A Critique

on the Diagnosis of Cerebral* Lesion

with ophthalmoscopic Illustrations.

*The word "cerebral" is used here to denote the whole brain, the cerebellum inclusive.

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An accurate diagnosis in cases of brain disease, is justly considered to be one of the most difficult problems in medicine. The description and discussion of cases and instances in illustration of this matter, generally commands from the profession, that sort of deference which we are accustomed to give to subjects, which make an imperative claim upon our attention, as much perhaps, for the speculative interest implied in them, as for their bearing on practice. For it must be admitted, regret it as we may, in a practical point of view, that cases of nervous disease, are, generally speaking, of greater interest to the pathologist and speculative enquirer, than to the therapeutist or the physician in busy practice.

At the same time, if it be true that the advance and success of medicine, is to be secured by the right knowledge of the cause, rather than of the cure, of disease, one is inclined to believe that any effort made in this direction, however unpretending, cannot be entirely without value.

In selecting cases illustrative of the purpose of this paper, I have preferred to confine my remarks to a very few ~~cases~~, only two or three, but these of very considerable interest, rather than to give a greater number of less individual value. I have accompanied the verbal description with some drawings of the ocular phenomena, as this is a matter of some importance. Now that the ophthalmoscope is, one may hope, coming into use in general medicine, it is as well to be on our guard against errors which are easily fallen into in this respect. I do not think it is generally known, I had no idea of it myself till quite recently, how deceptive these ocular appearances may be. The signs of inflammation of the optic nerve, which the ophthalmoscope enables us to detect, are considered by the most experienced authorities to present a most valuable index, perhaps our most reliable one, in the diagnosis of organic cerebral lesion. Yet the two main cases recorded in this paper, and I refer to others incidentally,

shew how mistaken we may be, if we pin our faith to any one symptom, however trustworthy in itself.
Nothing shews the uncertainty of medicine more than this. And where so much obscurity, unfortunately, exists in matters of such great moment, it is perhaps scarcely necessary to make any apology for a treatise which attempts to deal with the subject in question.
I will therefore at once proceed to the discussion of the illustrative cases. The first is that of an Irish labourer who was a plasterer by trade. A trade which, I may remark in passing, may fairly be called a laborious one, necessitating as it does, the carrying of heavy weights, mostly on the head, and the assuming, for hours together, of constrained postures. Of late this man had found himself unequal to his work and felt so far unwell as to induce him to seek medical aid. When I saw him, he was complaining of what he called "bilious attacks," characterized by headache, dimness of sight, vomiting and general malaise. But the man's aspect and manner, the unhealthy pallid hue of the skin, and the constancy of the headache and vomiting made me suspect some deeper mischief than a disordered liver.
On Examination I found some increase in Cardiac dulness outside the nipple, the first sound was thumping but without bruit, the second (aortic) was accentuated and reduplicated and the heart's impulse heaving. The arteries were ~~palpated~~ corded and tortuous, indeed remarkably so for a man of forty. The pulse was hard & full, and I observed a well marked arcus in the upper segment of either eye. I was not surprised therefore, to find that in addition to a laborious occupation his health had been subjected to the strain of reckless living, with free indulgence in alcoholic and other excesses. He had had one attack of Gonorrhoea; there was a doubtful history of Syphilis (upon which however a careful inspection threw no further light), no history of rheumatic fever or malignancy

In the chest- nothing abnormal was discovered except a few mucous râles.
In addition to this there was some fulness of the liver, with slight tenderness on percussion, though not an overtuning to much. The Spleen could not be felt. There was no ascites Patient had been jaundiced, but was not so now.
On examining the urine, I found it slightly albuminous (the albumen was never absent, although sometimes it did not amount to more than a trace) with some degenerated granular matter and altered Epithelial cells: but I never succeeded in detecting a cast. The specific gravity was low, seldom rising above 1015, and the amount passed was slightly in excess The urine was pale in color, acid in reaction, and sparing in solids. There was some frequency of micturition -
The bowels were sluggish.
What little could be gleaned from family history was important in its bearing on the case. His father, patient said, had died of asthma and his mother had paralysis (presumably from hemorrhage) The offspring of such parents could hardly escape some predisposition to arthritic or vascular disease. The present instance, indeed, as the sequel will show, was a case in point.
Here then, it appeared, was a simple case of premature decay brought on by an unfavorable conjunction of circumstances; heavy work, dissipated habits, the abuse of alcohol, and some hereditary predisposition: the stress falling, in the first place, on the abdominal viscera, stomach, liver and kidneys, the head and head suffering secondarily; and that the urgency of the vomiting and the constancy of the headache were to be put down to the gravity of the case. But when I came to examine the eyes I was inclined to doubt the correctness of my conclusion. The condition of the eyes (see next page.) was, it is true, that which one would expect to find in chronic Bright's disease, ~~the~~ a number of white and brilliant

(right) (left)

Double Optic Neuritis with appearances characteristic of Albuminuric Retinitis in a case of Morbus Brightii, in which there was no brain lesion besides hemorrhages.

specks in the vicinity of the macula, the mealy character of the fundus around the disc, and a few hemorrhages, chiefly linear and pointing to or from the disc. But then there was more than this, for in both eyes (and especially in the right) there was very considerable swelling of the disc, the margin being quite indistinct, and a spreading of effusion more or less into the fundus. At the time when the annexed drawing was taken, the neuroretinitis of the right eye, (which took the lead) had advanced so far as quite to obscure the spots and hemorrhages, and to produce, I imagine, some atrophy of the choroid. This then, the optic neuritis, was the most prominent symptom; prominent, I mean, in the sense of impressing the mind of the physician with the gravity of the case, for it is a symptom which is suggestive of serious brain disease. Now was that all, there were other symptoms in favour of a cerebral diagnosis. The headache, of so constant a character and the frequent bilious vomiting are accessory symptoms which may be confirmatory in establishing a diagnosis of cerebral lesion. But these symptoms to be of value, must be of a certain character. The headache which is diagnostic of organic brain disease is generally pretty constant (sometimes paroxismal) and is a severe pain, not merely a sensation of weight or fulness; and then it is often localized in an unusual situation, such as at the back of the head or at one side. Now the headache in the case in question, although constant was certainly not very intense, and it was generally frontal and not very localized; and although it was hardly of that neuralgic type, to which the name of migraine has been applied yet I was inclined to place it to the account of the digestive troubles or the vitiated state of blood, rather than to any cerebral affection. And indeed if the headache had been very severe it would not be decisive proof of a cerebral origin, although more suggestive of it: for we have it on the authority of one of the best clinical observers,

† Murchison on "Hepatic Derangements" 1874. p 111

*. Day. on "Headaches" 1878. p 229.

(Dr. Murchison) that neuralgic headache occurring in connection with contracted Kidney is sometimes so severe that more than once he has known such a case diagnosed as one of Cerebral tumour: and Dr. Murchison urges the propriety of investigating with care the conditions of the Kidneys in all cases of neuralgic headache occurring for the first time in a person of middle & advanced age.† And similarly Dr. Hughlings Jackson lays great stress on the necessity of Examining the urine in all cases of suspected cerebral disease

At the same time bearing in mind the fact that in cases of Extreme granular Kidney there is often no albumen to be found in the urine; so that absence of albumen does not in itself negative renal disease. Now again, (to do justice to this Symptom,) I would the fact of headache being in an ordinary site, (in the forehead for instance as in this case) and being somewhat diffused in character, rather than localized, quite negative its value as a cerebral Symptom - "It may (says Dr. Hughlings Jackson) be referred to all parts of the head and exactly simulate those which are of less serious origin."* So difficult is it to arrive at certainty, and so much a matter of probability, is the Science of medical diagnosis!

Then as to the vomiting: this was hardly of the purposeless character which is supposed to indicate a cerebral origin. For the patient often threw up his meals and complained of nausea. The Gastric mucous membrane was clearly at fault. The tongue was coated and the breath foul: and more than this, there was that redness at tip and Edges, with Elongation of the fungiform papillæ which Dr. Murchison has described as almost pathognomonic of hepatic congestion; a state of the tongue commonly put down to atonic dyspepsia. The patient also complained of flatulence and was troubled with eructations So that, whatever, and wherever, the Structural lesion might be, he was undoubtedly suffering from some functional derangement

of the digestive system. The bilious character of the vomiting really means nothing more than that it is strong enough and long enough to force bile out of the gall bladder & duodenum, and does not in itself point to either liver or brain as "pons Varigo mali". In even where vomiting is purely cerebral, as in the pregnant or the hysterical, the reversed peristalsis is generally set up in the first place by the contents of the stomach acting as an exciting cause. In seasickness, or in an attack of migraine the stomach will often wait, as it were, for provocation by the ingestion of food before vomiting is induced; and then when the mechanism is once set agoing the nervous system perpetuates the unfortunate trick of purposeless retching and vomiting of nothing but bile and gastric mucus, forced up by the urgency of abdominal compression. And indeed when so sickly a thing as bile once gets into the stomach, this, one would think, is quite enough to account for some continuance of nausea and vomiting. But in the case in question the symptom might fairly be accounted for by other than a cerebral explanation. The liver was evidently swollen and congested, in the early stage, no doubt, of alcoholic cirhosis, (the patient had been accustomed to drink spirits "neat".) Might it not by its inordinate pressure act mechanically to cause vomiting, in the same way as it has been suggested a full stomach will, through juxtaposition set in action the peristalsis of the colon and thus induce defecation. But a more likely mechanical reason would be the pressure of a distended liver on the semilunar ganglia. Or again, the vomiting might be due to a loaded portal system, affecting the enteric and gastric mucous membrane with œdema. This œdema, again, might be traced more to the renal than to the hepatic disorder. In the present instance however there was no evidence of œdema anywhere else. Vomiting, again, is a common symptom of renal disease, in a toxic point of view, owing to the

uraemic, or at any rate, lithaemic state of the blood: for in advanced Kidney disease urea has been detected in the vomit. Or, again, coming back to a more simple explanation, the long ~~[illegible]~~ neglected and chronic gastric catarrh which this man was probably suffering from ("inter alia"), might, one would imagine, be quite enough to account for the vomiting. Taking these facts into consideration therefore, I concluded that this Symptom also must be absolved from any cerebral connection.

Were we then, in like manner, to refer the dimness of sight, of which the patient complained, to his disorders of nutrition and digestion? Now as far as the diagnosis of organic cerebral lesion was concerned this Symptom would not help us much. For it could only be of value, in this respect, in connection with the affection of the optic nerve, which was discovered here by quite other means viz. the ophthalmoscope. Besides this, one cannot depend on amaurosis as being a correct indication of optic neuritis. At the time when I first saw the patient the neuritis of the right eye was more advanced than in the left and yet on applying the usual ophthalmic tests he seemed to see equally ill with both eyes; so that I was not inclined to attribute the amaurosis to the inflammation of the optic nerve, at this stage of the case. When the disease in the right eye had advanced (as it did very rapidly) to atrophy of the disc, there was of course total blindness in this eye, and the cause was obvious. But well known that, short of atrophy, optic neuritis may exist even in a marked degree with no defect of vision (the patient being able to read the smallest "Snellen" or "Jaeger") thus misleading both patient and physician unless the ophthalmoscope be used: and as the inflammation may clear off, leaving not a trace behind, it is often quite impossible to tell whether it has ever existed, unless it be actually seen at the time. Optic neuritis being, in fact, a Symptom in itself, has no symptoms of its own

† Wilks: "Pathological Anatomy" 1875 p 258

Seeing that the patient was the subject of renal incompetency, probably in an advanced degree, I inclined to ascribe his dim and imperfect vision, like his headache, to this, as a general cause: especially as the defect in sight kept varying from time to time and was accompanied with "muscæ volitantes". Whether it was especially due to the albuminuric retinitis, or to the hemorrhages shed on the fundus or to the anæmia of the retina, or to the generally unwarranted condition of the blood (to any of which it might be due) I do not pretend to say.

If then the cerebral origin of the headache, the vomiting, and the dim eyesight was as improbable as the foregoing remarks seem to shew, what should we say to the double optic neuritis, the symptom, which, in this respect, I called the most prominent one of all? Could this stand alone as being sufficient in itself in arriving at a diagnosis of cerebral disease? The sequel of this case proves that it could not. The affection of the optic nerve, which can come and go with so little apparent inconvenience to the patient, is of such intense interest to the physician and so much for its own sake but for what it may reveal as a symptom of intra-cranial disease. The diseases of the retina are, as Dr Wilks remarks, the diseases of the brain. And this may well be so because the posterior chamber of the eye is originally formed by a diverticulum of the anterior cerebral vesicle, the lining membrane of ~~which~~ the protrusion becoming the choroid and retina. As a matter of clinical experience, it has been found that double optic neuritis is the best symptom we have of gross disease of the brain (such as a tumour or an abscess) but that in itself, in the absence of other confirmatory evidence (E.g. affections of other cranial nerves etc.) or when that evidence is so slender, as in the present case it must not be held to be absolutely decisive. What then was the meaning of the neuritis here? If it did not shew

* Hughlings Jackson on "Nervous Symptoms in Congenital Syphilis". Journal of Mental Science 1875

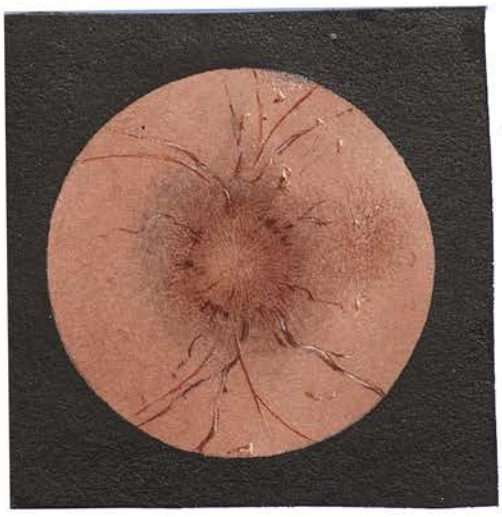
against brain disease, what did it shew?
If we give up the diagnosis of Gross cerebral lesion, this would exclude Syphilis (which in spite of the patients denial and the physicians inability to detect always lurks in the back ground as a possible cause). That is to say if it be true that "Syphilis causes the symptoms not because it is Syphilis but because it generates the condition which causes it" tumours". * Now any Syphilitic formation a gumma occurring in the favourite seat, (the interpedunicular space a thereabouts) capable of producing ischaemia and neuritis of the second nerve would in all probability lead to other results as well, such as paralysis of ocular nerves in their intra cranial course; moreover we might expect to find in such a case that other characteristic affection of the fundus the disseminated Choroiditis of Syphilis. But there was nothing of the kind here. But a tumour may and perhaps usually does set up optic neuritis not so much by acting mechanically like a foreign body, as by exciting local inflammation around it, which, creeping down the nerve appears as a "neuritis descendens". Was there then any evidence in this case of inflammation in the brain apart from tumour? Now in spite of Sir. Jacksons ascertain quoted above one is inclined to ask, might not syphilis cause Optic neuritis by setting up as it is known to do, meningeal inflammation of the base. The suggestion is favoured by some remarks of Sir. Broadbents, which I will quote. He says. "No strict line of demarcation can be drawn between cases in which there is extensive Exudation in the membranes (of the brain) and those in which the morbid process results in the formation of distinct tumours." And again "If a syphilitic tumour is present and is not complicated with meningetis, its Effect on the retina is to produce a choked disc, as the result of pressure interfering with the venous return from the retina. On the other hand if basilar meningitis of the anterior lobes be present we may have true optic neuritis from its spreading downwards

* "Patholog. Anat. of the Nervous Centres", 1874.
W. Tilney Fox. p.p. 145. 6.

† W. Fox. "Patholog. Anat." p 389.
and W. Broadbent "on Syphilitic affections of the
Nervous System". Lan. 1874 vol I p 255

along the nerve of the meningeal inflammation" *
Still the same negative objection would apply, as stated above
for if there were syphilitic inflammation of the base, we should
expect to find some evidence of the nerves in their intracranial
course, as the base of the brain being surrounded and involved
by the inflammatory lymph. But no such evidence was forthcoming
here. Then again, according to Dr. Broadbent, the existence
of syphilitic encephalitis as distinct from meningeal inflam-
mation has been very much doubted, and the same may be
said of neuritis.
Again, the chronic meningitis of drunkards attacks the
convexity of the brain and would not be likely to lead to Eye-
symptoms
Was there any acute meningitis from some cause or other?
But - this did not seem likely in the absence of febrile symptoms
It is true that the temperature in tubercular meningitis is
sometimes only very slightly raised; and again, the pulse
in this disease is often not much accelerated; but if not
accelerated it must be ~~abnormally~~ not only slow, but abnormally slow,
and irregular as well, and such was not the case here.
Had the patient had any cerebral hemorrhage before I saw
him? "Apart from the albuminuric alterations in the eye
Cerebral hemorrhage seldom leads to any change in the fundus;
but optic neuritis is occasionally a sequence of it." † But
there was no reason to suppose he had; he could give me
no history to lead me to suspect it, and he was tolerably
truthful and intelligent. The possibility of abscess in the
brain, acute or chronic, was negatived by the absence of other
signs of gross cerebral disease, as well as other special symptoms
of such disease (Deafness, Earache, discharge, deafness, giddiness,
& other signs of bone disease) Of course this does not prove
that the man had no abscess in his brain; for an abscess
may be quiet throughout life, as Dr Wilks says. "A man
with ounces of pus in his brain may continue to do good

* Wells' "Pathology Ocul." p 233



Optic Neuritis with hemorrhages in a severe case of Plumbism

† Fox. Op. cit. p 298.

mental work" and have full use of her limbs"; * but only proves that the optic neuritis is not sufficient evidence singly to determine such a disease.

As a last resource I thought of lead, as a possible cause. I had seen only a short time previously a case of lead poisoning in which there was well marked double optic neuritis. I give an illustration, of the most characteristic eye, as I do not think the association is very common, and the appearances are almost exactly like a "tumour disc".

Amaurosis is sometimes said to be met with in lead poisoning depending on optic neuritis: and Dr. Meyer has put on record two cases occurring in sisters, who were employed at Brussels in bleaching lace with the aid of white lead. In one complete atrophy came on in four months, the other was similarly affected though not to the same extent. † I have seen another case besides this one from which the amaurosis was taken: but of course the association might have been accidental. Still, lead seems to have a special affinity for nerve tissue, and if it affects other nerves besides the one which suffers most commonly & obviously (musculo-spiral) one does not see why the second nerve should be exempt. The suggestion of plumbism is confirmed by the known resemblance which exists between the effects of lead poisoning and the symptoms of lithiasis (with or without kidney disease). The introduction of lead into the system will, as a matter of fact, arrest the oxidations and elimination of nitrogenous waste, thus causing a true lithiasis, with all the attendant evils of functional and structural change in heart and kidney, with high arterial tension. Now this man's case quite answered to this description. Was there then any evidence of lead poisoning to account for it? I cannot say that I could glean from the history anything that was at all conclusive, and (not unmindful of therefore of the occasional misdirections of the afflicted)

in the absence of the more obvious and usual manifestations I considered the idea too problematical to be of much weight in diagnosis.

Gouty inflammation is known to attack nerves all over the body, and a promiscuous sort of fashion, but I never heard optic neuritis ascribed to this as a cause: and I must confess I do not know what had occasioned it in the present instance - unless it might be from the alcoholic excess. The patient was not much of a smoker.

There was therefore not sufficient evidence in this case to establish a cerebral diagnosis.

I concluded the case was one of functional derangement of stomach and liver, resulting in a state of kidneys, with structural degeneration of the kidney and secondary affection of heart and vessels. For the present purpose it is not necessary to discuss the etiology, significance and sequence of the abdominal and other affections, or their correlation as cause and effect.

This patient then ~~died~~ was the victim of disease of more than one important organ, but it was not of any of these affections that he died. As the sequel will show, he rapidly sank through the rupturing of vessels. He died because his vessels had been weakened by disease. Had he lived longer, the organic disease of the liver, which, I suppose, was at this point of developing would have advanced, with an aggravation of abdominal symptoms and the superventions, perhaps, of anasarca and serous inflammations which never appeared throughout the case.

Before detailing the progress of this case, I will refer to another of a somewhat analogous nature, and which suggested the same difficulty to mind. A man of middle age applied at an ophthalmic hospital. He had uræmic retinitis, and as he was also found to have albuminuria, with vomiting, and other digestive troubles no doubt was entertained as to the nature of the

complaint, clearly a renal case. But, a short time after, the man died, with symptoms of tubercular meningitis. At the "post-mortem", tumours were found in the brain and the kidneys were perfectly healthy.

The progress of the case was uneventful for the first two or three weeks. Considerable benefit seemed to be derived from a simple palliative treatment afforded by saline cathartics alkaline diuretics, rest and dieting. The case did not admit of anything more than alleviation.

Then the patient one day had a fit. This is what was to be expected. When hemorrhage occurs in the retina and from the nose in a man, the subject of vascular disease, one is not surprised to find, some day, that a meningeal artery has also given way. This was, in fact, the first of a series of apoplectic seizures which now hurried the case to a final issue.

From this time, therefore, the case had entered on a new phase: for it was now a case of true cerebral lesion.

The first attack was of gradual onset. It came on while patient was at stool (there was no undue straining and no melaena). The account the patient gave of it was that he felt a strange feeling came over him and a darkness before his eyes, but he was able to struggle back to bed. He did not lose his senses nor was he convulsed, with the exception of a slight trembling in both arms. On seeing him immediately after the fit: I found him lying on his back, breathing stertorously, he told me what had happened in quite an intelligent manner, but his utterance was thick and tremulous. The face was slightly drawn to the right, the tongue on protrusion deviated to the left - there was, in short, the ordinary incomplete facial palsy of hemiplegia, only marked on the execution of voluntary actions. Both pupils were somewhat dilated, and equally so, and responded naturally to light. On testing the muscles of the limbs I found some loss of power in the arm and leg of the left side

"The muscles were lax and responded with undue readiness to the galvanic current. Tactile and thermal sensibility seemed unimpaired

"The second attack came on quite suddenly. It was characterized by complete loss of consciousness, both arms and the left leg were strongly convulsed. On Examination a few minutes after the attack had occurred, the palsy and aphasia were found to be more complete, and there was some loss of common sensation on the affected (left) side In addition to the Symptoms resulting from the previous stroke, the left pupil was observed to be sluggish and unnaturally dilated: the uvula was drawn to the left side, and there was some dysphagia: that is to say, in the attempt to drink the fluid was apt to regurgitate. Now also, there was some intellectual impairment.

"From this time forward the case rapidly advanced to a fatal termination. This occurred three weeks after the first attack. During this time the patient lay mostly in a lethargic, semicomatose condition. Several fits occurred and in the intervals occasional involuntary spasmodic jerkings of the limbs were observed. The fits were characterized by loss of consciousness and convulsions. During these fits the left arm was sometimes rigidly extended, while the right would at the same time be flexed at wrist and elbow the left leg being extended and sometimes convulsed; but the right leg was thought to be not much affected.

"Towards the end there was conjugate deviation of the eyes to the right, and the fits were generally ushered in by a turning of the whole body to this side. The sphincters were relaxed and there was a generally increasing feebleness both of body and mind till he died.

No rigidity of the muscles in the affected limbs. Either "early" or "late" was observed throughout the case.

As to regard now to the significations of the cerebral symptoms, let us first take those resulting from the first seizure. The occurrence of this event - was as clear an index of what had taken place in this man's brain, as if we had seen the vessel, probably a small one, breaking, and letting a little blood ooze out of its normal channel and settle down somewhere in the cerebral tissue, which is thereby more or less torn, and at the same time squeezed. The comparatively deliberate onset - and the absence of coma would point to a comparatively mild lesion. It may be asked, considering the nature of the case, how did we know that blood had been extravasated, might not the paralysis be due to anaemia? Apart from the circumstances of the seizure, (which would suggest rupture of a cerebral vessel, through a vascular lesion, which proved too much for arteries weakened by disease) the coexistedness of the symptoms would be sufficient I think to negative this hypothesis. Then the renal element in the case would favour the diagnosis of clot, rather than of the plugging of an artery or the softening of cerebral tissue. As to the situation of the lesion, the left hemiplegia and facial palsy point to the region of the right corpus striatum: nor would we expect the optic thalamus of that side to have been touched in this attack as there was no anaesthesia or paresis of sensation at this time. And again we should expect only slight injury, if any, to the cerebral hemisphere, seeing that there was no impairment of the mental powers; the symptoms being confined to physical phenomena. The affection of speech, at this time, was clearly ataxic and no doubt acknowledged the same causation as the tongue deviation and the facial palsy.

But very different - was the significance of the second attack which therefore stands in sharp contrast to the first one.

The second attack came on quite suddenly, without a moments warning, and there was complete insensibility

It was moreover accompanied with convulsions. The mode of onset was therefore Epileptiform. The spasm not only affected the paralyzed limbs but engaged the right arm as well. Now as the hemiplegia was limited to one side (the left) till near death, I should conjecture, from this symptom alone, that the seizure was the result of another extravasation of blood into the right corpus striatum. The large clot found here, at the autopsy, might well, from its appearance, have occurred about this time; and to the laceration of brain tissue and the contiguous convolutions I should attribute the Epileptiform character of this attack. ~~That is~~ To say this, is perhaps to adopt the hypothesis of Dr. Hughlings Jackson, who suggests that a convulsion of this nature signifies a discharging lesion of the convolutions near the corpus striatum, probably developing ~~those movements~~ (in a rough explosive manner) those movements which are lost in the accompanying hemiplegia. Whether the implication of the right arm in this attack shews some slight lesion in the left side of the brain so transitory as to leave no trace of paralysis, or whether it was due to the spreading of the spasm from the side first affected (left) to the opposite (right) side of the body, I do not know. If the latter conjecture be true, one would expect the right leg to have been the limb next engaged rather than the arm: now the leg, as far as I know, was not affected till near the end, when the paralysis had become almost complete and bilateral, and many lesions had occurred. It may be asked, in reference to what was determined by the "post-mortem" inspection, why may not the affection of the right arm have been due to the the Effusion found in the left corpus striatum. I think the clot found in this region was too recent to have formed at this time; but supposing it to have been the cause of the convulsion in the right arm, one would not have expected it would be limited to the arm: the leg ~~also~~ of that side would have been also affected. Besides, this would demand a simultaneous double apoplexy, which, to say the least, is very rare. Again, it may be suggested that the clot found in the left cerebral hemisphere might have been the cause

Dr. Broadbent. on "Syphilitic Affections of the Nervous System"

The same objection as to time would apply here: besides a clot in this situation, if it produced a convulsion at all would cause a general and not a partial one. A slight tremor, it will be remembered occurred in both arms during the first attack. Two hemorrhages were found in the right optic thalamus. The loss of sensation, in the paralyzed side which was discovered after the second attack, would, I imagine, be due to the oldest clot in this region. The suddenness of the onset and the complete loss of consciousness points to the gravity of the lesion, to the rapidity and force with which the cerebral tissue was, doubtless, at once lacerated and compressed; and marks a more extensive effusion of blood than had before taken place. The other symptoms also bear this out. The paralysis now fell with greater stress on the affected side, & also involved the muscles of the pharynx. This symptom is open to discussion. Quoting from Dr Broadbent, "When both sides of the brain are affected we sometimes have the symptom of bilaterally coordinated muscles suffering;" and he refers to the pharynx as an instance of this kind. I do not think however that both sides of the brain in this case were affected at this time. And further, I would question the value attaching to this notion of muscles being bilaterally coordinated. Are not the muscles of the tongue and of the face bilaterally coordinated, we do not talk with half our tongue nor use the muscles of expression of one side alone, any more than we breathe with one side of the chest, and yet a central lesion will affect the tongue and face unilaterally, but the chest escapes. A more reasonable explanation of the matter is given, it seems to me, by the consideration of the line of pathological action (which is indeed a true imitator of physiological or healthy action) viz that the paralysis falls firstly and most severely upon the muscles which are most under voluntary control, or in other words, that it is the volitional (the most highly specialized) rather than the automatic function of muscles which is first to suffer. Just as in senile decay it is the last acquired or highest Bulbar ground which go first -

† Prof Sanders on "Vertical Hemiplegia of Palate in Facial Palsy". Edinb. Med. Journal. Aug. 1865

‡ Todd's "Clinical Lectures" 1856. vol I p 67.

‖ Hermann's Physiology" 1878. p. 365.

Q. Dr Hughlings Jackson on "Nervous Symptoms in Ear Disease" 1877. pp 4-5

the earliest and most automatic remaining to the last. And in this sense the muscles of the tongue and face stand in sharp contrast to those of the chest. But it should imagined that the imperfect action of the pharyngeal muscles of one side alone could not fail to cause some dysphagia. As a clinical index of unilateral pharyngeal palsy we might regard the deviation of the uvula, when this is observed, which is not often. But the explanation of this again is quite open to doubt. A crooked uvula is not a very uncommon natural defect: but if there be an alteration of the palatine arch as well, especially during muscular action, and both these phenomena are observed for the first time after an apoplectic seizure, it is but reasonable to infer that they are in some way causally related to it. But what is the diagnostic significance? This is not easy to say, for there is some difference of opinion as to the mechanism engaged. From a careful clinical investigation, Prof. Sanders, came to the conclusion that this symptom is due to an affection of the Portio Dura;† another good clinical observer, Dr. Haldane, attributes it to the same cause, but says that the deviation towards the paralized side is not always constant. Dr. Todd, in speaking of Bell's paralysis says, "in some instances the velum of the palate participates in the paralysis, and when you look into the mouth you find the uvula inclining away from the paralized side and the velum drawn to the sound side".‡ And Dr. Todd in explanation, refers to the connection of the facial nerve with the palate, by fibres running to Meckel's ganglion through the vidian. This opinion as to the nerves involved is probably correct; it is endorsed by Hermann Weber another eminent authority, Dr. Hughlings Jackson, makes the following sweeping statement: "In no case of uncomplicated facial palsy has he ever seen any paralysis of the same side of the palate", and that "any added paralysis of the palate goes almost always with paralysis of the vocal chord on the same side" and is due, he thinks, to disease of the bulbar part of the Spinal accessory nerve. And Dr. Jackson further adds that were he

† Todd's. Clinical Lectures on Paralysis. Lec. I.

to meet with paralysis of any one of the palate (the uvula with hers has no diagnostic value) in a case of Bell's paralysis he should make the diagnosis of two lesions. Since Experimental and physiological evidence is no less conflicting than clinical evidence (for the intricate connection of the nerves in question, in the persons here, makes it quite doubtful whether we are to attribute motor effects to the Facial, the Spinal Accessory or the Glossopharyngeal,) and it is a matter of no small difficulty to arrive at a satisfactory Explanation of the muscular mechanism of this part even in health; we cannot attribute to this any diagnostic value to the affection of the uvula and palate - not at least in the sense of localization of the seat of disease. And precisely the same statement may be made with regard to the state of the pupil, in hemi paralysis: the second attack left its mark in this respect -; but with what significance it is impossible to say But it was safe to draw the general conclusion that the Supervention of these symptoms indicated a deeper involvement in paralysis. The mental faculties also now began to suffer, and the aphasia was now, I think, not solely a palsy of articulation but a failure in memory as well. Indeed, ~~I should venture to say~~ the time had now come when one could no longer hesitate to give a decided prognosis, though unfortunately an unfavorable one: with regard to her paralysis, at any rate, the condition of the patient was incurable. She might have recovered from the first seizure, because of the compensatory power still in the unimpaired portion of the corpus striatum: the effects of this attack were rapidly wearing off, and (at the time of the second attack) were limited to little more than inability for the finer muscular adjustments (as in picking up small articles with the fingers) But the symptoms of the second seizure imply, I should imagine more injury to brain tissue than would admit of recovery; for, to quote Dr Todd "nerve tissue is one which is never regenerated quickly and seldom completely, so that a great or long continued lesion of its structure is not likely to be recovered from." †

† Todds. Clin. Lect. vol II. p 329.

※ Aretæi Apoplexy. Reynolds System of medicine

⁂

Ξ Ibid.

With regard to the fits which followed the second attack (and which were, on the whole, similar as kind though differing in degree) there are two points of special interest viz - as to the manner in which the paralysis affected (1) the limbs, and (2) the eyes. First as to the limbs, Both arms were now affected but with a difference. The attacks were characterized by an Epileptiform order. It is quite conceivable that the active results of old hemorrhages in the brain, such as a process of induration, a local encephalitis or the formation in the cerebral substance of a cyst or cicatrix, should produce an Epileptiform seizure without any further extravasation of blood. But from the fact that the paralysis in question seemed gradually to deepen as the case progressed, and that I never observed the transient pallor tongue biting, foaming at the mouth &c which characterize the pure Epileptic paroxism, I should imagine that the occurrence of the fits pointed to a renewed outpouring of blood, generally perhaps, in the site of the old lesions. Still, bearing in mind the Stubbins case (quoted on page 23) one cannot help being impressed by the remarks of Dr. Todd, when lecturing on a case of Bright's Kidney in which fits had occurred, "The retention and accumulation of urea and other elements of the urine in the blood in an already much vitiated state of that fluid were quite sufficient to create the irritation of the brain on which these fits depended." † But the testimony of Dr. Hughlings Jackson is of still greater significance, and he says "I know of no means of distinguishing with certainty the convulsions of uraemia and Epilepsy from cerebral hemorrhage" * and again, "A man hemiplegic from clot, with renal disease, is liable to convulsions, single or in batches, limited or nearly so to the palsied side, and they do not necessarily nor even usually point to a fresh effusion" ‡ I cannot but think that these words sound strange as coming from Dr. Jackson, who as well known attributes even the transitory and slight prodromata of approaching paralysis, to the occurrence of small hemorrhages, so small perhaps as to leave no vestige at the "post mortem". All that can be said as to the condition of the limbs resolves itself into a

Todd. op. cit. vol I p 235

† On "Rigidity and Hemiplegia". Medical Examiner. Apr. 5th 1877.

※ Todd, op. cit.

discussion of the nature of clonic and tonic spasms. ☞ I find a somewhat similar phenomenon mentioned in Todd's clinical lectures on hemiplegia. The paralysis in this case was on the left side, and the onset of renewed attacks was epileptiform. The arm, he says, would be jerked about in convulsion whilst the leg was at the same time affected with a tonic spasm. In my case, the leg was convulsed, whilst the arm was stretched out in a tonic spasm, but the other (right) arm seemed also to be engaged in tonic contraction, with this difference, that it was flexed instead of extended. This would only last for a few moments, and at the relaxation of the spasm I did not observe convulsive movements in either arm, nor in the leg that was convulsed did I detect any spasm preceding the convulsion. The rigidity did not, in fact, last long enough to allow of my ascertaining whether there was any dissipation of energy in the form of heat, as has been observed in this condition. This implies a theory; the theory advanced, I think, by Dr. Jackson, with regard to this muscular rigidity or tetanic spasm occurring in brain paralysis viz that it implies a series of cerebellar shocks, so rapid as to maintain the muscular fibre in state of continued tension, (similar, in fact to that induced by the passage of the galvanic current,) or, in other words, an "unimpeded cerebellar influx", unimpeded, because of the cerebral influence being destroyed by a paralysing lesion somewhere in the cerebrum † Dr. Todd. in speaking of "early" and "late rigidity", agreed that the phenomenon indicated "a cause which exercised at once a paralysing and irritating influence on the brain" * But he did not draw any distinction between cerebral and cerebellar influences. I think however we have come to see that such words as "irritation", used by the old writers, really stand, not so much for our knowledge, as for our want of knowledge: and it is in this respect that the thoughtful labours of Dr. Jackson are to be commended. I see, recently, that Dr. Ross, another worker in cerebral pathology, has come to the same conclusion, and independently. Still, it must be confessed

that the aetiology of tonic contraction is as obscure as its "modus in quo" - to say that it shews the muscles have "accepted the situation", is, of course, a description and not an explanation. In a pathological point of view it is generally regarded as characteristic of softening ie "ramollissement": but in the case quoted on page 23 as a "negative instance" no pathological condition was detected. This hypothesis then (which it would be out of place to discuss in detail here) might be extended, I suppose, to all cases of tetanic spasm - that occurring for a short time (as in my case) which occurs permanently in the state known as "Early" and "Late rigidity". And if it be applied to the case in question, one can understand why for instance the right arm should be flexed while the left was extended; flexion implying a less degree of paralysis: because the paralysis we suppose fell on the right side secondarily to the left, which was the side primarily and most extensively engaged. This conjecture is of course rendered doubtful by the discovery, "post-mortem", of a clot in the left corpus striatum. We may at any rate believe that the palsy which fell upon the right side towards the termination of the case, and which never compared in severity with that on the left, was in some degree due to this last mentioned lesion But the point in question, is, whether the peculiar convulsive tendencies which engaged the limbs at this time (and the similarity of which would point to a similarity of causation) were not due to something taking place in the region of the first affected (right) corpus striatum? If this were so, we may remark that the tetanic spasm occurring in these later attacks would indicate (on the above hypothesis) a further extent of involved action than when both arms were engaged in clonic spasm during the second attack.

If such considerations, as the foregoing, excite the criticism of over refinement, I would answer, that where all is so obscure and uncertain, we can ill afford to heal with contempt conjectures, which at first sight may seem fanciful. At the same time we must temper our acceptance of any proposed hypothesis by bearing in mind "the unexplained residuum" of negative

Graves' "Clinical Lectures on Medicine" 1864. pp 393-5.

†. Articles "Apoplexy" & "Cerebrum" - Reynolds System.

instances. As an illustration of this, I think it worth while to refer to a case which has been put on record. In these days of the localization of cerebral disease, so ably worked out by the patient labours of such men as Ferrier and Hughlings Jackson we are apt to turn away from "negative instances" with the reply that there must have been some causal lesion, though undetected at the "post-mortem", and again arises in our difficulty with regard to what is "organic" and what "functional". But when our informant is a man like the late Dr. Graves, who, I fancy, will yield the palm to none in accuracy of observation and devotion to scientific research, we may well hesitate before we pronounce against the evidence of the great Dublin authority. Now, in his clinical lectures, Dr. Graves mentions a case of left hemiplegia in which this condition of tetanic spasm was observed in the arm of the affected side, and continued till death. "This", he says, "combined with the hemiplegia, seemed to furnish indisputable evidence of some local affection of the opposite side of the brain, and yet," (to his great surprise and with a very confident assertion as to the case taken at the autopsy, see p 393,) "none such was detected." I will leave this case to speak for itself; only adding this remark, in the words of Dr. Jackson, "that while we see nothing, we are believers that there is nothing to be seen" + In the first attack, it will be remembered, tremor of both arms was observed, this would imply, on the hypothesis of opposed cerebral and cerebellar influences, a minor degree of tetanic spasm, which was more fully developed later on. A condition, perhaps, acknowledging the same cause as the tremor of Paralysis Agitans. It may be mentioned that a similar tremor was observed in the Dublin case first quoted.

Before quitting the subject of tetanic spasm, I should like to add that I have seen it well marked, even to opisthotonos, in a case of pure hysteria; and to quote Dr. Jackson's Experience, who says he has never known it further removed than Exceptional cases which have been either proved or

‡ Dr. Bastian "Lectures on Hemiplegia". Lan 1874.

believed to be cerebellar.

With regard to the Eyes, what was the signification of the Conjugate deviation to the right side? In the first place it is a symptom betokening gravity; it shews that the paralysis of the affected side is then complete. It is probably analagous to the rotation of animals after severe cerebral lesion (see p. 36.) * It is, I think, generally accompanied by a rotation of the head in the same direction. But in the present instance it was not the head alone, but the whole body which had a tendency to turn to the right at the commencement of a fit. So much so, indeed, that the patient was, more than once, in danger of falling out of bed. This lateral deviation of the eyes is often broken by a convulsive twitching of the ocular muscles, generally occurring simultaneously with the convulsion in the paralysed limbs, and when this happens the eyeball is jerked to the paralysed side at a slow rate to the side which is engaged in convulsion. There is, in short, a temporary nystagmus in one direction, and it is to be explained on the same ground as the convulsions which attack a hemiplegic limb. This symptom again is "locally" one, i.e. the patient is said to "look to his lesion". Now as the deviation and rotation, in this case was always to the right I could not doubt that the later hemorrhages, (or, at any rate, whatever the causes of this symptom might be,) were in preservation, like the hemorrhage of the first attack, on the right side of the brain.

— Autopsy —

On removing the calvarium the dura mater was observed to be remarkably adherent in the situation of the Pacchionian bodies, and these were unnaturally large. The arachnoid at the base had something of a opaque opalescent appearance, frequently seen in the case of drunkards, but no signs of inflammation or deposite were discovered in this regard. The basilar artery with its branches, were extensively engaged in atheromatous ~~disease~~ disease. On slicing the substance of the brain, the

puncta cruenta were very conspicuous, and apoplectic clots or the indications of them were found in the following situations
Right corpus striatum: anteriorly and approaching its upper surface a shallow circular depression, 1/4 inch in diameter, containing the remains of a decolorized blood clot, which did not fill the cavity. The posterior portion of this "corpus" had been extensively destroyed; a firm coagulum, the size of a hazel nut, evidently undergoing absorption lay surrounded by detritus of yellowish and dark colored cerebral tissue. This lesion involved the contiguous convolutions, and impinged on, but did not involve the optic thalamus of that side
Left corpus striatum: An excavation very similar in size and situation to the smaller lesion of its fellow, but containing a soft and recent blood clot, which almost filled the cavity.
Right Optic Thalamus: in the centre, an excavation, in which lay a somewhat firm and yellowish coagulum, size of a pea; just above this and rather posteriorly, and smaller in size, a more recent clot was to be seen.
Cerebral hemispheres, right side; A recent clot, the size of a small walnut, lying immediately above posterior horn of the right lateral ventricle. Left side; a recent clot, in size rather less than the preceding, situated, half an inch from the surface of the posterior lobe, above the fissure of Rolando.
There was no softening around these two clots.
There was no extravasation into the ventricles.
Heart: in systole; large and loaded with fat, the left ventricle was moderately hypertrophied, the right heart was dilated, no marked valvular disease, except some induration of the valves, which did not affect their competency. Patches of atheroma in the ascending aorta.
Kidneys; typical of interstitial nephritis, the right most advanced both much contracted, surface puckered, capsule thick & adherent cortex wasted, and in parts, apparently, quite thinned away, a few cysts among the tubules
Liver, weight 4 3/8, deeply congested ("nutmeg") & fatty

somewhat firmer under the knife, than natural, but there was hardly any true cirrhotic induration to be seen.
Spleen: congested and considerably enlarged
Stomach & Intestines: mucous membrane highly congested. I could not say whether there was any signs of inflammation in the Stomach
Lungs; congested and somewhat emphysematous.

The next case I shall give is one in which the association of symptoms clearly pointed to some organic disease within the head. The interest of a discussion of the symptoms lies more in their nature in regard to localization than in telling us the nature (pathological) of the lesion. It would be impossible to know this, in the absence of a "post mortem" examination, and this, unfortunately, was not permitted. There was nothing in personal & family history which could throw much light on the case. The patient was a carpenter by trade; lived, as far as I could make out, a temperate life; there was no reason to suspect he was the subject of Syphilis, or had any hereditary taint or dyscrasia. He was a young man of about 30 years of age, of considerable intelligence.
My description of the case will be confined to a discussion of the symptoms, and these I will in the first place enumerate in detail: —

1. Double optic Neuritis
2. Deafness of right Ear
3. A staggering gait
4. Vertigo, and some headache
5. Diplopia (slight) and nystagmus.
6. Anæsthesia of right side of face with paralysis of the muscles of mastication on that side.
7. Facial palsy (slight) of right side.
8. Hemiplegia (motor) of right side.

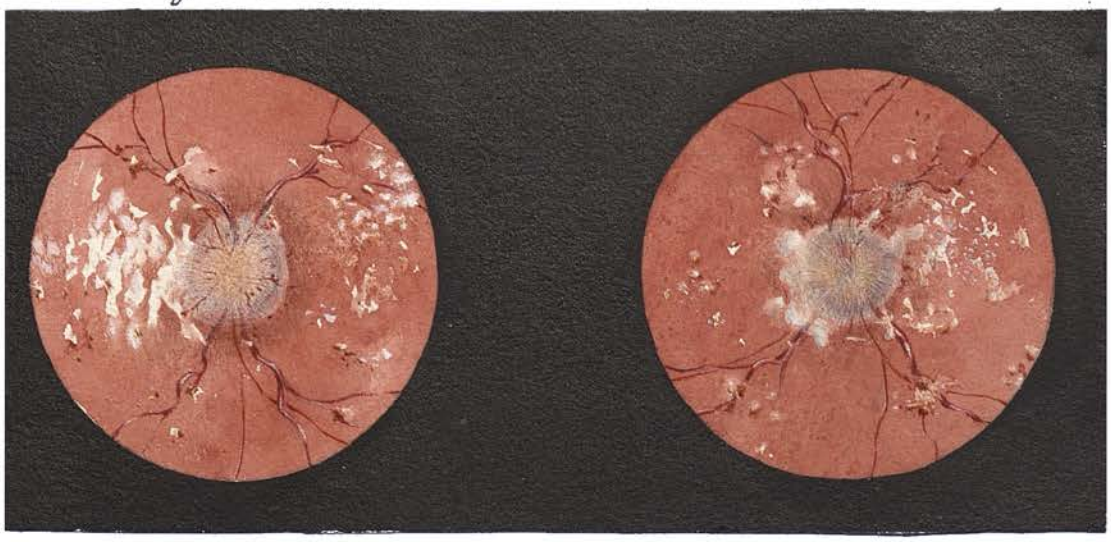
I have placed the symptoms in the order of value as indicating the kind of lesion. Given this catalogue of symptoms, the kind of lesion is tolerably clear. But, before I proceed to discuss them, a

word or two seems necessary with regard to this arrangement
In the drawing up of such a list it might appear that my
diagnosis was arrived at by an "a priori" method; whereas it is my
intention to illustrate quite the opposite method of arriving at Knowledge, viz, the inductive or true scientific method. I wish to put
the case as it would present itself in actual life to the mind of the
observer as a clinical study. I am aware of the danger of twisting
facts to suit a theory (a proceeding, only too easy, where so much,
as in medicine, rests upon conjecture) But to any one who urges
that in this arrangement of symptoms, I have adopted a pre-
conceived idea (itself really resting on data foreign to the matter in
hand) which has guided me in placing them so rather than any
other way, I would reply that the objector is partly right and
partly wrong. He is right in crediting me with a preconceived
idea, he is wrong in supposing that this prevents me using the
true scientific method - a method of induction. This is hardly
the place, however, to discuss such a matter at length, for it is more
a question of dialectics than anything else. ~~But I want to point~~
~~out~~ But, I think, it is worth while, to make the remark, that
all Knowledge is relative; that it is only possible to Know any
one thing in so far as we Know other things. That any thing which
we are pleased to call a "fact", really implies a host of notions
or theories, and is in itself a generalization. Nay further, the
man who has thought much, is least positive about facts. An
Experienced physician will, generally speaking, be less positive
in his diagnosis than are fresh men from the schools. The distinction
between fact and theory is often mischievous. There should be
no hard and fast line of demarcation between them, any more
than (to borrow a similie from another department of Science)
"faith" and "works" should stand opposed. All life is problem-
atical to the wise man; and it is to be hoped that as the world
grows wiser, it will lay aside, as it is laying aside, the fatal dual-
ism which has plagued the ancient mind and is still too often the
fetter of those who penetrate the secrets of nature. At the same

time. I do not deny the great value of drawing these distinctions for practical purposes: but solely, be it understood, as a matter of utility, and not as implying an Essential difference. In making his diagnosis then, the physician must form some idea, then a general notion (this in medical language is called a "provisional diagnosis") which serves as a sort of mental scaffolding to work upon. The above list, is, as it were, such a means for working out the general idea. To put it more graphically A man presents himself complaining of certain troubles, he can't walk straight (perhaps this is plain enough without his saying so) he can't ~~see~~ hear well, he can't hear well in one ear, he says he feels giddy and has a pain in his head, he has observed a weakness in his arm and leg, and perhaps he has noticed his face a little drawn (or perhaps the physician has already seen this while he is talking) and probably this is all he will tell you. But already the physician has made a shrewd guess. He then proceeds to examine his patient's nervous system; the results of his examination he notes down in his memory or his note book, and while doing this he has formed a general notion of the case, he has made a provisional diagnosis) in fact he cannot help drawing some sort of conclusion, and this, as ~~this~~ inevitably will guide him in his arrangement of the Symptoms for further considerations. But he has yet carefully to "go over" the case. He has got the so called "facts", but they of themselves will not guide him to the truth, he has yet to lay his thoughts side by side with these facts, before they will yield fruit. He has in short to bring all his available knowledge to bear upon the Symptoms singly and collectively, When this is done, but not till then, and often not even then is he justified in pronouncing his diagnosis.

Double Optic Neuritis. I put this at the head of the list as being the most valuable Symptom of Cerebral lesion, E.g.a tumor. The Ophthalmoscopic appearances here however were not simply confined to a "tumor disc". The retina was also, and extensively involved. It was studded (see annexed fig.) with patches, scattered on the fundus.

(right) (left)



Double Optic Neuritis with appearances simulating Albuminuric Retinitis, in a case of Cerebral lesion, believed to be, Tumor Cerebelli, and in which there was no Albuminuria.

* Lan. 1874. Vol. I. p 255.

in the right eye, there will be seen to be of considerable size, dull and opaque like dashes of white paint, in the other eye they were smaller more glistening and refractive. Now disappearance of the retina, with the exception perhaps of the dull white patches, is generally considered to be quite characteristic of renal disease. But in this case, there was no albumen detected in the urine all the time patient was under observation, and that it was not a case of Bright's disease was known by the absence of the functional and structural changes which accompany that disease, nor did the history point this way. Now then should we explain the retinitis, if it was not of albuminuric origin? Does not often a symptom of cerebral tumor, but it occasionally is. If the inflammation of the second nerve were due to some disease in the brain, I suppose it would not be unreasonable to conclude, in absence of evidence to the contrary, that the retinitis was an extension of the same effect. But as to the general question, if the ophthalmic appearances signified, in this case, brain disease such as a tumor, in what way did they signify it?

This yet a matter of conjecture as to the reason why a gross lesion of the brain, whatever its situation may be, should cause changes in the optic disc. When a tumor is far back and median in position, increased intracranial pressure by impeding venous return through the "vena Galeni", might account for a "choked disc". But then as Dr. Broadbent says "it is not even settled whether there is or is not a distinction between ischaemia of the disc — and a neuritis descendens — Till this is decided we cannot go much past the threshold of the enquiry as to the mode of production of optic changes in diseases of the brain." *Although this statement applies to a certain extent with the quotation from the same author given on page 7 I cannot but think it is but too truly indicates our ignorance on the subject. Dr. Hughlings Jackson never uses the term "ischaemic" or "choked disc", but regards the optic changes in the light of an inflammation spreading down the nerve, and suggests that it is due to inflammation around the tumor (a local encephalitis) acting

* Medical Times & Gazette 1872.

in some way reflexly on the optic nerve. It is very seldom, he thinks that a tumour causes this symptom by direct pressure.
But when encephalitis occurs we generally have intense pain in the head. There was no such symptom here however. This is a difficult question and invites further investigation.
The defect in vision, which the patient complained of, I should attribute to the state of the retina. Amaurosis is due to the change in the nerve, only when they have progressed further than in the present instance. Such patients no doubt often become totally blind before death, and this is due to optic atrophy, a sequence of the neuritis. Amaurosis may also occur in tumor cases from anaesthesia & inflammation of the cornea, when the tumor involves the trigeminal nerve.
"The optic neuritis then could tell us nothing more than the probability of some gross lesion somewhere in the Encephalon. But the patient complained of Deafness in the right Ear. Now if this were ascertained to be of nervous origin i.e. if the right portio mollis were involved, it would furnish a clue to the whereabouts of the lesion.
Before proceeding with this symptom I might say that the headache and vomiting (so often associated with Optic neuritis in brain disease) were neither of them (although they were present to a certain extent) of such a kind & to such a degree as to be of value in diagnosis.
Now deafness of a cerebral origin is a usual symptom in cases of intracranial disease, and according to, perhaps, our best English authority in such matters, Dr Hughlings Jackson, "it never results from disease of any kind in any part of either hemisphere." * Dr Jackson goes on to say, in contrasting the above two symptoms, that his impression is that "deafness does not arise from intracranial tumors, or other adventitious products, unless the portio mollis be actually involved & directly pressed on. If so the significance is altogether different from that of amaurosis from optic neuritis, as in optic neuritis the adventitious product scarcely ever involves any part of the optic nerve as it enters the skull". Again," if the fact be as I suppose deafness is a localizing symptom, whereas so far as is

* "Errors in Auditory Vertigo"
Medical Times & Gazette vol I 1877

yet known, optic neuritis occurs sometimes in many parts, probably in any part of the Encephalon". The cause of the deafness, (whether the portio mollis was involved or no) was to be arrived at by a method of exclusion; the means adopted is what Dr Gowers calls "transcranial audition", *i.e. making the bones of the skull the medium of transmission of the Rinne vibrations rather than the External air. Otherwise we could not distinguish whether it was the internal Ear or the nerve itself that was at fault, and this is just the point to be made out - The hearing can be tested in this manner by placing a watch, for instance, or a tuning fork against the forehead or somewhere in the median line of the head, or in the mouth, or against the teeth. Now if the Ear be blocked (i.e. if there be disease of the tympanic cavity) the sound is heard ~~best~~ better there than on the sound side. This is an Empirical fact which anyone can prove for himself, by closing the Ear with the finger, which comes to the same thing in this sense as the "blocking" of disease: Now in applying this test, in the case in question, and it was done not once or twice but several times, and with care, the patient (who was an intelligent man) always said the same thing viz that he could not hear the ticking of the watch (or whatever it might be) in the right Ear. This being so, and there being none of the ordinary ~~signs~~ symptoms of Ear disease, E.g. discharge, Ear ache, tinnitus (with the exception of Vertigo, indeed, which will be discussed presently) it was reasonable to conclude that there was something wrong with this man's right portio mollis; and if it was suspected that there was some intracranial disease, this symptom would help to localize it.

But - I do not quite know, as far as localization goes, which Symptom would rank first in the physician's mind, whether this one or the next, which will now be discussed.

Now the patient had a staggering gait. It must be remembered that he was also hemiplegic and had vertigo and diplopia, might not his unsteadiness be well put down to these? What does a staggering gait imply, as seen for instance, in the reeling

† Physiology 1878 p 517

× Hermann's Physiology 1878. p 504

of a drunken man. It evidently means that the balance is lost, that the body is, physically speaking, in unstable Equilibrium & in physiological language (regarding the body as a muscular mechanism) it means loss of coordination. It means therefore, or may mean, that the centre of coordination, if such there be, is at fault. Now the general result of Experiment and Observation inclines us to credit the Cerebellum with the function of coordination. Speaking of loss of coordination being referred to lesion of this part of the brain, and commenting on the discordance of clinical evidence, S. Michael Foster, goes on to say, "Still, Experimental Evidence is so strong that we must consider the cerebellum as an important organ of coordination, though we are at present unable to define its function more exactly." † And this opinion is also advanced in the latest and perhaps the best physiological text-book in the language (Hermann's) ×

In the consideration of this Symptom I may contrast the case in question, with that of a little girl who came under my notice about the same time. There was a history of a severe fall, after which some kind of convulsive seizure had occurred; the head also began to enlarge more rapidly than was natural, and the sight began to fail, so that the girl was taken to an oculist. At the time I saw her, there was descending optic neuritis in both eyes: the right disc soon became atrophic. There was a strong hereditary history of struma, and judging from the age of the patient the size of the head &c. the diagnosis pointed to a glioma of the hind brain probably located, from the Symptoms, somewhere in the cerebellum. This Symptom was the peculiar kind of walk: it was not exactly the staggering of the ataxic, nor the shuffling of the paralytic, it was more an attempt to broaden the base of support, a straddling, with the faintest suspicion of a tendency to fall backwards; reminding one in character, though not in degree, of the "repulsion" spoken of by some writers. There was obviously loss of coordination here, and the action was quite like that due to alcoholic Excess. Indeed this

* Lond. Med Record. 1878 p 247.

so-called "cerebellar reel," is to take the reel of the drunkard that I first read of in the London Medical ~~Gazette~~ Record that "one of Nothnagel's patients (suffering from cerebellar ataxy) has several times been locked up by the police, as drunk, owing to his fabric ~~gait~~ gait!" * The diagnosis suggested in this little girl's case was, then, tumor in, or of, the cerebellum; whether one could localize further is questionable; there was no headache, or other localizing symptoms; and I should doubt whether a symmetrical enlargement of the head, would point, as has been said to a median position of the tumor, viz in the middle lobe of the cerebellum, or about the tentorium, by which the "vena Galeni," would be obstructed, and the hemispheres therefore unduly distended. Six cases are on record, I have seen two such, in which there was symmetrical enlargement with hydrocephalic distension, and yet the tumour was found after death to be in one or other of the hemispheres.

But to return to the case under discussion; the patient's walk was quite of the kind described in the little girl's case; it was noticed however that when he attempted to walk straight without support, there was always a tendency to incline to one side. Whether it was more to one side than to the other my notes do not say, and I do not think this was clearly made out.

But it may be urged, this unsteadiness may have different significations all no doubt pointing to the cerebellum as the "origo", but not necessarily implying a gross lesion in that situation. Reeling may come (as Dr. Jackson has shewn) from Ear disease, but that was excluded here. Might it not be due to diplopia, for the palsy of ocular nerves is quite enough to make a patient giddy and even reel; there is such a thing as ocular vertigo; and vertigo is but an incipient reel. But I think this eye symptom would not account for it here, indeed the unsteadiness was just the same when both eyes were shut.

And as before stated it was different from the shuffling of the paralytic

* Foster's Physiology - p. 518.

† Dr Liveing on "Migraine."

and then his hemiplegia did not amount to much. Might it not be due to the vertigo.' Let us discuss this symptom. The patient complained of being giddy. Now this is a symptom of very various import. The association between giddiness and staggering is so obvious that one cannot doubt that there is a causal nexus, either between them or with some cause that is common to both. When the electric current is made to pass through the head (placing the electrodes on the mastoid processes) vertigo is produced, "objects appear to move in the direction of the currents, the eyes performing a shatching movement like nystagmus, and the body inclining in the opposite direction, and if the current be strong enough the sensation of giddiness passes into an actual reel." * And again, "All these phenomena are best explained by supposing that the current interferes with the cerebral coordinating mechanism from which result as efferent effects, the compensating movements of the body and eyes, the changes in the mechanism at the same time so affecting consciousness as to produce a feeling of vertigo". But all this may be known by observation in the experience of any individual - viz the association of giddiness and motion of the eyes when rapidly turning round. We say the movement makes us giddy, and the eyes resting for a brief moment on each object presented to view are rapidly hurried on to the next in succession, causing a natural nystagmus. The phenomenon is known as the "losing of ones equilibrium", but to speak more particularly and scientifically, that which is lost, or as it were, thrown out of gear, is the muscular sense. The sense which is the most important means we have for adjusting the body to its environment. As Dr. Livesay says "Vertiginous sensations are not rarely seen but felt, but in both the muscular sense is involved." † After speaking of the loss of coordination in animals deprived of the cerebrum (the organ of consciousness) Dr. Foster says, "Since the peculiar movements characteristic of vertigo may take place in the absence of consciousness where the vertigo being actually felt we may with security assert that

~~constitute that there is disease (E.g. a clot) on the side~~
~~of the brain towards the rotation is directed. If this be so~~

*. Foster, op. cit. p 497.

⊙. Ibid p. 517.

† Lan. 1876. vol I p. 350

the failure to stand upright and the feeling of giddiness are both concomitant effects of the same disarrangement of the coordinating mechanism". * The causal nexus is then not between the vertigo and the incoordination, but they both acknowledge a common source. Vertigo, is in fact, itself a disorder of coordination, it is on its physical side, as Dr. Jackson has pointed out, a motor symptom. That is the common cause implied here: we have spoken of an affection of the muscular sense, and we have referred to the cerebellum as a centre of coordination. But, to quote Dr Foster again, "it is probable that its functions (i.e. the cerebellum) are especially connected with afferent impulses proceeding from the semicircular canals". Fibres of the pons varolii have been traced into the substance of the cerebellum (the flocculus in particular) and the labours of W. Lockhart Clarke have now proved beyond doubt that this nerve (probably of all the cerebral nerves) is in connection with the cerebellum. If it were not for this we should be at a loss to explain why Ear disease (apart from propagation through petrous portion) should cause the symptoms of loss of Equilibrium. Menière believed that the cause of the association of deafness, tinnitus and vertigo, in the disease which bears his name (and in which also there occur sometimes obvious motor symptoms) was some derangement of the semicircular canals. He arrived at this conclusion by reasoning from the well known Experiments of Flourens. And the speculation of Goltz was that injury to this part would explain the symptoms of vertigo and incoordination. In this view Dr Wyllys Jackson coincides, and mentions cases of deafness from injury, in which there was a prominent symptom - one in particular in which the patient always felt giddy in walking and reeled like a drunken man, "so that it might be supposed that there was also cerebellar disease". † Ferrier, has no doubt that the Semicircular canals are centres of Equilibration; when one

* Ferrier. West Riding Lunat. Asy. Rep. vol III. 1873. p. 69—

‡ Foster op. cit. p. 577.

‖ Lond. Med. Record. 1878, article on Cerebellar disease.

ampulla is irritated the impulsion is away from it, thus, irritation of the left-posterior are will cause the animal to fall forwards and to the right".* In this view the ampullae form a sort of ~~[illegible]~~ peripheral sense organ for the centre in the cerebellum, of the muscular sense.

In Menière's disease a Auditary Vertigo, there is generally, as before stated, an obvious motor symptoms; that is to say the patient has often a tendency to fall, or actually does fall, to one side or the other. (Generally away from the side of the affected ear) and sometimes with very great violence. Epileptic seizures are often ushered in by a feeling of giddiness and either before or whilst falling the patient will often turn in one direction. In cases of Epilepsy with an auditory aura (not a usual thing) this is very noticeable. I cannot tell from my notes to which side this patient generally tended, but there was always a deviation, when he walked, to one side or the other. It would appear from Experiment as well as from clinical observation that the deviation, falling or rotation is as a rule towards the side of the lesion. "Section of the middle peduncles of the cerebellum on one side almost invariably give rise to rotatory movements, the motion generally though not always being towards the side operated upon - and this is accompanied by nystagmus, i.e. a peculiar rolling movement of the eyes suggestive of vertigo".† But there are many clinical exceptions on record. It is however the opinion of one Eminent authority (Dr. Nothnagel) that, "a tendency to fall always in one particular direction is to be observed when either of the middle peduncles of the cerebellum is affected by disease".‡ The lateral deviation of the eyes, and rotation of the body, which occurred in my patient with cerebral hemorrhage, ~~it~~ may be mentioned in this relation. (v. p. 24.)

In those cases of brain paralysis with Epileptiform onset where this symptom is observed, it is considered safe to

† D. Ewen. Op. cit.

conclude that there is disease (Eg. a clot) on the side of the brain towards which the movement is directed.
If this be so, one does not see why the falling in Auditory Vertigo should generally be away from the affected side. It may be said, however, that the analogy is incorrect for in the one case there is an organic lesion, whereas in Menière's disease it is put down to functional disturbance but (putting aside the question which one may well ask, and which haunts the enquirer into the Epilepsies for instance, as to the exact relative meaning of the things we call "organic" + "functional") one may remark that convulsions can occur in the paralysed side of the body the same lesion (speaking roughly) producing both palsy and convulsion; there being at the same time what Sr. Todd would have called, a "destroying" and an "irritative" lesion; what Sr. Jackson would call, a "destroying" and a "discharging lesion".
But to take a more analogous Example viz that of true Epilepsy with an auditory aura (and cases of this sort are very apt to be, and I dare say generally are, confounded with cases of Menière's disease) the reverse seems to hold. An Epileptic of this description, has, for instance noises in the right ear; immediately preceding his seizure the head sometimes turns to the right, "so as to look almost over his back," and then he falls insensible. Now, if auditory vertigo is as Sr. Gowers describes, "an Epileptiform neurosis in which the Semicircular canals, or centres of Equilibration, are supposed to be in a state of instability", & one would have expected that the impulsion should have occurred, not the reverse way, but the same way as in Epilepsies ~~of the auditory~~ in which the auditory apparatus is also evidently involved. It must be confessed that this Kind of Epilepsy is very rare. If I had not myself seen the case, quoted above, at the Edinburgh Infirmary, and observed others recorded in Sr. Hughlings Jackson's notes, I should hesitate to refer to them.

† Hermann Op. Cit. p 470.

It is true that Ferrier in his experiments produced the impulsion in the same way in which it is alleged by such careful observers as Dr Gowers to take place in Auditory Vertigo But according to Flourens the impulsion was always to the side of the lesion.

This conflicting evidence which is interesting in a clinical point of view, strikes more at the possibility of localization in these cases than at the hypothesis of the connection between the cerebellum and the acoustic apparatus.

A more serious objection to this view is the fact that "after section of both nerves (auditory) the animal does not lose the power of controlling its movements in Space, and when subjected to passive movements or to the action of a current passed through the head, it reacts as if uninjured. Moreover persons who are destitute of a labyrinth exhibit no other abnormality but deafness".† There also certain considerations in Pathology, as well as the physiological fact that the Semicircular canals do undoubtedly subserve an acoustic function, are against the hypothesis in question. The recent experiments of Cyon have led ~~to doubt its~~ that distinguished observer to doubt its accuracy, or rather to a modification of it, which it is not necessary to mention further here. Indeed it would be out of place to prolong the discussion of this question in a paper such as this - and I must confess I have not studied it sufficiently to pronounce an individual opinion. But for clinical purposes, I should incline to the view which associates the auditory Labyrinth with the cerebellum, when vertigo and loss of equilibrium is a Symptom.

But this patient had also an affection of his Ocular muscular System. He said he saw things double, but when he was looking at his tools (he was a carpenter) he did not see them double. Why was this? Possibly when looking at a near object, he unconsciously to himself closed one eye, and

1/ Living op. cit.

※ Paget "Clinical Lectures & Essays" 1875

Kinnier. op. cit. p. 95.

only looked with one eye, & inclined his head.
His diplopia taken in connection with the following
symptoms must be put down to the common cause of his
motor troubles. S. Levering says, "Equally there is
diplopia from derangement of muscular sense" * And in the
commonest instance of incoordination viz. in drunkenness,
double vision is an early symptom. It is, I presume,
exactly analogous to the staggering of the locomotor apparatus,
the stammering of the organs of speech, and the fitful twitching
of the muscles of expression. Indeed, Sir James Paget,
in some remarks upon nervousness in emptying the bladder
makes use of the expression, "~~stammering~~ stammering
with other organs than those of speech". * Diplopia is then,
I suppose, a "stammering" of the eye, and we are safe in
putting it down to some affection of the ocular motor
nerves, the sixth in particular: (Another localizing symp-
tom.) but it is more difficult to assign the cause of the
nystagmus. In the present case the jerking seemed to be
in all directions. That it is intimately connected with
the symptoms of loss of equilibration has been noticed in
the foregoing remarks. Ferrier produced it in his experi-
ments, and he describes the jerking of the eyes which is
associated with giddiness and unsteadiness as an "Epi-
leptiform affection of the cerebellar oculo-motorial centres." #
Irritation of what he calls the auditory centre, in the cerebrum
of a monkey, produced movements of the eyes to the opposite
side: and it is interesting to observe that Cyon has
also found that experimental irritation of the semicircular
canals produces ocular deviation, each canal ~~its~~ its own
deviation: Another proof of the association of the labyrinth
with the muscular sense, or rather the sense of equilibration.
But then it must be remembered that nystagmus often occurs
in cases in which we do not expect any affection of the cere-
bellum: one case, I have seen, of ataxy who died of tumour

† Physiology II. 318.

in are cerebral hemisphere : two nystagmus was very marked but nothing amiss with the cerebellum could be detected at the "post-mortem" ~~after death~~, although it was carefully looked for, as tetanic spasms had occurred before death. Then again we meet with persons who have nystagmus, perhaps have had from childhood, and yet there is no giddiness, or other associated symptoms. But these cases may well be instances in which the organism comes to adjust itself in relation to abnormal conditions. Although we cannot explain the symptoms of nystagmus more accurately, this is, at any rate, a suggestive one and not without value in the formation of a diagnosis

From the foregoing remarks it seems right to conclude that the disease in this patient was situated in the cerebellum and on the right side. But we have not yet exhausted the symptoms which are a help in localization.

The remaining symptoms comprised, paralysis of the fifth nerve and the pertho dina, and hemiplegia of the limbs, all occurring on the same side viz the right.

Let us consider, in the first place, the paralysis of the two cranial nerves.

According to Prof Bennett, paralysis of the fifth rarely occurs except in association with paralysis of the seventh (facial) † and in our most recent and reliable textbook in medicine (Bristowe) I observe the statement that the fifth is generally to some degree involved, (as well as the facial) in hemiplegia

The symptoms in this patient which chiefly attracted my attention as implying an affection of the trigeminal, was the weakness of the masticatory muscles of one side : the loss of sensation was not very marked. There was no distortion of the countenance, at least not when at rest.

In combating Dr. Todd's erroneous opinion, that it was the fifth and not the seventh that was involved in facial hemiplegia, Prof Sanders says. "He" (Dr Todd) "says nothing of falling or obliquity of the lower jaw, which that

† . Lan . 1865 vol I.

1. Bell, "Exposition of the Nervous System" 1824. p 164.

paralysis would certainly occasion". Now this was not seen in the present case; one should expect it. For the lower jaw does not owe its apposition to the upper jaw solely to muscular action: it is hardly correct to speak of it as being thus "suspended". The function of muscles anywhere has reference to motion, and is, in fact, a question of dynamics, not of statics. For knitting the frame together there are other means, such as ligaments, not to speak of other circumstances in the anatomy of the face. This remark is intended, however, to apply to the symptoms in question viz the falling of the jaw on one side, and that too while at rest (in which sense alone I understand the expression) That the jaw ~~th~~owes its apposition chiefly at rest, to some extent, to muscular action no one will deny; for the dropping of the jaw in sleep and after death could mean nothing else. Sir Charles Bell (whose opinion on this matter Dr Todd has adopted) mentions the hanging of the cheek and angle of the mouth in cases of ordinary hemiplegia or paraplegia, believing this symptom to be due to affection of the trigeminal: does it not seem strange that it never occurred to this acute observer who made himself master of the mechanism of expression, that that is done by disease, finds its counterpart in health in certain states of mind. For we are apt to say, for instance, of a person in trouble, as ~~for~~ on the receipt of some bad news, that "his countenance fell", This change in facial expression no one would have been more ready to ascribe to the facial nerve than Sir Charles Bell. But then it may be said he drew his conclusions not only from clinical evidence but from experimentation on animals as well. There are two or three remarks to be made here. Firstly, it is well known that to argue from the animal to the human being is often fallacious, and this might make all the difference in the case in question. I can imagine that the long prognathous jaw of the ass, the animal

⁂ Redl. op. cit. pp. 115. 116.

chiefly used in his experiments by Sir Charles Bell, ※ would be much more likely to fall, as the removal of muscular support than in the case of man, and then again section of the fifth nerve on one side is a much more serious affair, for the muscles supplied, than ever happens, I imagine, in central disease which affects cranial nerves. Then it must be remembered that Sir Chas Bell resorted to vivisectional experiments after his own mind had been made up, and only for the sake of proving to others what to himself, he says, was in need of no further proof.

I should not therefore expect partial palsy of the fifth, in man, to produce any thing more than weakness of the Temporals, Masseter & Pterygoids of that side, and which would not affect the pose of the face while at rest: and only to be ascertained when the muscles were put in strong action. It was only when I made my patient clench his teeth firmly that one could detect the difference in the stiffening of the muscles of the two sides. But it was then so obvious that no one could mistake it.

It seems strange that D. Todd, who also remarked that the palsy of the fifth was always only partial, in such cases, should have attributed to this, as the chief cause, the facial phenomena that often occur in hemiplegia.

The facial paralysis in this case was of the right side, and was slight in degree, being entirely confined to voluntary and forced actions of the muscles of expression: the usual incomplete facial paralysis of hemiplegia. The orbicularis palpebrarum being scarcely, if at all affected. Of course in such slight palsy there was no alteration in the natural pose of the face, no "falling" of the cheek, from this cause. I did not observe any tongue deviation.

* Lan. 1876. vol II. p.80.

The results of testing the senses of taste and smell were equivocal, as indeed is often the case, even when the patient is intelligent enough. The paresis of the fifth would necessarily vitiate any conclusions drawn as to the special nerve apparatus of these senses; and in the case of taste, where there is this triple association of paralyses the problem becomes too complicated to be of any value in diagnosis.

The affection of the fifth would suggest some impairment in common sensation in the cornea, the Schneidman membrane, and the mucous membrane of mouth and tongue, but this was not definitely made out here.

The partial paralysis then of the right trigeminal and facial nerves served to corroborate the conclusion that the lesion was in the right side of the small brain, and interfered with these nerves probably in their intracranial course. But there was right hemiplegia as well.

Now it will be observed, if we have localized correctly, that the right sided symptoms, (I refer particularly to the three last mentioned) are on the same side as the lesion in the brain. This is very uncommon. The evidence on this head is derived chiefly from clinical experience.

Dr. Brown Sequard mentions a case in which, at the "post-mortem" inspection, a tumour was found to occupy one side of the pons; extending also into the cms cerebelli of that side: during life the following symptoms were observed - hemiplegia with facial paralysis of motion and sensation, all on the same side as the lesion in the brain. # In this respect I may quote the authority of Dr. Hughlings Jackson (which is always trustworthy) who gives out as his opinion that a lesion in this

situation viz. in the region of the middle crus or peduncle of the cerebellum on one side will not only account for these symptoms, but that in no other case does he believe that hemiplegia is to be attributed to a lesion occurring in the same side of the brain. Bearing in mind, then, the results of experimentation on this part of the brain - and Dr Yorke-Davies testimony, quoted on page 36. I concluded that the lesion was, some adventitious product, probably a tumour or cyst, of, or pressing upon, the right crus cerebelli and flocculus, involving the trigeminal, portio dura and abducens nerves, and perhaps the portio mollis, in their intracranial course. It must have been limited in extent, and not deeply situated, or else the hemiplegia would have been on the other side. This is of course only conjecture, as this patient did not die till after he had passed from my observation, and no "post-mortem" examination was then allowed. There was therefore no verification of our inductive reasoning). But I think the facts detailed above favour this conclusion. No convulsion occurred throughout the case.
