent to changes of instability) of the convolution which discharges through it. Palsy depends on destruction of fibers and convulsions on instability of grey matter." (65) He regarded the brain as an organ that stores up energy in the cortex and discharges through the fiber tracts. Under normal conditions the discharge arises from a definite, adequate stimulus, and discharges in an orderly, quiet

fashion. A focal epilepsy, on the contrary, is a disorderly overpowerful discharge that follows a pathologic form of stimulus acting on an unstable cortex. Areas of cortex that are the more richly endowed with cells are more likely to become unstable since these areas control great amounts of energy, are potentially more explosive, and are more easily discharged. In these complex relatively easily discharged areas are represented movements themselves correspondingly complex; for example, those of the fingers and hand.(65)(59)

Jackson considered each case from a threefold angle of investigation: Anatomical: the lesion, he said, is of this or that part of the cerebral cortex. Physiological: The underlying cause is basically a local high instability of cells of that part. Pathological: The disordered nutritive process is the primary cause and is the result of occlusion an artery supplying that part.(55)

Jackson noticed that when convulsions began on the right side, speech was lost, and this observation laid the foundation for later work on aphasia. He also dwelt at considerable length on the peculiar phenomena that a ligature tied proxi-

mal to the starting point of the convulsion may abort that attack (59) and that convulsions may start in areas which are the site of local injury. (57) In the further development of his comparison of hemispasm with hemiplegia he recalled the observation that certain muscles acting together, for example, those of the eye and the intercostals, are not paralyzed in complete hemiplegia. Broadbent's theory of double innervation satisfactorily explains this by assuming that muscles on both sides of the body which happen to work together receive a nerve supply from both hemispheres. In lesions of only one hemisphere they are therefore not paralyzed. If Jackson's theory as to the relationship between hemiplegia and hemispasm is correct, then in a case of unilateral convulsions the reverse ought to occur, namely, such muscles ought to be bilaterally affected. Jackson demonstrated that this was so, further supporting both his own ideas and those of Broadbent. (59) (12) (86)

Jackson hypothesized that there were three levels of function in the central nervous system. These are based on no anatomical or physiological subdivision but fit in well with his theories. The lowest level includes all the motor cells and fib-

ers below the level of the cerebral cortex and is called the first or lowest level of function. The second or middle level includes the so-called motor cortex of both cerebral hemispheres; the third level includes all portions of the cerebral cortices aside from the motor areas. At first Jackson believed that all convulsions were due to discharge from the cerebral cortex but toward the end of his work he hypothesized that convulsions initiated at each of these levels. The convulsions beginning at the second level of function, or in the motor cortex are the localized type that are now commonly known by Jackson's name. (49) (53) (71) (110)

Jackson recognised that this type of localized attacks were caused by "coarse disease" or the pressure of foreign bodies such as gummata and tumors upon the gray matter in the region of the cerebral cortex motor areas. He also observed the changes in the optic discs that so often accompany these attacks. (61) (71) (55)

Jackson gave a possible suggestion for the fact that the attacks begin in the fingers or the toes, the most voluntary muscles of the body. In his opinion these muscles were represented in the cortex by large numbers of relatively small cells that pre-

sent a large total surface area which is easily influenced by any change in the composition of the blood. (71) Jackson concluded that an insignificant lesion in the highly organized cortex may produce local convulsions by forcing the surrounding healthy tissue to cooperate with it.

Fritsch and Hitzig, in 1870, mapped out definite areas of the motor cortex by means of the application of electrodes. (72)

Broadbent in 1883, (13) described well localized and unilateral convulsions in Bright's disease. A year later Ballet and Crespin (4) described partial convulsions as occuring as part of hysteria. In 1885, Fournier (29) added yet another classification which he called syphilitic epilepsy. Laborde was the first to ascribe alcohol as an etiological factor. Chantemesse and Chauffard, urea; (72) and stated that in uremia the toxic substances were capable of exerting a selective influence on the motor area to produce typical focal convulsions.

In 1890 Bonhoeffer called attention to the fact that localized convulsions could be the manifestation of an idiopathic epilepsy. (72) Rausier in 1893 (94) classified Jacksonian epilepsy from an etiological standpoint. Fuchs in 1904 (31) presented

a resume of his observations on sensory phenomena.

Dr. Jackson further described a variety of tonic spasms which occur in cases of tumor of the vermis, and also the uncinate tasting group of fits due to an area of softening in the uncinate gyrus. (51) Collier in 1904 (19) reported several cases in which (neoplasms) located in the cerebellum produced Jacksonian convulsions and advanced the idea that the late onset of Jacksonian convulsions indicated a lesion distant from the motor cortex.

Bonhoeffer, in 1906, (8) in a study of unilateral convulsions stated. (a) "if it is true that Jacksonian epilepsy is a frequent symptom of a lesion of the Rolandic convolution, it is not less certain that it may result from a lesion situated distant from this region, even on the side. (b)Cerebellar lesions produced convulsions generally on the same side. (c) Jacksonian epilepsy sometimes occurs in individuals with generalized epilepsy due to another cause. (d) Jacksonian epilepsy sometimes occurs in individuals due to hydrocephalus. (e)it may appear spontaneously without explanatory findings. (f)There might be signs of Jacksonian epilepsy pointing to tumor or abscess and the autopsy be negative."

Further references which could be placed under

this historical resume will be referred to under other sections to which they bear a direct reference.

PHYSIOLOGY AND PATHOLOGY

Physiology:

Few, inconclusive, and conflicting theories and observations have been brought forward supporting any one physiological precept underlying focal epilepsy.

The underlying mechanism in producing focal spasms, was, in Jackson's opinion, the production of a local area in the cortex having a high instability due to disordered nutrition; which, in turn, was caused by an occlusion or plugging of the arterial blood supply to that region. This, he felt, was true, no matter what the underlying etiological cause might be. (55)(64)

Little more is known today than the theories advanced by Jackson. In support of the above, Leriche in 1920 (75) reported a case in which he witnessed the development of an attack while performing an exploratory craniotomy. He noted a sudden spasm of the cerebral arteries which produced an immediate and pronounced anemia of certain portions of the cortex.

Potzel and Schoffer in 1924 (91) noted local edema of the surface of the brain coincident with

a seizure during operation. Following this observation further, they produced similiar changes in dogs by faradic stimulation of the brain cortex.

Dragstedt in 1924 (24) demonstrated experimentally produced Jacksonian epilepsy in dogs by electrical stimulation to the motor area by means of brass screws which just penetrated the dura over the cortex. He made no note, however, of any changes accompanying the spasms.

Leriche (74) brought about attacks of true focal epilepsy in predisposed individuals by inducing abnormally high intra-cranial pressures. The pressure was modified at will by intra-venous saline. In one his cases repeated spinal taps to bring tension to normal was all that was needed to cause a cessation of attacks.

In another of his reports (73) Leriche presents thirty cases of focal spasms following war skull injuries. He believed that in these cases neither foreign bodies in the brain nor lesions of adjacent meninges were responsible for the convulsions. He also believed the share of infection was insignificant. He noted that the occurrence and frequency of convulsions were directly related to changes of tension of the cerebro-spinal fluid.

He could induce experimental spasms by hypotension as well as hypertension. In his opinion the withdrawing of an excess of fluid and the injection of hypotonic or hypertonic solution as the case may be should often be preferred to operation.

Pathology:

The pathology varies to some extent with the etiological factor----tumor, gumma, trauma, etc., assuming the characteristics of each to some extent. The area affected by any of these undergoes fairly constant morbid changes. The membranes may be adherent to one another, thus forming the starting point for wedges of connective tissue which grow downward at the expense of brain tissue. The nerve cells in the affected area are found in various degrees of degeneration, or they may have entirely disappeared, having been replaced by islets of neuroglia which have developed in the course of the arteries that pass perpendicularly from the surface into the brain cortex.

These changes are sufficient to permit focal epilepsy to be regarded as due to organic focal cerebral lesions, but in furnishing a pathological anatomy they do not explain the paroxysmal

nature of the symptoms. If the cerebral lesion is sufficient to cause paralysis, that symptom can be understood by remembering that injury to the cortex has injured or killed the cells which are the essential factors in voluntary motion. But why cells, whether they do do not retain the power of causing voluntary movement, should from time to time become the seat of irritation and thus cause convulsions, remains unexplained. (3)

It is a question today as to whether the movements are due to stimulation of the cortex (irritation) or to the removal of an inhibitory factor. (27)

In either event a group of muscles only has been cut off from effective associational relationships with higher levels. There is no defect of biological adjacents. A portion only of the machine has been damaged; and this damage is confined to sensory and motor components of the motor cortex. (69)

ETIOLOGY

The exact causative type of lesion is not as easily surmised as the location in most cases. Bailey (3) believes, as a generalized opinion, that no matter what the cause, it is probable that in the predisposed the tendency of the convulsions to become generalized and progressively worse is more pronounced; but the first seizure occurs independently of predisposition.

The literature is replete with reports of groups and single cases of focal epilepsy as a result of injury to the cerebrum and intra-cranial neoplasms. There is no single large group of different etiology.

Of historical interest is Rausier's classification in 1893 (94) of what we are speaking modifiedly of as Jacksonian epilepsy from an etiological standpoint according to: (1) Affections of the Bone: (a) tumor, (b) exostoses, (c) splinters. (2) Affections of the Meninges: (a) tumor, (b) acute, sub-acute, and chronic meningitis, (c) hemorrhage, (d) blood extravasation in pachymeningitis. (3) Affections of the Brain: (a) old encapsulated abscesses, (b) cerebral sclerosis, (c) tumor, (d) hemorrhagic foci, (e) areas of softening. Special emphasis

was laid on trauma. This classification still encompasses most of what is known today as to the etiology of focal epilepsy.

Fincher in 1931 (27) reported a series of one hundred-and-thirty cases classifying them as follows: (a) Trauma 34%, (b) Brain Tumor 24%, (c) Post-Influenzal 10%, (d) Cerebral Atrophy 5%, (e) Syphilis 5%, (f) Arterio-Sclerosis 3%, (g) Miscellaneous 3%, and (h) Undetermined 8%. Other reports citing syphilis as an etiological factor include Abrahamson (1), and Berardinelli (7) with others. Sauz (99) emphasizes post-influenzal focal epilepsy.

Trauma and tumors, especially of the motor cortex, but not always, are preeminently associated with this dramatic form of epilepsy. The lesions of the motor cortex other than tumor which may be associated with it are: (1) Sub-Dural Hemorrhage, (2) Meningo-Encephalitis, (3) Localized Meningitis, (4) Depressed Fracture, (5) Brain Abscess, (6) Cerebral Softening second to Vascular Disease, (7) Foreign Bodies, (8) Multiple Sclerosis, (9) Angioma. (114) Harris (34) reports meningitis as a cause and Patrick (80) gives a case report of multiple sclerosis giving rise to typical focal spasms.

Focal spasms are sometimes observed in toxic

states, namely, diabetes, (93)(81) uremia, acute infectious diseases, and alcoholism, without any demonstrable pathological change in the motor cortex. Oppenheim (84) says that alcohol is especially liable to produce this form of spasm.

Hysteria may simulate the condition (21) and idiopathic epilepsy frequently has an "inside focal spasm." (114)

Numerous articles have appeared in the recent literature designating cysticercus as an etiological factor in the production of focal epilepsy. Minchin (81) and Perry (90) report the most cases. Perry says that the effect of cysticercus on the brain depends upon the location and size of the cysts. He advances two theories as to the cause of the focal convulsions: (1) Due to toxins from parasitic abscesses. (2) Due to cysts being free in the ventricles and from time to time blocking an aqueduct.

Somewhat in keeping with Perry's toxin theory is that advanced by Sironi (101) in reporting a case of focal epilepsy due to ascaridiasis. He theorizes that some relic of a previous infection or toxic process had left a point of lessened resistance in the cortex upon which toxins of the helminths were acting

to cause the typical spasms. His cases all recovered after medication for the worms was given.

DESCRIPTION OF MOTOR AND SENSORY FOCAL SYMPTOMS

Motor

Irritation of the cortical motor zone produces convulsions in the muscles of the opposite side of the body. (84) The most frequent attacks of focal epilepsy are of a motor character and consist of rhythmic clonic jerkings or spasm of the muscles of one side of the body. (26) A tonic spasm generally precedes the clonic twitching. (84) These spasms most commonly begin in the upper or lower extremity but not infrequently they have their beginning in one side of the face. (26) (59)

Spasmodic turning of the head and eyes, or of the eyes alone, to the right, indicates irritation of the posterior part of second frontal convolution in the left hemisphere. Such movements to the left indicates irritation in the same area of the right hemisphere. The head turns in a series of jerks and the eyes have conjugate lateral nystagmus. These go on until the patient looks over the shoulder. (105)

Spasmodic twitching of the facial muscles on one side indicates an irritation in the lower third of the motor area at the foot of the central convo-

lution. Such a spasm may begin in the forehead, in closure of the eye, in the muscles of the nose, in the tongue, about the mouth, or there may be laryngeal spasm. Wherever in the facial area the irritation begins it usually spreads throughout the entire region so that the entire side of the face is thrown into spasm. The spasm is clonic, after its preceding tonic spasm, the rate of movement being six or eight contractions to the second and may continue for several minutes. (105) The twitchings excited by repeated or several irritations do not limit themselves to the muscle group first involved, but extend over the whole side of the body usually, as if the irritation in the brain had extended to the neighboring centers. (84)(26)

As Jackson stated in 1868 (63) the spasms "usually begin in the arm, nearly always the hand, and mostly in the thumb and index finger." He says (59) the convulsions most frequently start in those parts on one side of the body "which have the more voluntary uses(which have the greatest number of different ranges and intervals of movement).

Convulsive movements limited to the upper extremity show an irritation in the middle third of the motor area. The spasm may begin with a drawing

upward of the shoulder and turning downward of the head, or by movements of the arm at the shoulder-joint; by movements of the elbow of flexion or extension, or by movements of the wrist and fingers and thumb. The spasm usually begins in the hand and ascends the arm, involving all the muscles in the reverse order named. Usually the hand is closed, the wrist flexed and pronated, the elbow flexed, and the arm adducted when the spasm is at its height. The flexors thus overcome the extensors in the conflict between both in the spasm. (105)

Spasms beginning in the upper extremity may extend to the face, eyes, head, trunk, or leg.(105) Beginning in the facial area the spasm extends first to the arm (hand and fingers first), and finally to the leg of the same side. If they commence in the leg they extend to the arm and finally the face. (84)

Convulsive motions limited to the lower extremity may begin in the thigh and extend down the leg to finally involve the foot and toes, or they may begin with a twitching of the toes which extends to the ankle and knee, and finally to the hip. The lower extremity is usually extended in an attack, is held rigid; the extensors overcom-

ing the flexors in action. (105)

Course:

The attacks may terminate as a complete seizure at any point in their progress or there may be a generalized convulsion as a finale of the localized seizure. The march of the attacks, following consistently the same pattern of advance, is but another characteristic of these motor irritative phenomena (26) and is governed by the spread of irritation from one cortical region to the adjacent regions. (105)

There is a tendency most commonly for the attack to increase; which increase may be both in severity and frequency. On the other hand, in a few rare instances, the attacks may diminish. This clinical observation should not be misleading, particularly if the etiological factor is undetermined. The causative lesion here may be a tumor. (26)

Consciousness:

Consciousness generally remains intact in the local muscular spasms; it may, however, disappear in the course of the spasms if they extend over the entire half of the body. Unconsciousness also generally occurs when the other side of the body becomes affected. (84) Views expressed by various auth-

ors are not unanimous on this point. Jackson's original idea seems to favor no loss of consciousness. (40) Some of his later works, however, stressed the fact that in focal epilepsy consciousness is lost late in the course of the spasm.

Phenomena After Focal Spasms:

In those instances where the localized seizures have terminated at some point in their progress, there is always aperiod of weakness of the involved extremity which may amount to an actual flaccid paralysis. (26) The weakness or paralysis presents an order of termination in the limbs which is just the reverse of the order of invasion of the spasm.

In permanent paralysis, the paralysis extends in the reverse order, following the order of extension of the spasm. Thus the order of extension of a spasm and of a permanent paralysis is just the reverse of the recovery in a temporary paralysis. (105)

The nature of the cause of the irritation often, however, is such that the cortical centers are not only irritated, but also to some extent injured. This is the reason that chronic conditions of paralysis are often seen in focal epilepsy.

Jackson (42) suggests that the temporary local paralysis after a convulsion is due to a temporary exhaustion of more or less units of the corpus striatum "by the excessive discharge of some part of the cortex in the paroxysm." An explanation that is accepted today. (84)

In right-handed individuals with right-sided attacks, after the spasms have subsided, there may be present a speech difficulty of a variable degree; ranging from difficulty in pronunciation to a complete aphasia. (12) (97) (42)

Diagnostic Value and Interpretation:

Local spasms do not invariably indicate a primary lesion in, and direct irritation of, the motor region. Such irritation may be indirect and irritation may start from a point on the cortex outside the motor area, and, as it extends, finally reach that area, and then sets up a spasm. The spasm will be in the limb, the motor area of which is the part reached by the irritation, and then if the spasm extends it may follow the regular order. (105)

A disease, however, which produces paralysis of the cortical centers must have its seat in the motor cortex, or impair its functions by pressure. (84)

Sometimes and irritation, starting in the occiptal or parietal region and extending forward, reaches the motor area of the fingers and toes simultaneously. Then the spasm will begin in the hand and foot together, extending up both limbs at once. This may therefore be a clinical fact of importance in showing that the lesion lies outside the motor zone. (105)

The movements and spasms due to a cortical irritation always resemble voluntary movements. They are never contractions of single muscles or of groups of muscles which may have a nerve supply from one nerve alone, or which may have a relation in their spinal representation. Cortical spasms can always be differentiated from spasm due to an irritation of a nerve trunk, of a spinal nerve root, or of the motor mechanism of the spinal cord. They are not like reflex acts; the cortical movements can always be voluntarily imitated. They are always apparently for a purpose and show a certain amount of coordination and adaptation to an end. (105)

Sensory

Focal epilepsy may also confine itself to the sensory area, be associated with, preceded by, or followed by sensory phenomena. (84)(105)

That this was known to early writers is seen by reports by Jackson (39)(54)(58), Bramwell (9)(10), Robertson (97), and Broadbent (12).

The sensory attacks are usually paresthesias subjectively described as a numness, prickling, or tingling of the involved part. (26) In comparatively rare instances they may be exclusively sensory, leading to hallucinations; when in the speech area lead to temporary attacks of aphasia; in other instances to disturbances of consciousness, will, or memory. (105)

Anatomical Basis:

The anatomical basis of these attacks, it is generally assumed in analogy to cortical focal attacks, to be an excitation of the post-central convolution which starts at a certain point and spreads to the neighboring cortex. It is therefore usually believed that the spread of the paresthesiae over the body is determined only by the arrangement of the sensory areas in the gyrus centralis posterior. Cases are usually classified as to whether or not the sequence of the parts of the body affected by paresthesiae

corresponds strictly to the arrangement of the sensory points in the post-central gyrus. (102)

Course:

When associated with the motor spasms the sensory changes most commonly follow the same progress of march as the motor attacks. The sensory attacks, however, may stop short of the entire range of the muscle spasms. In extremely rare cases there have occurred sensory attacks on one side of the body with motor spasm involving the opposite extremities. (26) Any of these forms of attack may go on to local spasm and a local spasm in its turn produce one of these peculiar states. (105)

The tingling sensation which attends many of the focal attacks may precede the actual movements by some seconds or even minutes. (105) It is interesting to note again that if a strong irritation of the skin is made in or near to where the tingling is felt, the spasm may be averted. (59) (71)

Such a powerful sensory impression inhibits the extent of the cortical discharge but the subsequent paresis is apt to be more extensive when this is done. (15)

The tingling sensation is more commonly felt in the fingers, hand, toes, and feet, probably be-

cause the sensibility of these parts is more highly evolved and keener than in the others. The coincidence of these sensations with a local spasm is an argument for the coincidence of sensory and motor functions in the cortex. (105)

In the few cases of purely sensory focal epilepsy, again the attacks have a fairly constant place of origin, a definite progress, and like the motor attacks they travel the entire half of the body. The residua following the cessation of the sensory attacks may be a disturbance of joint sense or sense of position. Such a patient may be unable to recognize objects placed in the hand of the involved side. (26)

Fuchs (31) attempts to systematize the knowledge on sensory focal epilepsy and states that the phenomena of sensory attacks alone, or accompanying symptoms of motor orign, are found in the following conditions: (1) In the prodromal stages of progressive paralysis. (2) In diseased condtions of the brain which are limited in extent such as tumors, abscesses, cysts, etc. (3) In encephalomalacia. (4) in hemicrania symptomatica.

DIAGNOSTIC VALUE OF FOCAL EPILEPSY

With the exception of monoplegia or hemiplegia, probably no single symptom or symptom complex is so often made use of by the diagnostician in his efforts to fix the site of a lesion for the purpose of operation.

Irritation or instability of portions of the cerebral motor area, either by trauma or neoplasm, in by far the largest number of cases, is a direct or indirect cause of this form of epilepsy. However, it is by no means true that it is always or even nearly always, due to gross lesions of the motor zone. The greatest care should therefore be taken not to attribute to the local spasms erroneous diagnostic value. While in many cases it is an important guide, in others it may be misleading.

When a patient exhibits such localized epilepsy it must be remembered that in addition to neoplasm involving the motor cortex that: (1)lesions other than tumor of the motor cortex may produce unilateral spasm. (2) Tumors in parts of the brain remote from the motor cortex are sometimes associated with monospasm. (3) Toxic con-

ditions may produce attacks. (4) The myoclonic type of epidemic encephalitis may produce somewhat the same picture. (5) The so-called reflex epilepsy may do likewise. (6) Hysteria may simulate the condition, and (7) idiopathic epilepsy frequently has an "inside" focal spasm. (114)

BRAIN TUMOR AND FOCAL EPILEPSY

Location:

The greater number of tumors causing focal epilepsy are situated anterior to the Rolandic area and are found as cortical lesions. This is in keeping with Jackson's contentions that the attacks are the result of irritation, whereas paralysis follows destruction. (26)(65)

Focal epilepsy due to cortical discharge may occur as the result of tumors in other parts of the brain than the motor cortex. The irritation, owing to its intensity, spreads to the motor cortex. It is probable that in some of these cases an unusual instability of the motor cells plays some part. Cases of this kind, in which the lesion is in the cerebrum proper, and absolutely outside the motor zone are comparatively rare, and the focal diagnosis can only be made by a full consideration of the non-motor symptomatology. (80)

In some instances, focal epilepsy, apparently of the usual motor area type, is observed in tumors of the cerebellopontine angle or of the cerebellum. (80)

Fleming (28) reported a case of a true angio-

neuro-fibroma of the acoustic nerve with typical focal spasms.

Behavior:

The spasm may be limited to the face, the upper or lower extremity. At times, in such cases, the spasm partakes of the nature of the "epilepsia partialis continua" first described by Koshewnikow, and later by Spiller (104), and Burr. By epilepsia partialis continua is meant a form in which clonic movements at different sites occur between attacks of idiopathic epilepsy. (114) (20)

Seizures are more common in slow growing than in rapidly growing lesions. The more diffusely infiltrating astrocytoma and oligodendroglia have a higher incidence of attacks than the better demarcated glioblastoma dnd ependymoma. Important factors for the production of attacks are, richness of glial fibrils, calcifications, and encroachment of important cerebral arteries by tumor. (77)

Deep-seated cortical gliomas seem to have a lesser tendency to produce attacks than superficial tumors. Cases with focal seizures seem to have a longer life expectancy than those without attacks, regardless of the type of tumor. (77)

The focal epilepsy may be the only sign of

of an intra-cranial tumor as in a case reported by Spiller (104) in which left-sided spasm of the upper extremity were the only indications of a small cerebral glioma situated at the posterior end of the right second frontal convolution.

Or, as Gotten (32) emphasizes, unsuspected tumors may be present only to be diagnosed by encephalographic studies. In his series of fifty-six patients upon whom he performed encephalograms, over 5% were found to have brain tumors of the motor cortex totally unsuspected.

Localization is often difficult. Taylor (109) reports a case in which a large fibrillary astrocytomata was found impinging upon the pre-central gyrus in very close proximation to the arm area yet the only focal signs the patient manifested were sharply delimited spasms of one side of the face.

Those cases in which localized spasm is a so-called distant symptom are often confounded with the comparatively common cases in which the spasm is an invasion symptom. A mid-frontal or even a pre-frontal tumor not infrequently invades caudally until the motor region is grazed or implicated; typical focal epilepsy showing itself after psychic, graphic, and speech disorders have become prom-

inent. In these cases, if the tumor be meningo-cortical in position, spasm may precede paresis or paralysis, although sooner or later, in accordance with the amount of pressure or destruction of the motor region, the patient will become monoplegic or hemiplegic. (80)

Tumors invading backwards from the pre-frontal region, are, in Mill's (80) experience much more likely to cause focal epilepsy than those which originate in the parietal lobe and advance forwards. He has recorded several cases of parietal tumor in which the motor cortex and sub-cortex were invaded and in which spasm was not present even at a late stage of the disease. Destruction of the sensory cortex and sub-cortex, before involvement of the motor projection fibers would seem to give a certain immunity from such spasm. (80)

It is generally believed that typical focal seizures do not occur when the sub-cortical parts of the brain, such as the corona radiata, alone are involved and that when attacks have occurred as a result of cortical irritation, subsequent destruction of the motor cortex or of the sub-cortical motor pathways will bring about

a cessation of the convulsions. (17)

Endotheliomas may originate in the meninges and involve the brain, not by infiltration, but by contiguity and compression of its cortical surface; and since gliomas may originate in the essential cerebral tissue and invade the brain by infiltration of the sub-cortical region, the early appearance of focal attacks serves to assist greatly in determining the operability of the lesion and its type. Endothelioma, as they are recognized quite early, are a form most suitable for removal. (80)(85)

In Cadwalder's (80) opinion, a tumor impinging on the central motor cortex will, in the great majority of instances, produce focal attacks some time during the disease.

In certain cases gliomas may grow very slowly and without being accompanied by convulsions and yet the growth may actually infiltrate the cortical surface. This type may be confused with thrombosis of the Sylvian artery. (80)

<u>False Localizing Signs:</u>

Collier (19) draws attention to cases of focal epilepsy as false localizing signs of brain tumor. In his report of one hundred-and-sixty-one cas-

es he emphasizes: (1) That local signs appearing late in the course of intra-cranial tumor, where general signs alone pre-existed, are often of false portent. In one of his cases the patient developed late signs indicative of a lesion in the left posterior fossa. Yet at operation was found to have a glioma of the left pre-frontal region; the local signs being the indirect and secondary result of the tumor situated at a distance. (2) He stresses the relative frequency with which local signs have been due to the presence of vascular lesions such as meningitis, hydrocephalus, local spreading edema of the brain, and secondary deposits of new growths. He also reports two cases of cerebellar tumor in which local convulsions confined to the arm and face were observed. Other symptoms were of long duration when the spasms first occurred. The occurrence therefore of local convulsions long after the general signs should be disregarded or considered with caution. Collier believes they are usually the result of secondary hydrocephalus.

DIFFERENTIAL DIAGNOSIS:

So important is the question of localized convulsions in the focal diagnosis of brain tumors that the site and nature of various lesions and conditions producing this symptom will be considered at some length.

Spasm Due to Dural Irritation:

This must not be overlooked, especially as certain forms of neoplasm commonly grow from the inner surface of the dura. Convulsions due to intense dural irritation may occur as one the symptoms of a brain tumor situated anywhere within the cranial cavity as has been demonstrated by clinico-pathological observation corroborated by physiological experiments.

One point of distinction between cortical spasm and the convulsive affections due to dural irritation is that in the latter the spasm, if observed at the start, will be found usually to begin in the face or limbs of the side of the irritation, spreading, however, rapidly to both sides of the body. This spread is so rapid that the initial spasmodic phenomena may pass unobserved. The spasms almost always become generalized, and tonic spasticity is prominent.

Such spasms are really reflex in character, the irritation being transmitted to the bulbar centers of the same side, and thence to those of the opposite side. In addition to the peculiarities of the spasm just noted, the diagnosis from cortical spasm will, of course, have to be made by the other focal symptoms present as by those indicating parietal, occipital, temporal or mid-frontal disease. (80)

Nature of Lesion:

The nature of the lesion of the motor area causing localized spasm will be next discussed. Decision as to the nature of the irritative lesion is particularly difficult in those cases in which the general symptoms of brain tumor such as optic neuritis and recurring headache are absent.

The gross lesions of the motor zone which produce focal epilepsy simulating that caused by tumors are: (1) Depressed fractures. (2) Localized Meningitis. (3) Meningeal Encephalitis or Cortical Polio-Encephalitis. (4) Focal Necrosis Occurring from Embolism or Thrombosis, including cases associated with Generalized Arterio-Sclerosis. (80)

Depressed Fractures:

It would only be the the cases of old fractures of the inner table with slight or no outward evidences of the injury, that the mistake would be

made of holding that the spasm was due to a tumor.

It occasionally happens that an injury to the head which leaves but a small scar results after a considerable time in the development of focal epilepsy. Usually, in such cases both headache and vertigo, more or less severe and persisting, are present and a tumor may at first be inferred. Close investigation into the history of the case and as to evidences of injury will usually make the diagnosis apparent.

Mills (80) holds that a mistake is not necessarily of such serious import in these cases, as tumors often develop at the site of the old traumatism, and any case surgery is indicated for the relief of the causative lesion of the irritation. Localized meningitis with adhesions or even an abscess may, of course, occur at the site of such an injury.

Localized Meningitis:

Localized meningitis which is not of traumatic origin nor connected with tumors, is usually of syphilitic origin.

In the form of a gummatous meningitis that is more or less amenable to active specific treatment, too hasty decision in favor of oper-

ation is occasionally given. On the other hand, operation is undoubtedly indicated in some cases with a luetic history and with evident vascular and meningeal specific disease, but in which the case does not respond even to the most energetic medicinal treatment. In some of these cases occlsuion of vessels takes place and deposits become organized so that they cannot be influenced by absorbent remedies, and the lesion, although at first a simple active localized meningitis, becomes an inert and irritative mass which should be dealt with surgically. (80)

Cortical Hemorrhage:

Cortical hemorrhage of slight depth and irregular distribution often times occurs in cases of sinus and venous thrombosis. Mills reports a case of this sort in which focal epilepsy clearly defining certain sub-areas of the motor zone was the most characteristic symptom. (78)

In such a case the history of acute or subacute onset, and symptoms of sinus and venous thrombosis guides one's final diagnosis. A hemorrhage or cyst remaining after a hemorrhage from one of the branches of the medial-cerebral artery may give rise to localized spasm and oth-

er symptoms simulating somewhat those of a tumor. The diagnostician is usually not called upon to differentiate in such a case until weeks, months, or years even have elapsed after the original lesions, and as the history is sometimes imperfect or confusing, and as the patients who suffer from such attacks are usually victims of arterio-sclerosis, they may have some headache and vertigo which may be regarded as general symptoms of a neoplasm. Associated with the paresis commonly present in such cases, there may be recurrent focal seizures. Close study of the clinical picture will usually be sufficient to throw the weight of opinion in such cases against tumor or localized meningitis. (80)

Focal Hemorrhagic Encephalitis:

This is a rare form of cortical disease which causes localized attacks and it may be regarded as a tumor of the motor zone in an early stage of its development. Exact diagnosis is difficult but fortunately, it is quite rare.

Arterio-Sclerosis:

Arterio-sclerosis which has caused either local instability or local softening must be considered when the question of operation for brain tumor

in the motor region is presented. It may bring out symptoms which simulate those exhibited mainly by neoplasms.

The cases of this kind which are most difficult to differentiate are those in which definite focal epilepsy is present. In such a case the history will be that of gradual cerebral failure following upon generalized arterio-sclerosis with its cardiac and renal accompaniment and a record usually of seizures and progressive loss of power of the limbs, or limbs and face of one side of the body.

Such a case is to be differentially diagnosed from one of brain tumor by a close examination of the manner in which the symptoms presented, by the exclusion of the general signs of brain tumor, and by taking into consideration the evidences or renal, cardiac, and general arterial disease. Optic neuritis is practically always absent and skillful ophthalmoscopic examination will often show evidences of sclerotic disease of the vessels and of some grade of optic nerve atrophy. Headache, if present, is not that which is often characteristic of brain tumor. Nausea and vomiting are usually absent. Dilatation with hypertrophy of the heart is present, and arterial tension is always high usually. The co-

existence of brain tumor and arteriosclerosis, however, should not be overlooked.

Somewhat of this same idea must be kept in mind in those cases in which vascular lesions occur with brain tumor, but in another location.

Focal Necrosis:

The occurrence of hemorrhage or necrosis due to thrombosis or embolism with brain tumor may give false localizing signs according to Collier. (19) He cites a case in which operation revealed an area of necrosis which was giving rise to the localizing symptoms and in which a following necropsy showed the presence of a large tumor in addition.

Hemorrhage is not very common in the progress of a brain tumor case; when present may be in, around, or at a distance from the site of the lesion.

Toxic States:

It is only in extremely rare cases that the diagnosis would be mistaken in the toxic states. Those in which headache or vertigo were present, or in which an accompanying optic neuritis of toxic origin is thought to be caused by a brain tumor.

Reflex Epilepsies:

The epilepsies due to dural irritation are of this type. A tumor situated in the dura, or a galvanic or faradic current applied to this membrane, stimulates the sensory branches of the fifth nerve distribute towards its inner surface, and this excitation conveyed to the bulb produces the severe and characteristic spasmodic symptoms.

Mills is of the opinion, that speaking broadly, the assertion may be made that peripheral irritation almost anywhere in the body may cause a focal attack in rarer instances. It is only in a very unusual case, Mills thinks, that the diagnosis of any cortical lesion such as brain tumor would be considered. He reports a case of this type, with typically focal spasms in both initiation and spread, was due to a fibroma of the palmar surface of the hand.

IDIOPATHIC EPILEPSY:

The question of the differential diagnosis of focal epilepsy due to tumor or other gross, and possibly operable, lesions of the motor zone, from idiopathic epilepsy is one of great importance.

Collier in1904 (19) says that the commonest cause of focal epilepsy is idiopathic epilepsy.

Mills (80) agrees with Collier in this respect.

Many operations have been done without revealing any tangible lesion, the diagnosis of the nature and site of the lesion having been made chiefly made because of the characteristic focal spasm which was the chief feature of the symptomatology of the case.

It is extremely rare in an idiopathic case for the spasm to remain limited to one limb, or even to one side of the body. However, this is not unknown, and patients may have abortive attacks of epilepsy with sensory or sensori-motor manifestation of a very transient character in a part to which the aura of a completed attack is commonly referred. (109) (Mills 80) (114)

In some cases in which focal spasms occur as part of the manifestations of an idiopathic epileptic attack, the focal attack occurs inside the general convulsion; in other words, the patient during the seizure has clonic and tonic spasms which involves more or less irregularly all parts of the body; but in which the spasm shows itself more pronouncedly in the limbs or in the face and limbs of one side of the body.

Jackson (60)(40) was of the opinion that almost every case of idiopathic epilepsy, if stud-

ied carefully enough, would be found to have had local spasm as the initiating phenomenon of the general attack.

The aura in idiopathic epilepsy is not as common in most cases, as that occurring in organic epilepsy and in those cases of epilepsy due to reflex cause. Idiopathic epilepsy is liable to occur at night; true tics and hysterical attacks scarcely ever at night, although one or two cases are noted in the literature. True focal epilepsy may occur at night. In focal or reflex epilepsy the patient is always awakened from sleep so that he is compelled to lie awake during the whole attack; in idiopathic epilepsy the patient is only awakened from sleep in those attacks which during the day would not cause him to lose consciousness.

An important symptom of focal epilepsy is the more or less pronounced paralysis or weakness of the part following an attack. This occurs most often in cortical or organic epilepsy.

Because of the frequency with which unilateral or focal spasm is used as a means of localizing brain lesions, it will be readily appreciate that many operations are performed and no explanation for the attacks found. (20) Wilson (114) says that a person with focal spasm should not be operated upon unless other signs

and symptoms of intracranial disease are present.

Jackson's own differentiation between idiopathic and focal epilepsy is as follows:"(a) As To Affection of Consciousness: the distinction is not that consciousness is lost in one and not in the other, but that consciousness is lost the first thing, or very soon, in one (idiopathic), and late or not at all in the epileptiform (focal) seizures. (b) As to Spasm or Convulsions: convulsions begin more nearly bilaterally in idiopathic epilepsy; is more nearly universal at once, is more rapid, and of shorter duration. In the other, it begins very locally on one side, hand, face, or foot, and becomes universal very gradually or not at all. (60)(40)

Hysterical Epilepsy:

Hysteria may be the cause of focal epilepsy. (21) Wilson (114) says that they are not nearly as common as believed. He could not recall seeing a single case among the numerous psycho-neurotics he came in contact with during his really extensive work in the army. He believes the general makeup of the individual and a careful evaluation of the signs and symptoms should render the diagnosis apparent. (114)

A condition which might be confounded with focal epilepsy occurs sometimes in those who have been exposed to long continued heat. It consists of violent tonic spasms occurring at intervals of several hours in the muscles of the legs and forearms particularly. The fact that the spasms are very painful and may be excited by voluntary motion as well as the history should prevent error. (25)

FOCAL EPILEPSY DUE TO TRAUMA

In cases due to trauma the spasms have a tendency to recur and ultimately to become general, and to affect the individual in much the same way as general epilepsy does.

Pathology:

The morbid changes are much like those described in a previous section. There is frequently a fracture of the skull so that splinters of bone press upon the cortex. Or without fracture, there may be an exostosis beneath the site of original injury.

Etiology:

Males, in the active periods of life, are the most liable to severe head injuries, so they furnish the large number of cases of focal epilepsy due to trauma. Blows on the head with clubs or heavy instruments, striking the head in falls or in other ways, are not uncommonly followed by the disease.

In most of the cases the skull is fractured, either in the internal or external table, so that the brain is directly pressed upon. In some cases, however, symptoms develop as a result of an injury to the head which has not caused a demonstrable fracture.

Pistol shots when not fatal, may be the starting point of focal epilepsy. In such cases it is difficult to tell whether the bullet, or the fragments of bone it carries with it or the resulting scar is the immediate cause. Abscess formation, so common after bullet wounds, may first manifest themselves by producing typical attacks months after the injury. (3)(5)(27)(94)(57)

Interval:

The spasms may ensue almost immediately, or may be separated from the occurrence of the original injury by a period often varying from months to years.

In cases in which as much as five or ten years have elapsed since the time of injury it is difficult to prove the causal relationship of the injury to the convulsions; especially, as the convulsions are almost always general in such cases.

In the interval between the accident and the appearance of the first spasm the patient may be perfectly well after recovery from the acute effects of the injury.

Symptoms:

The focal attack is much the same as those

due to other organic lesions. When they have existed for a number of months or years the attacks become quite similar to the grand mal attack of idiopathic epilepsy with the exception that they continue to begin as a localized spasm.

On examination of the head there may be found the scar of the original scalp wound and a depression in the skull. The scar is usually not tender, and pressure upon it does not cause a fit. The skull may be fractured, however, without leaving any depression.

When a depression exists, the injured portion of the brain is not necessarily immediately beneath it. The bone may be splintered, or hemorrhage have occurred in such a way that the cerebral lesion is situated at some distance from the site of the external injury. (3)(27)(94)

TREATMENT OF FOCAL EPILEPSY

Surgery:

Practically all authorities agree that surgery is the only rational treatment to be undertaken in the effective treatment of focal epilepsy. (3)(109)(80)(82)(98)(98-a)

Sachs (98)(98-a) gives the following points in the surgical care of focal epilepsy: (1) Surgery, he believes, is advisable in those cases in which not more than one, or at most, two years have elapsed since the traumatic injury or the beginning of the disease which has given rise to the convulsive seizures. (2) The prospect of recovery is brighter the shorter the period of time in which symptoms have existed. (3) Simple trephining may prove sufficient. (4) Excision of cortical tissue is advisble if the epilepsy has lasted a short time and if the symptoms point to a strictly delimited lesion. (5) In cases of long standing, surgery is of no particular value.

He cites a series of sixty-nine cases upon which he operated as follows: Group I: In this group were eleven cases of typical focal spasm with extensive cortical degeneration. Nothing was accomplished by operation because the entire,

or greater portion of the cortex was involved. (2) Group II: Nineteen cases of typical focal spasm with findings of definite focal scars. Here the degeneration was very extensive; often containing huge degenerative cysts with areas of gelatinous tissue. (3) Group III: Thirty-nine cases, only eleven of which showed strictly focal signs accord-to Sach's criteria. He performed sub-pial resections according to the method of Horsley.

In all but one of these cases the convulsions were completely stopped. He emphasizes the importance of removing the entire center which is the seat of the irritative phenomena.

Rhodes (95) advocates the use of the electro-surgery-cautery in removing the area believing that there is less bleeding and fewer adhesions.

Tumors if encapsulated, as are the ones arising from the meninges, can be completely removed. Fincher (26) states that the cortical gliomas may be of a benign character, such that with evacuation of their cystic cavity and removal of the mural nubbin a complete relief from the attacks may be obtained. Other gliomata with subcortical invasion may be excised or removed by section or coagulation. In some instances bloc dissection and

if necessary, the removal of an entire lobe may be indicated. In other cases only partial removal is necessary. Fincher states that regardless of what the indications are at the time of exploration, surgery offers the most in an attempt to relieve these patients of their attacks. Permitted to continue to grow results first in total disability and ultimately in death.

Fincher further emphasizes that it must be borne in mind in discussing the tumor groups that too frequently stressed general symptoms of increased intracranial pressure, namely, headaches, choked disc, and vomiting, will not be present in about one-third of these cases. The absence of these symptoms is no contraindication for cerebral exploration and in patients with a history of trauma, with a history of encephalitis who exhibit motor or sensory attacks of a localized nature, the burden of proof lies in the exclusion of a neoplasm, even to the extent of an exploration.

In the traumatic cases cited by Fincher the removal of ablood clot either of recent or older origin results in a complete disappearance of the focal attacks. The excision of a localized brain scar, ev-

acuation of a traumatic cyst, or the release of a sub-dural collection of spinal fluid may be the indicated procedure in other traumatic cases.

In those cases, where there is no known factor as a causative agent and there is no demonstrable lesion of the cortex Fincher and Bailey(3) advocate that the cortex be stimulated by faradic current, the so-called epileptic zone demonstrated and destroyed.

Ochsner reports a case of focal epilepsy following trauma in which upon operation a sub-dural cyst was found over the motor area supplying the right arm and leg. His method of procedure in a case of this type is to transplant fascia lata and a considerable amount of fat over the area and he reports satisfactory results. (82)

All cases, of course, are not materially helped. Schultze (100) reports two cases in which operation gave only temporary relief and was soon followed by similiar attacks as before operation.

Alcoholic Injection of Brain Cortex:

Dowman (22)(23) believes the Horsley excision procedure is of doubtful value in that the defect is replaced by scar tissue, which, in turn,

may give rise to irritative phenomena. He reports two cases in which the so-called epileptic zone was first mapped out by faradic stimulation and then injected with 95% alcohol. In animal experiments the operation is excellently borne and no secondary adhesions of the structures occur, although histologically all the cortical cells in the area of injection, except the neuroglia, were found degenerated.

One of the cases reported was one in which the attacks began with twitching of the fingers of the right hand, particularly the index finger; soon after the onset the jerking involved the whole hand, then the arm and shoulder and right side of the face. The operation consisted of mapping out with mild faradic stimulation the areas for the arm and hand. These lay entirely in front of the Rolandic fissure. The stimulation was so exact that the isolated area for flexion of the index finger, for example was determined. The areas controlling the movement of the hand and fingers were then injected with 95% alcohol by means of a fine hypodermic needle, inserted from 0.5 to 1 cm. into the cortex. The whole in-

jected area was not larger than one's thumb nail. After the injection had been completed, faradic stimulation failed to give any response.

No attacks followed and the patient was dismissed. About two months following, the patient was doing very nicely. He had had two symptoms which seem to have affected only the muscles of his face and neck, and lasted only a few seconds. He had had one complete seizure which caused him to fall but did not last more than thirty to forty seconds. His fingers were very sensitive to heat, or the pricking of a pencil or pen even more so than his left hand, but the sense of touch was almost gone and there was a feeling of numbness at all times.

Dowman, in discussing the case, felt that perhaps too small an area of the cortex was injected. He believes that the history following the operation suggested that the so-called epileptic zone must have been more extensive than was at first suspected as the patient still had irritative phenomena. Of particular interest from a neurological point of veiw was that the sensory disturbances that still existed to a certain extent, although the injection was entirely in the pre-central convolution.

Dowman, in reporting these cases made no claim that injection of the brain cortex with alcohol would cure focal spasms. He felt, however, that the method suggested is worthy of being tried. In his eyes, the advantages over the excision procedure are that the alcohol method does not promote adhesions between the dura and the pia arachnoid, does not cause sufficient damage to promote adhesions between dura and brain, is safer and simpler, and apparently does not cause permanent paralysis.

Roentgen Ray Treatment:

Strauss reported in 1913 (107) that he had succeeded in improving conditions in certain cases of focal epilepsy by roentgen exposures. The war interrupted this work but he has recently resumed it and now reports favorable results in some cases. Opinion, in general, is that it is of doubtful value.

Use of Tuberculin:

As a specific in cases of tuberculous tumors. Broadbent (14) reports success in treating this type with tuberculin beginning with 1/100000 units and very slowly increasing the dosage.

CONCLUSIONS

1. Focal Epilepsy is a symptomatic epilepsy due to an irritative lesion affecting part of the motor area, not necessarily a gross lesion of the motor zone alone; it may be motor (usually) or sensory in nature.

2. From an etiological standpoint tumor of the brain and trauma are the two large causative factors in the production of focal epilepsy.

3. Irritation of the cortical motor zone produces typical clonic spasms in the muscles of the opposite side of the body. These spasms most commonly begin in the upper or lower extremity but not infrequently begin in the face. They exhibit an initial symptom (which may be sensory) and a serial order of progression; usually followed by weakness and temporary paralysis.

4. The occurrence of focal signs long after or late in the course of a suspected organic lesion should be considered with caution.

5. Focal epilepsy caused by trauma have a tendency to recur and ultimately to become generalized.

6. Surgery, consisting of excision of the so-called epileptic zone is the best method of treatment.

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