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The Pathology and Localization of Disease of the Cerebral Cortex

Thesis for the Degree of Doctor of Medicine, presented by Samuel Mackenzie, M.B., C.M.
It is our purpose here, to consider some of the more important symptoms accompanying disease of the cerebral cortex—chiefly as far as they bear upon the question of localization of the lesion. The localization of cortical disease is the clinical aspect of localization of cortical function. Seeing that pathology must have for its basis physiology, or rather its physiology under altered conditions. The brain is not, not regarded as a single organ, having a single function, but as an aggregation of organs or groups of organs, having distinct and independent functions, one or more of which may be modified by disease, the others remaining intact. What applies to the brain as a part, is considered by the supporter of the localization theory to apply to the cortex as a part. The localization of cortical function implies that these are certain definite regions in the cerebral cortex to which definite functions can be assigned, and the pathological corollary, that the phenomena of cortical disease will vary according to the seat of the lesion.
The question of localization is many sided, and requires for its elucidation the exact seat and extent of the material and palpable changes in the Cortex, and the comparison of the results of clinical observation with experimental facts, and the establishment of the connection between functional or functional disturbances during life and the lesion as revealed by examination after death.

The cerebral Cortex may be affected by many morbid conditions, but yet from the point of view of Symptomatology—which we have here restricted ourselves—the exact morbid character of the lesion is immaterial except in so far as it irritable or destroys the cerebral structure.

We shall then speak of lesions as being irritative or destructive remembering however that an irritative lesion may develop into a destructive one, or at one of the same time may develop into a destructive one, without reference to one part of the Cortex, and irritative to another part.

Again in studying the symptoms accompanying clinical disease, it
will be necessary to distinguish between those symptoms which are modified cortical functions, and those adventitious and accidental symptoms produced by implication of the neighbouring organs and structures. This distinction is very important, since not only does it simplify explanation, but in many cases of cortical disease the most prominent symptoms are those, thus indirectly produced, which when rightly understood, are very useful in assisting us to localize the lesion. Therefore we shall divide the symptoms into two classes:

I. Direct, or those produced by the lesion on the cortex itself.

II. Indirect, comprising those due to implication of the neighbouring structures.
Direct Symptoms of Cortical Disease

In cortical disease, the variability, range, and character of the symptoms, are very striking, especially the seeming disparity between the morbid cause and clinical effects. Often we meet with in life symptoms, grave & disturbing, which we attribute to gross brain disease, yet the autopsy only revealing some minor & trifling lesion. On the contrary, many cases of cortical disease run their course without presenting any well-defined or disturbing phenomena. As far as our knowledge of cerebral physiology & anatomy will permit, we will endeavor to indicate the condition affecting the presence or absence of these phenomena.

The doctrine of cerebral localization is by no means new. Prokopa taught that the brain was the principle organ of mind, that there was a localization of function in the several parts, and in the early part of this century Gall & Spurzheim, Martianista, their celebrated phrenological system, teaching, that the cortical
had no special function, but was simply the matrix of the "nervous areas".

In 1840, Flourens ([10] the first) Scientifically explored into the doctrine of cerebral localization, showing as the result of his experiments, that the brain was simply an organ performing its functions both as a whole and in every part. His conclusions were as follows:

1. On peut rechercher, soit par devant, soit par derrière, soit par en haut, soit par côté, une portion assez étendue des lobes cérébraux, sans que leur fonction s’en perde. Une portion assez petite de ces lobes suffit donc à l’exercice de leurs fonctions.

2. À mesure que ce développement s’opère toute la fonction s’affaiblisse et s’étendra graduellement, et par certaines limites, elles vont être à l’état de les lobes cérébraux donnèrent par tout leur ensemble à l’exercice plein et entier de leurs fonctions.

3. Enfin dit qu’une perception est perdue toute le sort, dès qu’une faculté disparaît, toutes disparaissent. Il n’y a donc point de lieux divers, où pour les diverses facultés, non pour

(1) Recherches Expérimentales (P. 97)
les claires percepts. La faculté de percevoir, de juger, de vouloir une chose résidant dans la même ince que celle d'en percevoir, d'en juger et de vouloir une autre, et par conséquent, cette faculté, en réalité, est une résidant essentiellement dans une seule organe."

(1) Recherche expérimentales.

(2) The doctrine of functional equivalence is once more appearing, with reference at all events to the frontal & occipital lobes. The results of Ferrier & his recent experiments, induce them to conclude "that the post-frontal & pre-frontal regions have essentially the same physiological signification & that portions only of the frontal lobes are sufficient for the exercise of their functions." This would also appear to be the case with the occipital lobes, as far as vision is concerned.
In 1874, Ferrier & Dyce showed that by stimulating the cerebral cortex certain movements arise uniformly, caused by the excitation of certain definite areas. Ferrier, immediately following, not only estimated the excitable area, but also defined a sensory & motor area, & further he divided each of these into distinct and separate tabulae, stimulation of which produced constant and predictable results.

Ferrier's observations have been repeated and in various ways. This is in addition to stimulation by electric current (Ferrier, Yeo, Harley, etc.) & destruction by allatation, injection of arsenic acid or corrosive sublimate, etc. Recently Larborde has carried out a series of experiments, which are worthy of special notice on account of the reproduction of a parasitic condition by a simple arrangement he introduced, first blood, directly coming from an artery, & under the normal premises of the animal's circulation into various regions of the cerebral cortex, so as to produce more or less circumscribed hypophlebitis, & such

...
a manner that an experimental injury of the adjacent part could complicate the results. The brain's symptoms following injury have a remarkable likeness to the clinical symptoms observed in cerebral injury affecting the various parts of the brain, as experiment shows. (1)

From the anatomical structure of connectives of the cerebellum, the theory of localization receiving indirect support. Whilst there is much divergence of opinion as to whether there is a true localization of function in the cerebellum, experiment has firmly established that doctrine with reference to subcortical parts of the cerebellum. - points to which we shall refer later on, more in detail. Suffice it here to say that the so-called motor zone of the cerebellum is in close anatomical relationship with the motor part of the cerebellum, while the posterior parts of the cerebral cortex bear similar connectives with the sensory part of the cerebellum. Hence, in studying the course of relationships of the various parts of brain and spinal cord, states that the pyramidal and fundamental spinothalamic tracts are developed at a later period than the others. (The period of their

(1) Lorenz. "Neve de Fari" 1880
Development, Co. Incides with that of the Cerebral Receptors, that their connection can be traced up into the Cerebral region surrounding the fornix of Rolandic — the seat of the "Motor Zone" — of downwards into the anterior lateral column of spinal cord. (1)

From the clinical data there is a mass of evidence — evidence that is increasing every day — that lesions of certain parts of cerebral cortex are followed by definite symptoms which it would be difficult to explain away by any theory of "fortuitous collocation."

Whilst these clinical observation would appear to support the evidence of motor and sensory centers in the cortex, limited to area and topographically distinct, yet there is much disagreement as to the exact inference, that can be drawn from them; some physiologists thinking that the phenomena adduced to support the doctrine of Cerebral Localization are only valuable in so far as they prove the localization of function in the subjacent white matter of Cerebrum, cerebral peduncles.

From Seckel's to be mentioned among the opponents to the theory. T. W. hi evidence as a Cerebral Physiolo
And a great weight to his objections. Not only does he dispute the general law of the cross action of the cerebral hemispheres, but also adduces experimental evidence to show that "must be in other organs as commonly conceived - that is, as an aggregation of cells, having one of the same function, and which form a more or less definite area, do not exist."

He thus summarizes his views: (1)

1. All the symptoms of organic disease of the brain, when the lesion exists in any part outside the cells, whereas in the last instance the fibre spring which go to form the cerebral nerves, even though they constitute two distinct groups (characterized the one by Cerebrum, the other by Manifetation of an activity) are the effects of an influence exercised on the part more or less distant by an irritation starting from the seat of lesion or its neighborhood.

2. The mechanism of the production of the symptom, characterized essentially by a cessation of activity (such as paralysis, anesthesia, amnesia, aphasia, and loss of consciousness) is identical with that of the arrest of the heart by irritation of the vagus, and consists

(c) Archives de Physiologie. March-April 1877.
to this: let an irritation proceeding from the point asked in the brain propagate itself to the cells whose functions are made to disappear, and produce a more or less complete arrest of their activity.

3. The mechanism of the production of the phenomena which ensue is a manifestation of activity (such as delirium, epileptiform convulsions, echonia, tremors, convulsions, hiccups, not to mention the pain) is essentially the same as when due to peripheral irritation whether of the skin or mucous membrane or any part of a centripetal nerve.

Regarding over central action, it is evident from the case, by quotes, that paralysis may occur or the same site as the central lesion but that rare exception to what was formerly considered to be a general rule, can be accounted for by the variations that Flechsig has shown to take place in the account of degeneration of the fibers at the pyramids. In the case he quoted examined, there was scarcely any degeneration, while, if that brain had been the seat of a paralyzing lesion, the paralysis would have
occurred on the same side as the lesion. That also various manifestations of reflex influence at a distance can be produced by cortical excision is also true; but in Barre's term "widespread experiments, we shall refer to later on.

These symptoms are entirely distinct from those produced by cortical lesion per se. It not only does not oppose the localization theory, but explains away many of the objections that have been brought against it.

In our opinion, then the fact has been clinically and experimentally proved that in the cerebral cortex are two separable zones, one situated on the other, that lesion confined to one will only necessarily cause change in mobility, lesion confined to the other change in flexibility, and that it is possible to diagnose the site of lesion by studying the relative extent of accompanying symptoms. From the localizing point of view, the pathology of cortical lesion would not appear the co-extensive with their physiologic, at all result, the exact extent of these areas, but the "psychic, motor, areas, "sensory areas," or "middle motor center" is immaterial, provided that lesion if
these areas, that sue to deficient symptoms, from which the site clinically may be recognized; which we hold to be the case.
Lesions of Motor Zone.

The motor area includes the convolution surrounding the fissure of Rolando, the ascending frontal at the base of the upper frontal convolution, the ascending parietal and posterior parietal convolution, and the internal surface of the same convolution - the parietal lobule.

In this zone are motor centers for the movement of limbs, head, eyes, the muscles of expression, those of mouth and tongue. The muscles of the leg and foot are situated in the posterior parietal lobule. Those for the arm in the upper third of the ascending frontal, those for the hand and wrist in the ascending parietal, those of the facial muscles in the middle third of the ascending frontal, those for the mouth and tongue in the lower third of ascending frontal of the base of the third frontal, and for the platysma at the lower extremity of the ascending parietal post parietal to the occipital lobule. The posterior third of the upper frontal convolution (corresponding part of second frontal) contains the center for the lateral movement of the head and eye. (1)

(1) Flourens, French dictionary of medicine. p. 246.
A lesion of the motor cortex may involve the whole of the motor zone, or may be only partially. In the one case a hemiplegia will result, in the other a monoplegia. As regards the general destruction of the motor zone, the resulting hemiplegia will closely resemble. That resulting from, say, destruction of the corticospinal tracts of internal capsule. In both of these pareses, we have greater impairment of those movements which are most independent or automatic, whilst those which are automatic, including the 'keyword' associated muscles, escape. Thus, the arm as a whole is more paralyzed than the face & leg, and the movements of the hand more than those of the shoulder.

(1) Though though there is a general solidarity of the whole cerebral-sensory system yet there exists in different animals a great difference in the degree of regeneration of such movements in the lower animals. This is, their relative independence in the higher animals. This is greatest in the frog, & pigeon, and least in the monkey and man. Hence the marked differences which we observe in different animals in the results of destruction of the cerebral hemisphere. Ferrier, "Loculations of Cerebral Disease", p. 22.
But although there is no actually diagnosable feature of hemiplegia resulting from purely cortical disease, yet the accompanying condition in many cases enable us to diagnose it from hemiplegia due to destructive lesions of other parts of the brain. These conditions are those included under the head of diabetic coma state; acute of onset of attack; course and termination of disease; together with certain indirect symptoms produced by reflex action of a diabet.

We know that certain modified conditions have their chief election, that the pathological region of the cortex is different from that of the deeper parts of brain. In this connection, we will only mention the frequency of cerebral embolism in vascular disease of the heart, cerebral haemorrhage in general arteroma, the thickening of cerebral vessels leading to cerebral infarction and haemorrhages associated with Bright's disease. Whilst the liability to haemorrhage in scotches and haemophiliacs must be noticed. Haemorrhages in such rare common in the corpus striatum than on the cortex, although
It is frequently met with there. The nervous structure is also much more liable to emboliene & local softening than other regions. Whilst the centre is much more frequently affected by growth from the adjacent bone and membranes, and also from its position more liable to traumatic injury.

We thus see how the degeneracy of the character of the motor nerve often gives us premunition evidence of its seat.

Mode of onset. It is necessary to remember that the fibre proceeding from the relatively large motor zone, courses and are woven into the nearest space lying between the caudate and pallien part of the caudicular nucleus and subsequently, a hemispherical hindered in extent, which produces a complete destruction of those motor fibres of the internal capsule. Areas of situated in the cortex just affect the motor zone or its extension. In the former case, we should have a hemiplegia sudden in its onset I complete as its character, in the latter a hemiplegia, which continuing might become a hemiplegia, though the hemisphere
...the large and continuous, but which would be more as liberal as deliberate if it were not regarded as an aggregation of hemiplegia. A hemiplegia developing into a hemiplegia is extremely characteristic of cortical disease. The clumping together of the fibers from the cortex into the anterior portion of the lateral capsule, and consequent greater destruction power of lesion in this part, as compared with those nearer the cortex is the dominating factor in the comparative pathology of these parts, whether the vertebrobasilar or the lesions.

Course of Disease. In permanent hemiplegia, due to destructive disease of cortical or basal ganglia, or large if patent even long enough, alternate contraction and rigidity of the paralyzed limbs, accompanied by wasting of parts. Post mortem examination reveals, in some cases, secondary degenerative changes, declining from the point of lesion through the white cornu, medulla into the motor tract of spinal cord. This condition is known as 'late Rigidity' to distinguish it from another group of symptoms, where contraction and rigidity are also prominent...
not occurring earlier in the course of
the disease, and probably due, as we
shall have to show, to reflex irritations
at a distance.

The preceding degenerative changes
only accompany destructive lesions
of the motor cortical zone, or the corona
dorsa of the cerebral spinae artis,
whilst lesions occurring in other
parts of the cerebral hemisphere are
rarely associated with the secondary
degeneration. (1)

These symptoms of late palsy may
be considered as rarely indicating
a destructive lesion. Of course, however,
Charcot, Delia, and Pierre are met with
earlier in hemiplegia, due to cortical
disease, than in that due to disease
of corpus striatum.

A consideration of the above point:

(1) There does not appear to be any atrophy, other
than that produced by want of functional
activity, unless the degenerative process
extends from the motor - cortical columns of
spinal cord, to the anterior large cells of the
cerebellum, bone, or gray matter.
In conjunction with other details, once sufficient to distinguish a paralysis due to cortical disease from a paralysis due to destruction of deeper structure, we have now to consider the effect of partial lesions of the motor cortex.

Partial lesions will declare themselves by localized spasms or localised paralysis, or monoplegia or hemiplegia. We have already drawn attention to the anatomic arrangement whereby hemiplegia are characteristic of cortical destructive disease, whereas hemiplegia of cortical irritation disease. It must however be remembered that partial lesions may induce both general convulsions and hemiplegia. Thus, in traumatic lesions we often find that the area of functional disturbance is not co-extensive with the area of anatomic lesion but, on the contrary, much more widely distributed. This is to be accounted for by a general suspension of the function of the cortex due to Suddenness of the injury, and the like, with movements which have been functionally suspended reappear, whilst more areas destroyed by the lesion.
will be accompanied by paralysis of a permanent character of the corresponding muscles. We have seen that a monoplegia developing into a hemiplegia is strongly presumptive of its cortical origin; so now we can add, that a hemiplegia dissociating itself into one or more monoplegias is also characteristic of cortical disease.

Clinical experience shows that there is a striking similarity of arrangement of the motor area of man to that which obtains in the case of the monkey as experimentally determined by Ferrier. Many extensive analyses of records have been made, among which may be mentioned those of Charest & Phil, Boudin, Maragles, V. Lecan & Champmione. These incontrovertibly support the localization theory, "and although we cannot be quite certain of the position or extent of a cortical lesion causing a sudden hemiplegia, we may take a monoplegia of the arm or of the leg, or of the arm and leg, as an indication of lesion of the upper extremity of the ascending convolution, close to the longitudinal fissure; cerebral..."
Monoplegia as a sign of lesion of the upper part of the ascending frontal convolution, or, if the paralysis affects the hand, more particularly of the ascending parietal convolution's back, facial monoplegia, as indicating lesion of the area front-parietal region, while facial and lingual monoplegia or their combined with aphasia indicates lesion of the lower part of the ascending frontal convolution where the third frontal center is. When local irritation of the motor zone with rise in tone of movements having the associated distribution of the symptoms results in a monoplegia, resulting from local destruction of the motor zone.

Any pathological condition producing hyperaemia or irritation of the cortex may produce these movements. The seat of the lesion may be ascertained by the same rule, which we have indicated in the localization of destruction lesions.

A lesion at first irritating may develop into a destructive one gradually implicating the whole of the motor zone giving rise to a monoplegia passing into a monoplegia.
which in turn becomes a hemiplegia of a permanent character. But an
irritating local lesion may also give rise to a hemiplegia of a
temporary character, where the
local lesion causes a general
discharge from the motor nerve, I
followed by a paralysis (according to
Weghlandt Jackson) of these areas—
a paralysis of extenuation and of
destruction. Consequently temporary
and not permanent.
This paralysis is then in post-epileptic
paralysis.
Lesion of Sensory Zone

This zone according to Terris is placed immediately behind the motor zone and is believed by him experimentally defined areas for the reception of various sensory impressions.

The right centre is situated in the angular gyrus, and embraces the occipital lobe — the occipito-angular region. The centre of hearing is localized in the superior temporal sphenoidal convolution; the tactile centre in the hippocampal region. While the centres of smell and taste are situated together at the lower part of the temporal sphenoidal lobe.

But although Terris thinks that he has never experimentally proved the existence of sensory areas in the cerebral cortex, it must be confessed that clinical experience, if not actually opposed to the theory of cortical sensory localization, does not afford anything like the strong confirmation support as in the case of motor localization. The want of clinical evidence to support it to be accounted for in some measure by the great rarity of the pathological conditions required.
for it would appear, that unilateral lesion of the sensory zone do not 
permanently abolish sensation on the 
opposite side of the body, this only being 
produced by bilateral lesion. & 
Consequently, the pathological sequences 
of lesion to the sensory zone are greatly 
less than those resultant in the motor area.
Another difficulty is the nature of 
the inference to be drawn from the 
symptoms attending such lesion of 
the sensory zone. But although or 
yes, there is no clear and indisputable 
evidence that a purely cortical lesion 
has caused any well ascertainued 
changes in any of the special senses, 
yet there is evidence of an indirect 
feature—a part from the experimental 
evidence of Ferrier & others—giving 
the prime reasons for believing in this 
sensory localization.
Nearer & frequent I am shown by 
their anatomical direction that 
in the monkey there is a partly 
detached group of fibers passing 
from the hippocampal and posterior 
part of the lateral capsule, lying 
between the optic thalamus & lateral 
nerve, & spreading out in the
Adducial fibres of the occipital, lobe & finally terminating in the grey matter of that lobe. Furthermore Meynert believes he has traced these fibres downwards through the crus, pons, medulla & thence into the posterior columns of spinal cord. In this part of the internal capsule, are also fibres passing from the corpora quadrigemina & medulla Oblongata, connected with the optic tracts. Meynert & Rendu have proved that section of this bundle of fibres, forming the posterior part of the internal capsule, is always attended by hemi-anesthesia on the side opposite to the lesion. Sarny & others have shown that lesions of this part of the brain are also always attended by hemi-anesthesia of the opposite side. For these reasons then, it would appear that the occipital centre was interested in the reception of sensory impressions. I am the unconscious phenomenon and angular region of the centre. Some physiologists, whilst admitting, are distributed to the differentiated areas in the entire body that the sensory fibres are spread out indiscriminately over the whole of the
Tactile, Olfactory, Occipital, and Temporal regions.

In the present state of our knowledge, Newell would seem to indicate that the posterior region of the cerebral cortex receive the centripetal impressions, whether these impressions are further localized is a question which must still be considered as sub judice. The change that occurs in the sense of sight in cerebral lesions, have received much attention, and are worthy of further study.

The results of ferrier's first series of experiments enabled him to locate the visual centre in the angular gyrus, derivations of which are observed would induce blindness in the opposite eye. It was thought that cerebral lesions affecting the integrity of the optic tract would produce hemianopia, or loss of sight of the corresponding half of the visual field, on the side opposite the lesion. Yet cerebral lesions themselves would only produce crossed hemianopia. To account for this, Charcot advanced his hypothesis of compensatory decennation in the brain, whereby the decerebration of the optic fibers as the optic commissures were completed at the brain, so
that the posterior part of the brain was
related functionally only with the eye
of the opposite side. But recent
experiments have shown that not only
may both occipital gyri be destroyed
without permanent loss of sight,
but there are several well recorded
cases where hemianopia resulted from
purely central disease (1)
Marcus places the visual centre in the
occipital lobe, and he states that
visual lesions will produce
paralysis in both eyes on the same
side as the lesion.
Leclercq & Tauriac place the visual
centre in the angular gyri, but state
that destructive lesion will cause
bilateral hemianopia & not crossed
anamnesis. In a more recent
series of experiments, Tauriac and his
found that both unilateral and
bilateral destruction of angular gyri
caused only temporary loss of sight
in both cases, crossed amblyopia
occurring that permanent complete
blindness could only be obtained
by destruction of both occipital gyri
and occipital lobes on both sides (2)

(1) Marcus, Cerebri, 3d. Edition, 1878
(2) Le papier sur Cerebral Amblyopia & Hemianopia,
Berlin, January 1887
From these facts Ferrier concludes that: "There is a twofold relation between the eye and the cortical visual centre. The one mainly cross - the central portion of retina probably bilaterally represented by the angular gyrus; the other bilateral - the corresponding side of both retina being represented by the occipital lobe, the temporal, and in conjunction with the angular gyrus.

He also suggests that when a hemianopia the loss in the visual field extends quite up to the fixation point: affecting central vision, the lesion is probably somewhere between the optic chiasma and the corpus geniculatum, but when there is a free area around the fixation point then it is probably central (1).

As regards the centres for hearing, touch, smell and taste, we have already indicated their position as determined by Ferrier; whether clinical evidence will confirm the theory of Deacon, localisation must be left for the future. At present

(1) Ferrier, op. cit.
The evidence at our disposal is both insufficient and contradictory.
It may however be stated that the sense of hearing, unlike the sense of taste, would appear to be more bilaterally associated than that of common sensation. Hence suffers less severely in unilateral lesions.
We have now to consider the effects of lesions of these parts of cerebral cortex not usually comprised within the motor or sensory areas. These regions are more akin to the cerebral zone of Broca and Wernicke's area, or the prefrontal and occipital lobes. Hughlings-Jackson believes that these parts are also motor or sensory respectively, being the seat of higher centres, which represent the various in more complex articulation what the "middle" motor or sensory centres (by which terms he designates the motor or sensory zone of Broca) represent in a less complex and more organized manner. He also considers that these higher centres constitute the physical basis of consciousness, that there is a destruction of these areas will cause a paralysis of consciousness (if we may use the term) the mental symptoms thus resulting varying as to the extent of the destruction and its site. Therefore, this theory suggests that an excitatory explosive discharge of the higher motor cells in the prefrontal region produce an epileptic seizure, whilst epilepsia partialis atletica...

(1) Demonin lecture, 1887.
without loss of consciousness, depend upon bilateral discharge from the "middle motor centers." Passing from theory to experimental fact, we find that electrical stimulation of the praecentral region produces no motor phenomena, and destruction lesions of this part are followed by no special physiological result. In speaking of the effect of Central lesions on vision, we saw that Calci's investigations pointed to the absence of some functional equivalence of the occipital lobe. I have acceded communication to the Royal Society.

Lesions of the frontal region vary according as they affect the postfrontal or praecentral region. Lesions of the praecentral region alone do not produce any discernible physiological symptoms. Lesions of postfrontal region cause temporary paralysis of the lateral movements of head and eyes. As the symptoms are only temporary, so long as any portion of the frontal lobe remains intact, it is concluded that the postfrontal and praecentral regions form essentially the same physiological configuration, and that portion only of the frontal lobe is sufficient for the exercise of their function.


April, 1884.
By Ferrier and Gou, it would appear that the formerly held view that the two divisions of the frontal lobe, which he now considers as physiologically equivalent, there are numerous recorded cases of destructive disease confined to these divisions of the brain, complicating one or both sides, unattended by any motor or sensory phenomena, usually however somewhat degree of intellectual impairment of the intellectual faculties. It is thought that there is a difference in the quality of the mental alienation in frontal and occipital disease, that while disease confined to the frontal lobe produces mental disturbance, one allied to dementia. On the other hand, when situated in the occipital lobe, the symptoms are more allied to ideational and illusionary spectra etc. It is possible that in these cases of epilepsy, there occur in the sensory aura, such as flashes of light, sudden noises, etc. So frequently there
Observed, that had been to do with some irritating action of the central sensory centres, while these are not unaccompanied original in the auditory central centres. The various eye symptoms due to general demence or, as has been suggested, may also possibly be dependent upon irritation of these sensory centres of allied condition of the superficial membranes.
II. Indirect Symptoms Accompanying Critical Lesion.

We have seen in the preceding section that the symptoms of critical disease vary, according to the extent and nature of the lesion. In turn, we can often localize its site by studying the nature of the resulting symptoms. The cerebral cortex being concerned with motility and sensibility, it also being the anatomical substratum of consciousness, disease of this part of the brain modifies in a more or less definite manner these functions. But in critical disease, we have to deal with other phenomena—symptoms as prominent and often calling for more urgent treatment than those we have as yet considered. Although these symptoms are real essential phenomena of critical disease, yet as we hope to show, they are very important from the point of view of localization. Whilst the degree of impairment of cortical function, often enables us to judge what specific part of the cortex is affected, these indirect symptoms enable us to determine the broader question.
As to whether the disease is cortical or central. Cases of intercerebral diseas may occur their whole course without any change of mentality or sensibility, with no very evident mental impairment, and it is in these cases, especially that these indirect symptoms are valuable in assisting us to localize the disease. These symptoms thus produced are headache, vomiting, spasm, or convulsion, with one or more of pulse, respiration, or general temperature, while each of these are met with in the course of other diseases, yet when associated with cerebral diseases, they present special character, indication of their source. Till comparatively recently they were vaguely supposed to be due to increase of intracranial pressure, which, with equally vague ideas of cause, motor disturbances have been so usual for so long. Stued as convulsions expression whereby to conceal our ignorance of central physiology. Now, owing more particularly to the researches of Durel and others of the French School
of neurology, our ideas about the role of central pressure in brain disease are much more defined and exact, and it has to a large extent lost the importance with which it was formerly endowed.

As regards increase of vascular pressure, M'Connon (1) maintains that it never causes symptoms except perhaps in death from strangulation and in sleepy tetanus, whooping cough, and other conditions where probably the venous engorgement is as great or even more, with its distressing symptoms. Concerning increased intracranial pressure we rarely if ever see.

In speaking of cerebral hallucination, Hutchinson (2) says that the idea that "slurred, breathing, laboured pulse and a suffused and sticky countenance are symptoms of compression is probable, a mistake," and a group more frequently declare laceration or cerebral excavation.

Ophthalmologists are doubting whether the pressure theory of Gowers can amount.

(2) Second dictionary of medicine, 1806.
one supplied with true sensory nerves, although it has been suggested, that the sensory motor nerves of the other membranes may possibly under altered circumstances become sensory. The sensory nerves of the dura mater are derived from the fifth pair, the majority of the fibers arising from near the ganglion ganglion of the terminals and organs situated nearer the liver than the plica septi to the nerve. It has been shown that stimulation of the central end of a divided sensory nerve, besides producing the sensation of pain, may, if long continued, produce reflex convulsions, and also reflex inhibition of the heart. What is true in this respect of different nerves in general would appear to be especially true of the sensory nerves of the dura mater.

Duret (1) has shown experimentally that the sensory nerves of dura mater are sufficiently excitable, and that irritation of the membrane gives rise to pain, hyperesthesia, neuralgia in the reflex species of contractions of the muscles of organic & animal life.

(1) Études experimentales sur les mammiferes, C. d'Arv. 1878.
Any cerebral lesion, then producing tension or inflammation of the dura mater, will tend to produce the reflex phenomena to tension the aqueous humour and decrease the pain. With a tendency towards inflation of the dura mater, besides any condition which will cause a stretching of membrane, in meningitis, cortical inflammation, so common in syphilis, disease of the brain, we have the characteristic feature headache, probably due in the first place to the inflammatory exudate of the meningeal afterwheels of tension caused by the collection of inflammatory products.

In addition, we may have the important function of the bulbomedullary region affected, causing characteristic symptoms. These can now we only be directly affected by those lesions which increase the intraocular pressure to a material extent. The water-bed formed by the cerebrospinal fluid distributes the pressure, whilst the change in size of the venous sinuses and the elasticity of the vertebral dural ligaments affect an increase in the accommodative compensating to a certain extent any
increased demand on the cranial space. When this limit known to
be exceeded, say by a large and rapidly
increasing tumour, then we shall have
symptoms of compression resulting,
in addition to the local symptoms,
produced by modification of the function
of cerebrospinal fluid at the point where the pressure
is directly applied.
Duret has thus summarized the results
of his experiments as to the effects of
"a slight pressure on the intercranial
pressure is not nearly modified, the
atmosphere of some of the cerebro-
spinal fluid, the extensibility of the
vertebral ligaments, and of the cranial
sinuses, all the sooner being made.
There are no general nervous
disturbance,
with medium pressure the compressed
vessel admits of the blood into the neuro-
membrane and there is therefore Ischaemia
more or less pronounced of those
important parts. These result cerebral
disturbance, viz. somnolence, fatigue
or muscular atrophy of the
loss of sensibility, ataxic disturbance,
by slow pulse, Cephalic pressure &

In Brussels 1878. April.
lowering of temperature, and medullary disturbance, or diminution of reflex excitability, loss of vascular tonus.

3. With a high degree of pressure the circulation in the active circle is almost completely stopped. The ensuing coma or sleep, from cerebral disorder, necessarily slow pulse, and labored or stertorous respiration, with prostration and considerable fall of temperature. From affective or fatal, and lastly complete suppression of the medullary functions shown in abolition of reflex excitability and of vascular and muscular tone.

IV. When the pressure exceeds the arterial tension, there is absolute arrest of circulation and death.

With these experimental data we can now consider the various symptoms more in detail, especially in regard to their localizing degenerations.
Pain \& Vomiting

Many cases of intercranial disease give rise neither to headache nor vomiting. Contrasting greatly with these cases, whose both symptoms are very marked and distressing, we will consider them together since they are so often met with in association. As we shall show, a determinate relationship. A priori, it would be supposed that, while the majority of cerebral diseases would be accompanied by cephalalgia, not in virtue of any sensibility of the cortex, but from the near vicinity of the highly sensitive membranes, or the meninges themselves, more directly placed, and consequently less likely to implicate the meninges, headache would not be nearly so common. Clinical facts give ample confirmation to this view. In all our text books severe cephalalgia is given as an important point to be noticed in the diagnosis of meningitis from cerebral disease.

Ferrier believed that the nearer the disease approaches the meninges, the greater the tendency to pain (1). Also Callender on the other hand, in analysing 91 cases of cerebral disease, states that

pain was absent in all, where the central ganglia alone were the seat of disease. But pain in the head is often clinically found associated with vomiting. The act of vomiting is a reflex act for which the simultaneous action of three sets of muscles are required, viz. the diaphragm, & abdominal muscles, and the longitudinal fibres of the stomach. The movement of these muscles being co-ordinated by a centre situated at the floor of the 4th ventricle. The effector nerves concerned in this reflex act are the phrenic, intercostal & pneumogastric, whilst in the most simple act of vomiting as that produced by the presence of some irritant in the stomach, the pneumogastric is the effector nerve, yet any sensory nerve would appear to be able to convey impressions producing an discharge of the vomiting centre. Vomiting also appears to be specially induced by stimuli passing by cutaneous nerves such as vasi skeletal, osseous & utricular nerves. Ferrier supposes that the brain in the cases a condition of 'distension' such as has been shown to obtain in other reflex cases, the reflex act consequent on
irritation of any particular spot, is at first very a less definite or limited in reference to the source of excitation, but if irritation is strong and more continuous, there is a tendency then to radiation or discharge of movement (1). Knowing the contiguity of the nuclei of the 5th nerve to the vomiting centre one should unhesitatingly fast to the question that any severe pain can produce after vomiting, such as that produced by a blow on the head, or a blow on the stomach. The headache then in cerebral disease is often of a peculiar, intense, character and consequently occurring by analogy, we should expect often to find vomiting associated with it — which conventional clinical evidence appears to have established. It is now recognised that vomiting is especially met with in acute cerebral disease, when headache is also accompanied as a symptom, and that therefore pain and vomiting are symptoms which when associated, point strongly towards cerebral complication.

But there are numerous cases of brain disease, where vomiting is unaccompanied by cephalalgia. It may be induced by the reasons dependent upon a disturbance of the sense of equilibrium seen in cases of Encephalitis, Meningitis disease, and also in those undefined conditions, where there is some disturbance of vasomotor arrangement, whereby vertigo is produced. But in addition to the vertigo a reflex irritation of the vomiting centre, the latter have a directly affect by increase to the intracranial pressure—say by a tumour—giving rise to anoxemia of the centre in the bulb medullary region as shown by Durand. 

Thus, any cortical lesion which increase the intracranial pressure enough to affect the medullary centre, will be liable to be accompanied by anoxemia, so that in dealing with vomiting met with in cortical lesions, it is necessary to recognize two varieties:

1) That produced by irritation of a distance of the vomiting centre by irritant of the outer, medullary bands of the 5th nerve. Independent of intracranial pressure

2) That produced by cerebral pressure giving rise to anoxemia of the bulb.

(c) of civ.
medullary centre, which may accompany any united intracranial condition, cerebral or central, which is capable of materially increasing the intracranial pressure.

In both these cases the vomiting centre is disturbed, in the first case the stimuli irradiating into the vomiting centre, or in other words a constant source of irritation giving rise to periodic discharge from the vomiting centre. This may be explained by Lewis "Law of Discharge of a summation of stimuli" viz. that "stimulation which falls short of actual discharge of a nerve centre increase the tension of the nerve after a certain accumulation of stimuli sudden discharge is readily induced." In the second case anaemia of the whole bulb-medullary region takes place and irritable state of the centres induced analogous to the general "convulsion" seen in many cases of general anaemia.

Lauder Brunton (1) has shown that a syncope of shock from cerebral commissure where vomiting is very common there is a rare word paralysing of the

(1) Physiological Basis of Mind, p. 240.
Industrial vessel, or consequent accumula-
tion of blood there, determining a general
aneurisma of the rest of the body, including
the cerebral centre. [1] Recent research
would also tend to show that the vas-
cular centre in the medulla is specially
related to intestinal blood vessels, &
Hence we see how any morbid condition
giving pain & elevation intracranial
pressure, & hence aneurisma of the skull
would again reaching fluid to increase
the bulbous aneurisma by its action on this
arrangement.

The diagnosis of these varieties of cerebral
vomiting will depend upon the
diagnosis of the site of the lesion
— whether cortical or central. [2] Also
upon the pathological diagnosis as
to whether it is a disease likely to
increase the intracranial pressure.
In the first or reflex kinds we shall
have it associated with pain at the
head, and probably also will
reflex spasm & convulsions, whereas
primary pain will be absent in the
second or aneurisma variety.
This latter will be found usually in
cases of gross cerebral disease or the

vomiting will be accompanied by other 
symptoms of bulbus-compression - as 
labourious pulse, stertorous breathing and fall of temperature. It is to be 
remembered that we may have the 
two varieties combined, especially 
in cases of tumours growing from 
the meninges, where at the earlier 
stage, when the tumour is not of 
sufficient bulk to produce the 
pressure symptoms, we shall have 
the reflex vomiting and pain, which 
will be modified in the later stage 
by the appearance of bulbus-medullary 
symptoms, when the tumour reaches 
a sufficient size.
Modification of Pulse.

In cerebral disease, we often see the pulse profoundly altered. The inhibitory center in medulla being affected in identically the same manner as the adjacent vomiting center. Richter (1) has shown that stimulation of aortic and carotid arteries in anaesthetised animals is capable of producing reflex inhibition of the heart. And Oudel (2) has shown that slowing of pulse rate invariably results when the arterial pulse pressure increases beyond a certain limit.

Clinically we see in this two conditions reproduced in the earlier stages of acute meningitis we have a marked slowing of pulse—a reflex inhibition—while in cerebral haemorrhage the carotid pulse and alteration of breathing are symptoms of direct medullary compression.

The cardiac inhibitory centre thus being affected by the same condition as the vomiting centre, it is unnecessary to repeat in this connection what has been said in the previous section, a few of vomiting.

(1) "Arch.," Dec. 16, 1871, p. 483.
(2) op. cit.
Reflex Spasms and Contractions. Long continued exposure to constant stimulus of a sensory nerve will produce general contraction and spasm, as will stimulation of any part of the sensory path of the cerebral spinal system. Here we are more concerned with sensory nerves of the face, head, and the special role they have in the production of these reflex phenomena. The contractions thus occurring, may be unilateral or, generally speaking, they are more near the localized character, as those produced by cutting of the motor tracts, in the cerebral cortex. Ferrier points out, that in these reflex spasms, we may have the explanation of those otherwise automobiles cases where convulsions exist on the same side as the cerebral lesion and also many of those cases collected by Pierre Diderot i Cited by him in opposition to the localization theory, belong to this category.

We have before alluded to the phenomenon of cafe' spasm, but of which we shall attribute to reflex spasm, but whilst affording an ample & satisfactory explanation of the early variety, that it can account completely for the later symptom appears more than doubtful.
Clinically these two varieties are quite distinct, the one being intermittent, often passing completely away—especially during sleep, whilst this latter is permanent; of rare occurrence amongst Chancellors of the Long Parliament, and standing always associated with secondary degeneration according to the wort fielch. Associated with headache, combing of fingers of pulse, these phenomena would point strongly to cortical disease. Chancellor has pointed out that lesions deeply placed, as in the thalamus and other parts of the brain, are rarely if ever followed by early rigidity, and also that late rigidity comes on earlier in cortical disease. Thus it becomes once more centrally placed. If these views be correct, early rigidity might occur in initial lesions of any part of the cortex, whilst the late rigidity would only follow destructive lesions complicating the cortical zone.
We have thus gone over some of the more important points of use in localizing cortical disease. It is true that we might also have considered other motoric conditions accompanying or produced by cortical disease, such as spastic paralysis, or the various affections of the cranial nerves, but the former, for localizing purposes is unimportant and the latter are more especially suited to localizing disease affecting other parts of the brain. Other than the cortex it is not our intention to consider in any detail the treatment of cortical disease, although it may be allowed to predict that both the trephine and knife will be used much more freely than frequently in the future.

If, as we hold, these induced symptoms of cortical disease are reflex phenomena, then we have a potential meathow at our disposal for combating these. Every reflex act however complicated may theoretically be resolved into three stimuli, viz. an afferent nerve, a nervous cell (cella) and an efferent nerve, and we are acquainted with drugs which, by acting on one or other of the elements, forming the reflex circuit, thereby modify the reflex act.
Thus in the local anaesthesia produced by the topical application of cold, or of cocaine we paralyse the afferent element. Carara on the other hand acts only on the afferent afferent element, while the central nerve element can be excited by drugs like strychnia, or depressed by others.

Such as Pilocarpine of Potassium, direct, artificially excites muscular and prevents the appearance of reflex convulsion by excising the afferent motor, thus removing the afferent (sensory) element. Similarly the drug Gual has reflex phenomena, often so disturbing both to the patient and physician, - either by trying to paralyse the sensory nerve of the afferent motor, or the sensory afferent themselves. For the former purpose we know that certon chloral appears to have a sedative action on the 5th nerve, and the special call for this drug in cases of severe headache with vomiting, & other symptoms pointing to meningitis, cerebral disease is very evident. Again in attacking the central nervous system we have a specially useful drug in Pilocarpine of Potassium. Laborde has shown that a frog placed under the influence
of Potassium, low the reflex excitability of the cord, shown by the absence of reaction on pricking the leg, and in the human subject the effect of large dose of Potassium in the absence of nausea and vomiting, where the drug has been given for laryngoscopic purposes. This latter fact is extremely suggestive, in the treatment of vomiting of a cerebral nature.

These two indications would be sufficient to indicate on what principle the rational treatment of these reflex symptoms accompanying cerebral disease, should be based.
Appendix of Clinical Cases
Pickenby John. 16. Stage: Stabbed to the Heart  
Admitted to the Hartford Public Hospital  
January 7, 1884. 
Complaining of weakness in left arm & leg.  

History:  
Three weeks ago, whilst walking at Mason's 
Lake, his horse fell. Pickenby being 
thrown heavily to the ground & striking 
his head against a large stone.  
He was picked up in an unconscious 
condition & carried home where he was 
treated by an officer of police. He 
remained unconscious for four days  
and when he came to himself found 
he had a large wound on the right 
side of the head, and in addition 
he noticed that his left arm & leg 
were to weak that the former was 
almost useless if that he was unable 
to support himself on the latter without 
the aid of a stick. He remained 
under treatment for the next fortnight 
and got a little stronger. There was no 
alteration in the sensibility of the 
left side, with the exception of a slight 
numbness.  
His present health has been good.  
No recent disease.  

Physical Condition  
He is a well developed, muscular &
Health looking well. Temperature under both axillae 37°. Skin cool, free from eruptions or blisters.

On the right side of head is a large scar corresponding with a depression of the cranial bone in extent. This scar begins in the median sagittal line about a centimetre in front of an imaginary vertical plane passing through both auditory meatus, 5 centimetres outward, downward and backward for 7 centimetres, terminating about 2 centimetres behind the said imaginary plane. For the whole length of this scar there is a sensory depression in the parietal bone. Palpation over this region causes no pain.

Nervous System

Motor: There is a well-marked weakness of left arm & leg. In left hand dynamometer reports 20 lbs. in right 98 lbs. He can perform all the movements of the arm & forearm in a slow & deliberate manner, but he finds considerable difficulty in performing the arm complex and co-ordinated movements of the hand. Cannot dress himself without assistance. He can walk slowly, both
the aid of a stick., but there is a
well marked dragging of the left
leg, and he is unable to rest much
weight upon it. He also tends to
roll over towards his left side.
The other movements of body are normal.
There is no affection of the muscles
of the face or tongue.

General & special Sensibility
unimpaired.

He is an intelligent lad, with fairly
good memory. He has no headache or
vertigo. He laps & eats well.
The organic reflexes are normal,
slight relaxation of tendo-flexor in
left great toe. No ankle clonus.
Plantar reflexes present & equal on
both sides.

Progress
He was ordered 30 gr. daily of Iodide
of Potassium together with Constant
Current v douches, and made good
& decided progress during his
three weeks residence in Hospital.
On discharge he was able to walk
without assistance, albeit there was
a little dragging of leg & a tendency
to roll over towards the left side.
There was also relative weakness in the
three jpd on left side.

Dyspraxometria in right hand reported 96
in left hand 43. th, he was able
to use his hand much more freely
than on admittance.

Remarks

The scar V depression in the parietal
bone, would correspond to the upper
part of ascending frontal convolution
V ascending parietal convolution,
as seen on referring to the following
diagram.
In both these convolutions, Ferrier has determined motor centres, one in the upper part of fissure of Rolando (1, 3, 4), for the movement of leg, arm, and trunk, as observed in swimming, the other in the ascending parietal convolution (a, b, c, d) for the individual and combined movements of fingers and wrist. From the character of the wound, these parts of the cortex must have been removed, especially the parietal convolution, and the symptoms observed would generally support Ferrier's conclusion.
Oakes, Alexander, 20 Single, Clerk. Admitted Jan 18, 1884, to the Shelton British Hospital, with the following history.

In the course of the preceding day he struck his head against the sharp corner of a projecting beam. Feeling somewhat stunned, he stood for a few moments. Then attempted to walk to a neighboring room, but immediately lost consciousness. On coming to literally about half an hour later, he had great headache, fast same time felt sick, giddy, 

He also noticed a peculiar weakness ofremembering of left arm, which did not appear to belong to him. He was able to walk, a little unsteadily, however, owing to dizziness, but noticed no special weakness of leg. He was told that he had had a fit. A.J. a friend, who was with him at the time of accident, stated that he became very pale, then violent and general convulsion supervened. lasting for several minutes. Then he became quiet, remaining conscious, for nearly half an hour.

He then came but suffered for the rest of the day, headache, dizziness, 

I sought admission to hospital until morning.
M. Condelem

He is a strong looking well developed
man, recently arrived from Australia.
Skin moist & cool. Temp. 36.8.
B.P. 82. Pulse strong & regular.

Nervous System

Mentally he is an intelligent man, o
memory good. He has no headaches
or dizziness, & sleeps fairly well the
previous night. He complains of
sensation of weakness in left hand, the
right of which is perceptibly weaker
than that of the right. Palpitation
regular, 110 right, 52 left. There is
also evident difficulty in executing
the usual movements of the hand,
seen well by the examiner between ri
which he attempts to button his coat.
He can walk perfectly well & there
is no apparent muscular weakness.

 Reflexes

Right reflexes exaggerated
of the knee jerk on right side, otherwise
no abnormality.

General and Special Sensibility

Unimpaired.

The other systems normal.

 Urine 1022 S.G. Dark yellow, depending
Locally no albumen. No cicatrices or pldris.

On the skull is one superficial linear wound, the superior and posterior extremity of which is about 3 centimetres above the advanced edge of the parietal prominence extending forwards & slightly downwards for 7 centimetres; this extremity being bounded by a vertical plane passing through both anterior meatus.

Remark. This man remained in hospital for 4 days, during which time there was no convulsive attack. He slept & ate well & altho' has a cheerful demeanour. On the third day, the 2nd use of the left hand appeared to have fully returned & he could move it about quite as freely as usual, before the illness. It may be here stated that he has never suffered from any recurrence of独一, or his family history is free from epilepsy, insanity, or habit, are regular & free from either alcohol or sexual excess.

Remark. The convulsive attack occurring immediately after the injury.
to the head, in a sense, whose personal or family history was hitherto free from epilepsy, makes it reasonable for us to suppose it be directly due to such injury. From the appended diagram it will be seen that the wound lay immediately over the ascending parietal convolution, where it will be remembered Ferri's place, the motor centre for the finer movements of the hand, which in this case were the only ones impaired. As to the nature of the lesion, the transverse character of the break would negative the supposition of any obvious injury to the cortex; probably a smooth cushion of a small uncapped vessel bruising compression, perhaps accompanied by calcification.
Clark J. W. 29. M. Composition admitted into Hartford Hospital December 17, 1884.

Complain of agonizing pain in the head, accompanied by vomiting and vertigo.

History.

Seven months ago, immediately after a driving bout, he suffered from severe headache which lasted for about a week during which time he was obliged to leave off work, walking to degree, which accompanied the pain. The pain off & he remained in his usual state of health for about three weeks when he again had to cease work on account of the recurrence of the headache and vertigo, this time accompanied by vomiting. At the end of several days he was able to resume work, but noticed that his eyesight was failing, causing him to make many mistakes in setting up the type, accident which before seldom occurred. Since then he has never been free from headache, with occasional fits of giddiness & vomiting. For a week previous to admission, the headache has been of a much more violent character, he has vomited several times daily, and two days ago he had a couple of "fits." On questioning his wife
Who was present, it appears he was seized with general convulsions, fancied at the mouth, and remained unconscious for about 10 minutes. He had an exactly similar seizure half-an-hour afterwards. She did not notice if any part of the body was convulsed more than another. She also states the during the past two or three months, her husband’s character and disposition have entirely changed—from being a kind & affectionate husband, he has become childish, changeable & irritable, and on several occasions recently has struck her, which he had never done previously.

Present history. Three years ago had a chill, followed by sore throat & loss of hair, for which he was under medical treatment for about 6 months when he married. He has one child, which he says is very healthy. Before marriage he drank rather heavily, but has been on the whole steady since.

Family history. Free from epilepsy, insanity or epilepsy.

Present Condition

Man of moderate height, in fair condition but pale & unhealthy looking. The skin is free from freckles, and there are no nodular growths on the body. Temp. 36°8
Nervous System.

Mutilly, he is childish and fickle, smiling in a weak manner & his conversation is interrupted by meaningless laughter. Memory is much impaired, and he finds considerable difficulty in choosing and finding the words to express his meaning. Says he cannot concentrate his attention as formerly & finds it impossible to read now. Even when free from pain, partly from that & also from feeling eight. He complains of constant pain in front part of the head which becomes at times to excruciatingly severe to be bearable. Pain is elicited on palpating over the left temporal region. Between the attacks of headache he lies in a quiet condition, to be helped fairly well. On making any movement and often when lying quite still he suffers from dizziness, the whole room appearing to whirl rapidly around him.

There is no modification of motion or general sensibility. The plantar reflexes are absent on both sides. Tendon reflexes are well marked & equal on both sides. Achilles reflex not obtainable, whilst organic reflexes are normal.
Special Status

Vision: On attempting to read, all the letters become indistinct - "convexity," as he terms it. He has no ocular pain. Vision is equally indistinct in both eyes.

Hydroscopic examination of both eyes reveals the edges of the undefined, blurry image to be circular; in right eye, the blurring of disc is less, but there is a small hæmatoma on the temporal side.

The other special parts are unchanged.

Digestive System

Teeth bad, tongue clean but flabby & pale, appetite poor. Vomited constipated.

He has had severe attacks of diarrhea. He has no pain in the body, nor does physical examination reveal any marked condition of the viscera.

Until 10:20 a.m. 30 T.p. in 24 hrs. faintly acid, albumin, no deposit. Plenty free urine albumen a sugar. On the plus, pernici in an old eeltrix.

Progress: This man was ordered antisypthatic morphia, and during the first week little or no improvement was observed. His fever & chlorotic disposition gave no
Some trouble arose in carrying out the treatment. The pain in the head remained constant, whilst during the paroxysms he rolled about in bed, shrieking, with violent times. On an average, he convulsed three

daily. The temperature never rose above 37° whilst the pulse varied between 50-55

regular but small & weak. After the

first week however improvement was apparent, the head ache less constant

and paroxysms fewer and less intense. At the end of a fortnight, he was able to walk about the ward, without suffering to any marked extent from dizziness which previously had absolutely

prevented him from leaving the bed. At the end of the month's residence

the headache, dizziness & vomiting had quite disappeared, whilst great

improvement had taken place in vision. He could now read, without discomfort &

and ophthalmic examination revealed a great change in the condition of the eyes, which

now, with exception of little blurring of detail outline, were normal.

He was discharged on Jan 20. 1873.

He reported himself on March 6, 1874, when

the improvement in his general appearance

was most striking, he was being
about 4 about looking to his excellent health, he has had no recurrence of the symptoms I can do his work in the printing as easily and efficiently as ever he could. In addition his wife expresses he being approval of the anchor or his disposition which is quite as striking as that of his physical health.

Remarks
In this case the previous history. The course & anchor of the symptoms under anti-syphilitic treatment showed that might probably be had to do with syphilis. The urinary pain with the frequent vomiting & dyspepsia, the absence of motor & sensory changes, and the evidences of depression in the mental state & in the left frontal lobe indicated that the pre-frontal region of left hemisphere was the part involved. The two bursts occurring in this man whose previous history, as well as that of his family, are interesting. They may have been reflex convulsions due to an irritation of the membrane or the motor zone of the cortex may have been irritated by irradiation.
from the centre of circulation. The accompanying loss of consciousness would tend to support the latter hypothesis.
Gilbert Thack, 21.5, Stablesman
admitted to the Hereford Union Hospital
Feb 22, 1884.

Complaints: constant pain in head, with vomiting
and vertigo.

History of Present Attack:
First complained of illness about 3 weeks ago
when he fell onto a stove, & had severe head
ache, on going to bed, where he did not sleep
but tossed about restlessly all night.

The next morning the headache was worse
vomiting, & he vomited twice; on attempting
to get up he was unable to stand, on account
of dizziness. He remained in this condition
for three days, but getting no better he
called in a medical man who told him
his stomach was out of order. He took
the medicine he prescribed, but his account
was no better; in fact got worse, the headache
becoming more constant and severe
and vomiting on an average 4 or 5
times daily, and it was with difficulty
that he walked about. Because of the
dizziness thereby induced. He accordingly
sought advice again to the hospital.

Prescription: the illness, he felt had been good
never having had any serious disease.
Four years ago he had gonorrhea, no
history of syphilis. He appears to have been
a fairly steady lad, not addicted to drink.
Present or alcohol seen.

Family history good. Father killed in an accident. Mother & 6 brothers and sisters alive & healthy.

Present Condition

Well developed strong and intelligent looking lad, of cheerful aspect, when not suffering from the paroxysm of pain in the head. His skin cool and free from perspiration. Tense 37.5

Nervous System

At times he usually suffering from a more or less constant pain affecting the whole head, which when he is lying quite still, is dull or achy in character, but becoming on movement sharp, cutting & stinging, accompanied also by severe vertigo. The room appearing to while rapidly around him usually from left to right. He has no special debilities, but when the pain is not very severe, he as a rule lie on his back with the eye closed & apparently sleeping. He answers intelligently any question that may be put to him, and gives a clear and coherent account of his past illness & antecedent life. Memory good.

Motor System

He complain of feeling a little weak on left arm, but on testing this, no appreciable difference can be discovered. Dynamometer, registering 60 on left
side. 64 on right. He can perform all the movement of both arms perfectly, quickly, and without any difficulty. On walking he staggers considerably, or may be says to stagger, and when lying down, there is no evident muscular weakness.

There is no impairment of General Sensibility.

**Special Senses**

**Vision.** The amblyopia of left eye, objects appearing indistinct and dim.

No hemianopia. The visual fields however considerably contracted peripherally.

In left eye the vision is distinct, 0 unilateral.

Optokinetic examination of left eye.

Show swelling of optic disc, with obscurity of outline. The disc swollen & much curved over the prominence of disc. No haemorrhage. In Right eye there is a slight blurring of the temporal edge of disc.

There is no modification of the other special senses.

**Reflux.**

Organic reflexes normal, plantar reflexes not marked on both sides. No ankle clonus. Biceps reflex present in both sides, but most markedly on the left.
Digestive System

Inquire corded to cut up greyish white fat, feels good. Appetite poor and bowel obliquely confined. He has no abdominal pain and physical examination does not reveal any abnormal condition with abdominal viscera. Hepatic dullness begins at upper border of 5th rib in mackellar line. It extends downward 3 pin edge of ribs below, measuring 4 1/2 inches.

During the first 24 hours in hospital he has been sick 5 times, he vomited mutton, being the almost unaltered contents of the stomach. The sickness is very likely to be provoked by movement and there is sudden and accompanied by much retching.

Physical examination of respiratory and circulatory systems reveals nothing abnormal pulse 48, full, strong, and regular.

Urini 10.20 A.M. 26 oz. in 24 hours. Deposits, mucous, triple phosphate, alkaline reaction. No albumen, sugar or casts.
Proptom. July 28. The Headache, stiffness and vertigo still continue, some days he feels a little better, but the succeeding day shows a returning relapse. Pulse 50.

His range from 42 - 52. Temperature 36.8°. He remains in an apathetic condition all day apparently dozing except when aroused to movement by the intensity of the pain, which latter affects the whole head. It is not localized to one part. He rarely awakens with pain, or nausea, but when questioned gives coherent answers. He converses for the first day or two has found difficulty in choosing the right word to express his meaning, and often says, "no," when it is obvious that he means "yes." Whilst seemingly remiss in amnestic errors, there is a decided weakness of the left arm; dynamic motor synergia 32 to left hand, 60 to right. He can

Fremt. 2. He has not been sick since last entry. The headache less severe. Still continuing lethargy, still more marked. Yesterday he vomited some in the morning.

The function of left arm rapidly progressing. He is able
completely blind in the left eye. I can only distinguish light & shade with the right eye.

Opticuscope examination shows well-marked v. advanced relucence in both eyes, with some hyperopia.

Sensibility still remains intact.

March 3

today he had another spell of the left arm lasting for about half an hour, and shortly afterwards, he sunk into a state of deep torpor, from which it was with great difficulty he was awoken.

Temperature 37.4. Pulse 48. Vomited once this morning.

March 8.

The arm is now absolutely paralyzed & the patient of left leg also, but actually accommodating to paralysis. Temperature 36.8. Pulse 48. Slight sweat but regular. Sensibility unimpaired. Absolutely blind on both sides.

Still passes the urine 5-6 times per day, kept incontinent of bowel since which it is impossible to arouse him, the pulse coming down to 32, very small & weak & occasional intermittent. The respiration shallow, irregular, exhibiting the Cheyne-Stokes variety.

March 10 To day had another spell of the left arm, affecting also slightly the left
leg also. He continued carlying for nearly an hour.

March 12. Died this morning. He passed into a state of unconsciousness, from which it was impossible to arouse him. Respiration became very shallow, I sighing, Y at last good became altogether.

Post Mortem examination 30 hours after death.

Post-mortem rigidly well marked. Skull very thick, there made neither adherent to the cranial bones nor. The brain & stripped quite easily. The vessels of meninges were much engorged. On removing the brain, which weighed 1420 grammes, a general bulging of the right prefrontal region was observable. On examining a large cyst was found bulging on the orbital plate of the frontal bone. Y. occupying a cavity in the orbital surface of the prefrontal bone of the cerebrum, from which it was easily removable. The right prefrontal region was thus hollowed out into a cup shaped cavity 1/4 wide, 1/2 inch deep & 1/2 inch in diameter for the reception of the cyst. The cortical grey matter was not destroyed, but quite distinct although
Aphrodis measuring about half a line in thickness, the hollowing out having doubtless been formed chiefly at the expense of the white matter of the cerebral cortex. The convolutions of the prefrontal region were flattened. V. Also to a lesser extent, the bases of the two lower frontal convolution, the inferior third of the precingulate frontal convolution. The rest of the convolutional arrangement of the brain was normal. There were no signs of meningitis or cerebral inflammation. A vertical transverse section of the prefrontal area, would be represented diagrammatically, as following the section being made two inches in front of the fissure of Rolando.

The basal ganglia were found very little disturbed, the anterior end of the external capsule being somewhat narrower than the corresponding part of the right hemisphere. Apparently due to a slight pushing backwards and forwards of the head of the ventriculus nucleus. There was no decussating degeneration. The cerebellum, medulla oblongata normal in appearance.
The Cyst Which Was Evidently a Hydatid One, Had an External Opalescent and Semitransparent Membrane I Measured When Uninflated from the Brain 2½ in. Across and 1 in. in Depth. It Contains a Colorless, Watery Fluid in Which Were Floating Minute Daughter Cysts, and When Examined under a Microscope the Characteristic Hydatid Hooks Were Found.

Remarks

From the point of view of localization the case although accompanied by well defined motor disturbances offers no special interest except in so far as showing the objection of citing cases of cerebral tumour as evidence for or against the doctrine of cerebral localization. A tumour may either compress or destroy, and from the alteration in the aachainel connection of parts, and the indirect result of pressure on distant structures much caution must be employed in drawing any conclusion as to the relation of the symptoms and the site of pathological lesion. In this case there was evident great destruction of the centrum ovale in the prefrontal region and also perhaps of post-frontal region, yet the
cortical grey matter, although absorbed did
not suffer to anything like the same
extent and this fact is worth remembering
in estimating the value of these recorded
cases, where in the cortical zone is implicated
by tumours etc. without any modification
of mobility ensuing.
The constant place in this case, thinking
that there was no inflammatory condition
of the membrane, must be attributed to
stretching of these membranes, which also
may account for localised localization of
the pain, which affected the whole head.
The mobility and elasticity of the tumour
may possibly account for the sudden
exacerbation of the pain on movement
together with the frequency with which
such exacerbation was followed by
vomiting.
John McFee. Wrote down.
Admitted Sep. 1, 1883, to the Hereford
British Hospital Paris

In care of Dr. Herbert

Complaining of headache, vertigo, vomiting &
Admission of vision.

About four weeks before admission he
began to suffer from severe pain on the
right side of the head, accompanied by
transient attacks of dizziness. Thinking it
depended upon disorder of the stomach
he took large doses of salt and caffeine,
pills but without experiencing much
relief. Since then the symptoms have become
gradually but decidedly more troublesome
and for the last three weeks he has
complained three times to three daily. He has
also noticed lately that his eyesight is
failing, which formerly was exceedingly
good, but now it is with difficulty that
he can see to read, and distant objects
appear blurred and indistinct.

For a fortnight he has not been able to
pursue his employment.

His previous health has been exceedingly
good, never being under medical treatment.
His skull had very few blood vessels
and family history is free from phthisi,
epilepsy or insanity. Both his parents
and friends testify to his strict sobriety
and steadfastness.

Present Condition
He is a well-developed man in fair
physical condition. Anemic & suffering
from depression. Temp 37. Skull more DO Cool
with no crepitations.
Nervous System
He lies on a pillow, on his back, taking little
interest in his surroundings, with eyes
usually closed. On being addressed
he quickly responds in a cheerful
and coherent manner. After conversing
some minutes, it is evident that the task
is an effort to him, and at last he
becomes irritable & his answers become
cold and monosyllabic. His recollections
are quite clear and apparently truthful,
and he finds no difficulty in the
choice of words, but his memory as
regard, time is evidently impaired
as he cannot say if a given event
occurred last week or a month ago.
He complains bitterly of the pain in
the head, which occupies the whole of
the right side, nearly always present
and of a dull aching character
with sharp, shooting paroxysms.
On making any sudden or external
movement the pain is increased.
Usually accompanied by vertigo, the surrounding objects being in a state of vibration. When asked why he does not sit down presently with the newspaper etc. he answers that thinking hurts his head.

**Motion**

There does not appear to be any impairment of motility. The grasp of both hands is strong and he can perform all the usual movements of the arm and hand. On attempting to walk there is some unsteadiness at first, accompanied by vertigo, but in a few moments the pain off and he is able to walk steadily.

**Sensibility.** There is no change of sensibility. The tactile sense is that of pain and heat being normal.

**Reflexes.** Organic reflexes normal.

Plantar reflexes "tends to extend, will mark one foot side. No ankle clonus.

**Special Senses**

**Vision.** Pupils equal, somewhat dilated and reacting normally to light and distance through somewhat sluggish. On attempting to read the letters run into each other, and in regarding distant objects the outline is blurred and indistinct.
Ophthalmoscopic examination of left eye.
The edge of the disc is indistinct on the temporal side, with engorgement of veins. No haemorrhage.
Right side, the general outline of disc blurred and indistinct with accentuated engorgement & also slightly situated on the upper half.
Vitreous mild & not contracted.

Hearing

Can hear the ticking of a watch on the left side at a distance of 14 inches.
On the right side only at 1 inch distance.
There is no change condition of the ear.
Ear & palate Cannot say that it is his normal habit, not being aware that he was deaf in that side.
Selling of voice and voice apparently not modified.

Digestive System:

Feces clean, breath offensive, color, constipated. Appetite poor. During the past 36 hours presence in hospital he has been seen four times, the vomiting not accompanied by marked diarrhea & being sudden in its onset. Inspection, palpation & percussion do not reveal any abnormality in the abdomen or rectum.
Circulatory System.

Pulse 42 full but soft & compressible & regular. No precordial pain or palpitation. After beat 3/4 of inch to the right of left mammary line at the 5th interspace. The first sound is weak in all the areas, the second sound occasionally reduplicated in the pulmonary area. No murmurs.

Urini 30 cc. l. 24 hr. 5.7. 1022.

Pure yellow color. Depositing phosphates no albumen, sugar or albumen.

Prognosis.

Sep 12. No amelioration in his symptoms. He lies in a lethargic condition, rarely moving except when urged to do so during his attacks of vomiting which occur as a rule twice or thrice daily. Allows his food and letters to remain untouched, and although he recognizes his friends when they visit him, yet immediately he appears to become oblivious of their presence. There is no change in mobility or sensibility. He still suffers from the headache, which is confined to left frontal region. Blood pressure between 26 & 36, 4, pulse from 38 - 44.
Last 3 days, and he nor
lie in a state of almost complete stasis.
Although he can be moved for his food,
I can answer questions in a somewhat
gestural, halting manner. He passed his
fees in bed to day for the first time.
He is becoming weak & nauseated.
Yet there is no special press of the
movements of the body. Neither is there
any affection of mobility.
He can only now distinguish light &
shade by ophthalmoscopic examination.
There will marked optic neuritis
with extravasation in both eyes.
Temperature 36. Pulse 54.
He still suffers pain in the right flank
region.

Oct 1. Yesterday the nurse reported
several convulsive attacks of spasm
of the left arm, and to day there is
contraction of this arm, the forearm
being flexed on the arm. There is
no change of mobility elsewhere.
Neither is there any change in mobility.
Temperature in right axilla 38.4. in left
39. Pulse 76. He has been very
since last entry. Small bed sore on
the sacrum.
Oct. 4. The contraction of left arm has now entirely passed away leaving it absolutely paralysed, although he remain perfectly sensible to touch, pain, thirst, &c. No paralys of any other part of body. Sometime there today, three yesterday, he has appeared somewhat brighter, but still complains of pain in the head. Temperature in right axilla 37.2 left 37.6.

Oct. 8. Died somewhat suddenly today. Yesterday had an attack of general convulsion affecting the paralysed left arm, as well as rest of body, lasting for several minutes. Temperature in day of death 37.6.

For three past few days he has been much brighter than in the former part of his illness. Headache continues to the last as well as the vomiting.

Post mortem examination 36 hours after death.

Region mortis well marked. The calvarium thick and not adherent to the dura mater. No fluid in subdural space. Venir on the surface of brain much engorged and on attempting to strip off the dura mater it was found to be adherent over
right occipital and right parietal regions from the rest of the brain it came away quite easily. Brain weight 1360 grams. The posterior part of right hemisphere was somewhat larger than the corresponding part of the left hemisphere, its convolutional arrangement being destroyed, its colour being of a dark red, markedly contrasting with the adjacent normally dense part. On making section through the occipital lobe, it was found to be of the same dark red hue, presenting numerous patches of soft half-thick blood-tinged material. The whole of the occipital right was thus affected. The medullated cylinder however being almost entirely distinct there, although not strictly defined, yet it merged very abruptly into the surrounding normal texture. As stated above the dura mater also was adherent to the to part of the parietal lobe, more precisely the upper half of the ascending parietal lobe and encroaching to a very slight extent on the adjoining ascending frontal convolution. On attempting to remove it, it was found to be impossible to do so, without tearing the brain substance subjacent, which was here soft and pusible of a reddish-yellow colour. This softening extended for about 3 of an inch into the occipital part of the brain.
The brain ganglia on both sides were normal in appearance and had suffered no distortion, the tumour in the ventricular lobe having apparently simply replaced the ordinary brain tissues. The corpora quadrigemina were also normal, as was there any degeneration of the optic nerve. The pineal body and cerebellum were also carefully examined, but no degenerative degeneration was found.

The thoracic and abdominal areas were also examined but no morbid condition was noted.

While examined microscopically the tumour was found to be composed of characteristic gliomatous appearance—rounded, oval, and epithelial nuclei, embedded in fibrillar network.

Remarks: From the cephalalgia, vomiting and neuritis, with absence of any change in mobility or sensibility, it was thought to be case of meningocerebral disease, which from the site of the pain probably affected the frontal region. The ultimate development of paralysis of the arm appeared to support this view. The autopsy revealed two distinct pathological conditions in a given gliomatous line and a
meringu susceptible, the latter being of a much more recent date probably than the former. The somewhat sudden appearance of signs, developing into complete paralysis, doubles, one Rieh, the remission of the inflammatory process. It will have been observed that coinciding with the motor symptom we had both a local & general rise of temperature, the temperature proceeding subnormal rising to 38.4 on the right side and 39° on the left. Kellner, Landor, and Spenic have shown that in paralysis of due to cortical disease there is a vessel motor dilatation of the paralytic part & consequently a rise of temperature and in quite recently Richet has shown that in vertebrobasilar lesion of the artery there is also produced at an interval a general state of pyrexia - a fièvre traumatique reverse - in which in a few hours there is a rise of as much as three degrees C°. In the general convulsions occurring before death, it is interesting to note that the previously paralyzed arm participated in the general spasm, and it is instructive to observe
This fact, with the experimental fact determined by Delas & Thomas, that if a cerebral pyramid be destroyed, yet this does not prevent the extension, of the convolutions produced by continued irritation of an other cerebral centre, to the sounds corresponding to the centre thus destroyed.

The site of cerebral reimplantation lesion is

The upper half of this uniting parallel convolutions containing the centre for movement of hand and arm, would account for the transient monoplegia occurring, in this case.

Although the right occipital lobe was entirely destroyed there was no hemispherical presented in this case, but distinctly analgesic paresis towards to complete blindness, with well-marked optic neuritis so commonly seen in cerebral tumours.