A Critique on the Diagnosis of Cerebral Lesion with ophthalmoscopic illustrations.

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An accurate diagnosis in cases of brain disease is, I should think, one of the most difficult problems in medicine. The description and discussion of cases and instances in illustration of this matter generally commands the attention, as much perhaps, for the speculative interest implied in them, as for their bearing on practice. For it must be admitted, respect it as we may, in a practical point of view, the cases of nervous disease are generally speaking, of greater interest to the pathologist and speculative inquirer, than to the therapeutist or the physician in ordinary 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 any remarks to a very few, only two or three, but those 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 as 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 external appearances may be. The signs of inflammation of the optic nerve, which the ophthalmoscope enables us to detect, are considered by medical authorities to present a most valuable index, perhaps our most reliable one, in this diagnosis of organic cerebral lesions. Yet the two main cases recorded in this paper, and I refer to theirs incidentally,
show how mistaken we may be, if we find our faith in any
symptoms, however trustworthy in itself.
Nothing shows 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 shall 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. The 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.
Once this man had found himself unequal to his work,
and fell so far from well as to induce him to seek medical aid.
When I saw him, he was complaining of what he called
"bilious attacks," characterized by headache, thinness of sight,
vomiting and general malaise. But the man's aspect and
manners, the unhealthy pallid hue of his skin, and the
sunken, deepened expression, the disorder and disordered
lives.
On examination I found some increase in Cardiac disease
outside the nipple. The first sound was thumping but without
fruits, the second (aortic) was accentuated and reduplicated
and the heart in pulse hearing. The arteries were pulse
over-drawn and tortuous, indeed remarkably so for a man of
forty. The pulse was hard at first, and observed a well marked
flow in the upper segment of the pulse. I was not surprised
therefore, to find that in addition to a laborious occupation
his health had been subjected to the strain of restless living,
with free indulgence in alcoholic and other excesses. He had
had an 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 minute rales.

In addition to this there was some fulness of the liver, with slight tenderness on percussion, though not amounting to much. The spleen could not be felt. There was no ascites.

Peterson had been jaundiced, but was not so now.

On examining the urine, I found it slightly albuminoid (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 1.015, seldom rising above 1.016, and the amount passed was slightly in excess. The urine was pale in color, acid in reaction, and sparingly insoluble. There was some frequency of micturition.

The bowels were sluggish.

This little could be gleaned from the patient's history was important in its bearing on the case. His father, patient's son, had died of asthma and his mother had paralysis (presumably from hemorragia). The offspring of such parents could hardly escape some predisposition to arthritis 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 chest falling in the first place, on the abdominal viscera, stomach, liver and kidneys, the heart and head suffering secondarily, and that the agency 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 at first such as would one would expect to find in chronic Bright's disease, the number of white and brilliant
Double Optic Neuritis, and the appearances characteristic of Albuminuric Retinitis in a case of Tuberculous Bright's, in which there was no lesion in the brain besides hemorrhages.
Spectacles in the vicinity of the macula, the nearly character of the fundus around the disc, and a few hemorrhages, chiefly linear and pointing to a 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 this time when the annular swelling 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, indeed, 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. Nor was that all, there were other symptoms in favour of a cerebral disease. The headache, of locomotor a character, and the urgent bilious vomiting are accessory symptoms which may be enigmatically in establishing a diagnosis of cerebral lesion. But these symptoms were of value, must be—certainly characteristic. The headache which is diagnostic of organic brain disease is generally pretty constant (sometimes, protracted) and is of a severe kind, or merely a sensation of weighty fullness; and then it is often localized in an unusual situation, such as at the back of the head on 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 cause of the digestive troubles in the dilated 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 suggestive of it: for we have it as an authority of one of the best-clinical observers,
Merchand as "Hypatic Divagaments," 1874, p. 111.

* Say, as "Meadows," 1874, p. 229.
(Dr. Marchionini) that neuralgic headache occurring in connexion with contracted kidney, is sometimes so severe that were there once he has known such a case diagnosed as one of cerebral tumour; and Dr. Marchionini 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 or advanced age.* And similarly, Dr. Humphry Jackson lays great stress on the necessity of examining the mind in all cases of suspected cerebral disease.

At the same time bearing in mind the fact that in cases of acute 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. Nor again (to do justice to this symptom) would the fact of headache being in an ordinary site (i.e., the forehead in 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. Humphry Jackson) be referred to all parts of the head and exactly simulate those which are of less serious origin," so difficult it is to arrive at certainty, and so much a matter of probability, is this science of medical diagnosis!

Then as to the vomiting: this was hardly of the purposeless character which is supposed to indicate an cerebral origin.

For this patient often threw up his meals, and was plagued of nausea. The sense mucous membrane was clearly affected. The tongue was coated, and the teeth foul; and worse than this, there was the redness at tip and edges, with congestion of the fungiform papillae, which Dr. Marchionini 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 whenever, this structural lesion might be, he was undeniably suffering from some functional derangement.
of the digestive system. The biliary 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, cholecystitis, and does not in itself point toward liver abscess as "fuso etrigino-nali". In every where vomiting is rarely general, 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 pyrexia. The stomach will often vomit, as it were, for no reason, by the ingestion of food before vomiting is induced, and then when the mechanism is once set among the nervous system perpetuates the unfortunate tendency of purposeless retching and vomiting of nothing but bile and gastric mucus, forced up by the action of abdominal impressions. And indeed when so richly 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 symptoms might fairly be accounted for by other than a cerebral explanation. The liver was evidently swollen and congested, with the liver, spleen, and liver, of all the conditions of cirrhosis (the patient had been accustomed to drink spirits "neat".) Might it not be true that pressure act mechanically to cause vomiting, in the same way as it has been suggested a full stomach will, though just as portent, yet in a hand 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 celiac lumen of the ganglion. Or again, the vomiting might be due to a loaded portal system, affecting the gastric and gastric hunger membrane with edema. This edema, again, might be traced more to the renal veins to the hepatic disorder. In the present instance however there was no evidence of edema anywhere else. Vomiting, again, is a common symptom of renal disease, in a toxic point of view, owing to the
advanced, or at any rate, lithaeic 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 large reflexes neglected and chronic gastric irritations, which the man was probably suffering from (inter alia), might, we would suppose, be quite enough to account for the vomiting. Taking these facts into consideration, therefore, I concluded that this symptom also must be ascribed from any cerebral connection.

Wise we then, in like manner, to refer the diminution of sight of核查 the patient complained, to his disorder of nutrition and digestion? Now, as far as the diagnosis of a gross 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 by quite other means viz., through the ophthalmoscope. Besides this, we cannot depend on an anaemia as being a certain indication of optic neuritis. At this time when I first saw the patient, the neuritis of the right eye was more advanced than in the left and yet applying the usual ophthalmoscopic tests he seemed to be equally ill with both eyes; so that it was useless to attempt to attribute the diminution to the inflammation of the optic nerve, as this stage of the case. When the disease in the right eye had advanced as it did before death, catachysis of the disc, there was of course total blindness in this eye, and the cause was obvious. But it will be noticed that when ophthalmoscopy, optic neuritis may exist even in a man of degree with no defects of vision (the patient being able to read the smallest Snellen's letters) thus misleading both physician and physician unless the ophthalmoscope be used; and as this influence may cause, 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
Seeing that the patient was the subject of renal incompetency, probably in an advanced degree, I inclined to describe his thin and imperfect vision, like his headache, to this, as a general cause: especially as this defect in sight kept varying from time to time and was accompanied with "muscui volbantes". Whether it was especially due to the albuminous excretion, or to the hemorrhages shed in the fundus or to the condition of the vitreous, or to the generally unclean condition of the blood (to any of which it might be due) I do not pretend to say.

If then the central origin of the headache, vomiting, and the dim eyesight was as improbable as the foregoing seems to show, what should we say to the double optic neuritis, the symptoms, which, in this respect, I called the most prominent of all? Could this stand alone as being sufficient in itself in arriving at a diagnosis of cerebral lesion? The sequel of this case proves that it could not.

This affection of the optic nerve, which can come and go with so little apparent inconvenience to the patient, is of such interest to the physician as to much practicioners, but for what it may reveal as a symptom of intra-cranial disease. The diseases of the retina are, as Sir W. Berkeley remarks, the diseases of the brain. And this may well be so because the posterior choridea of the eye is originally formed by a cyclotropium of the anterior ciliary vessels, the lining membrane of the sub-choroidal becoming the choroid and retina. As a matter of clinical experience, it has been found that double optic neuritis is the best symptomatic of almost disease of the brain (such as a tumour or an abscess) but that in itself, in the absence of other confirmatory evidence (e.g. effusions of the cerebral vessels in the brain abscesses) it must not be held to be absolutely decided. What then is the meaning of this neuritis here? It did not arise...
* John T. Duckett on "Newer Symptoms in
Syphilis," Journal of Medical Science 1875
against brain disease, what did it show?"

If we give up the diagnosis of gross cerebral lesion, this would exclude syphilis (which in spite of the patient's denial and the physician's inability detected always lurks in the back of a man's possible cause). That is, we say, is the true that "syphilis causes this symptoms as because it is syphilis but because it generates the conditions which causes it for tumors." Now any syphilis forms a granula occurring in any favorable site, its sympathetic place in the bones, capable of producing inflammation and swelling if 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 the characteristic affection of the fundus the disseminated choroiditis of syphilis.

But there was nothing of this kind here. And a tumorous may and purpura usually, does not affect the retina not so much by acting mechanically like a foreign body as by forming local inflammation around it, which, creeping down the nerve, appears as "restitution descendens." Was there then any evidence in this case of inflammation in the brain apart from tumors? Now in spite of St. Bacchi's authority quoted above one is inclined to think that might not syphilis cause optic neuritis by setting up as it is known to do, meningial inflammation of the brain. The suggestion is favored by some recent Roentgen worker's, which I don't quote. He says, "No specific line of demarcation can be drawn between cases in which there is extensive inflammation in the meninges (of the brain) and those in which the worst process results in the formation of distinct tumors." And again "If a syphilitic tumo is present and is not complicated with meningitis, it often in the retina is to produce a choked disc, as the result of pressure interferring with the veins, return from the retina. On the other hand, syphilitic meningitis of the anterior type he found we may have true optic neuritis from the spreading downward...
* "Pathology, Anct. of the Nervous System," 1874.
D. Claydon, pp. 145-6.

† M. Fox, "Pathology, Anct." p. 389.
along this nerve of the meningial inflammation." *

Still the same negative objection could apply, as stated above.

If there were any phthisic inflammation of the brain, we should expect to find some evidence of the nerves in their irritation caused at the base of the brain being surrounded and involved in inflammation through. But no such evidence was forthcoming here. Then again, according to Dr. Brodie's, the existence of phthisic susceptibility, as distinct from meningial inflammation, has been very much doubted, and it seems more likely to lead to eye symptoms.

Are there any acute meningitis for 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 the disease is often not much accelerated; but if under-accelerated it must be not more slow but abnormally slow, and irregular as well, and such was not the case here.

Was the patient had any cerebral hemorrhage before he died? But from the albuminuric alterations in the urine cerebral hemorrhage seldom leads to any change in the fundus; but optic neuritis is occasionally a sequela of it. * But there was no reason to suppose he had it; he could give no history leading one to suspect it, and he was tolerably truthful and intelligent. The possibility of abscess in the brain, acute a chronic, was negatived by the absence of other signs of gross cerebral disease, as well as other special symptoms of such disease (anemia, anaesthesia, discharge, delirium, giddiness, to the signs of base disease). Therefore this does not prove that the man had no abscess in his brain, for an abscess may be quiet throughout life, as Dr. Willis says. "A man with changes of pus in his brain may continue to do good
Ophtho Nemicus with haemorrhage is a severe case. Plumbium

For. P. cit. p. 293.
mental work, and have full use of her hands." But only proves that the optic neuritis is not sufficient evidence 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 type, as I do not think the association is very common, and the appearance is almost exactly like a "tumour disc".

Anamoso's is sometimes said to be caused with lead poisoning or the symptoms of optic neuritis; and Dr. Meyer has pub. in read two cases occurring in his father of bleeding lace with the aid of white lead. In one, complete atrophy came on in four months; the other was similarly affected. The case of the same patient I have been recording was beside the one from which the neuritis began over the leg, and of course the association might have been accidental. Still, lead seems to have a special affinity for nerve tissue, and other effects on the nerves besides the are which suffers most commonly, namely, (muscologial) one does not see why the second nerve should not be affected. The suggestion of plumbism is confirmed by the known result of its influence between the effects of lead poisoning and the symptoms of lithiaosis (in this without kidney disease). The introduction of lead into the system will, as a matter of facts, arrest oxidation and eliminated of nitrogenous waste, thus causing a true lithiaosis, with all its after-standards of functional and that change in heart- and kidney, and high arterial tension. Now the man's case quite answers to this description. Was there then any evidence of lead poisoning to account for? I cannot say that I could glean from the history anything that was at all conclusive, and (not unmeaning & therefore of the occasional insidiousness of these affairs).
in the absence of its more obvious and usual manifestations, I considered this idea too problematical to be of much weight in diagnosis.

Futile inflammatory attempts to attack nerves allowed alone the body, in a precarious sort of fashion, the I never heard of optic neuritis ascribed to this as a cause; and I must confess I do not know who had occasioned it in the present instance, unless it might be from the alcohol excess. The patient was not much of a drinker. There was therefore no sufficient evidence in this case to establish a central diagnosis.

I concluded the case was one of functional derangement of the brain and liver, resulting from stimulation 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, in their correlation as cause and effect.

This patient had been the victim of disease of more than one import, and it was not of any of these affections that he died. As the sequel with these, he rapidly sank through the changing of vessels. He died because his vessels had been weakened by disease. Had he lived longer, an organic disease of the liver, which I suppose, was at the point of developing would have advanced, with an aggravation of abdominal symptoms and the superficial, perhaps, of anaemia and serious inflammations which never appeared throughout the case.

Before detailing the progress of this case, I shall refer to another case in which analogous nature, and which suggested at least difficulty of mind. A man of middle age applied at an ophthalmic hospital. He had degenerative, amaurosis, and was also found to have albuminuria, in its coming, and other digestive troubles. No doubt was entertained as to the nature of this
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 chemicums, rest, and dieting. The case did not admit of anything more than alteration.

Then the patient one day had a fit. This was what was to be expected. When hemorrhage occurs in the retina and from the nose in a man, then, proof 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 hurled the case to a final issue.

From this time, therefore, the case had entered upon a new phase: for it was now a case of pure cerebro 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 come 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 uncooked, with the exception of a slight trembling in both arms. On seeing him immediately after the fit, I found him lying on his back, having the hand, he told me what had happened in quiet an intelligent manner, but his utterance was thick and husky. The face was slightly drawn & shrunk, the tongue a little nodular and deviated to the left—this was in that case, an incomplete facial palsy of Bell's type, 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 remained 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 palpebral 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 cornea 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, semicoma to condition. Several fits occurred and in short intervals occasional involuntary spasmoded contractions 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 on, at rest; but the right leg was thought to be not much affected.

Towards the end there was conjugate deviates 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 unnecessary 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 this case.
It was moreover accompanied with convulsions. The much-g"lassed was therefore epileptiform. The spasm usually affected the paralysed limbs but engaged the right arm as well. Now, as the hemiplegia was limited to one side (the left), the near death, I should conjecture from this symptom alone, that the Lesene was the result of another hemorrhage of blood into the right corpus striatum. The large clot found here, in this autopsied subject well, from its appearance, have occurred about this time; and to this section of brain tissue and its contiguous convolutions I should attribute the epileptiform character of the attack. To say this, is perhaps to adopt the hypothesis of Hjellming Jeklem, who suggests that a convulsion of this nature signifies a changing lesion of the convolutions near the corpus striatum, probably developing them (in a rough equivalent manner) those movements which are lost in the accompanying hemiplegia. Whether this implication of the right arm in this attack there was some right lesion in the left side of the brain, so manifesting as to leave us trace of paralysis, or whether it was due to the spreading of the spasm from the site 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 like near the body, when the paralysis had become almost complete and bilateral, and many lesions had occurred. It may be asked, in reference to this was determined by the "post-mortem" inspection, why may not the affection of the right arm have been due to the the spasm 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 on the right arm, one would not have expected it would be limited to the arm; this leg 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 hemispheric might have been the cause
S. Bradburn's "Lymphatic Affections" to be printed soon.
The same objection as to time would apply here besides a difficulty in this situation, if it produced a convolutional ataxia would cause a sudden and acute partial amnesia, it will be remembered occurred in both cases during the first attack. Two hemorrhages were found in the right optic chiasma. The loss of sensation on the paralyzed side which was discovered after the second attack, would, I imagine, be due to highest 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, paralysed, upon, lacernated and emasculated, and made a more intense effusion of blood than has before taken place. The other symptoms also bear this out. The paralysis was felt with greatest stress on the affected side, it also involved 135288 the muscles of the pharynx. This symptom is of great importance to discussion. Citing from Broadbent, "When both sides of the brain are affected we sometimes have the sympyotism bilaterally coordinated muscles suffering," and he refers to pharynx as an instance of this kind. The case has since been 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 must take with half our tongue how use these 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 true by the consideration of the line of pathological action (which is indeed a true imitation of physical action in a natural action) viz that the paralysis falls firstly and most severely upon the muscles which are most under voluntary control, a natural result that it is the volitional (the most highly specialized) rather than the automatic function of muscles which is the first to suffer. Just as in a smile decay is the case acquired a higher substrata ground which go first.


A. H., Dayman in "Nervous Symptoms in Ear Disease," 1877, pp. 4-5.
the earliest and most-automatic remaining to the larynx. And it is this sense the muscles of the tongue and face stand in dignity to those of the chest. But it should be observed that the imperfect action of the pharyngeal muscles of one side alone could not fail to cause some dysphonia. 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 symptom 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 ultimate significance? This is not easy to say, for there is some difference of opinion as to the mechanism engaged. From a careful clinical investigation, Dr. Sanders, came to the conclusion that this symptom is due to an affection of the submaxillary, and this good clinical observer, Dr. Hal dane, attributes it to the same cause, but says that the deviation towards the paralyzed 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 paralyzed 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 the fibres running to Meckel's ganglia through the chorda. This opinion as to the nerves is "doubtfully correct;" it is contradicted by Hermann. But another eminent authority, Dr. Hughlings Jackson, makes the following sweeping statement - "In no case of uncomplicated facial palsy has he seen any paralysis of the same side of the palate," and that, "in every case of paralysis of the palate goes almost always with paralysis of the vocal chord on the same side," and so it, he thinks, to disease of the bilateral party the spinal accessory nerve. And Dr. Jackson further adds that one he
† Todd's Clinical Lectures on Paralysis. Sec. I.
to meet with paralysis of one end of the palate, (the uvula
end lends no diagnostic value) in a case of Bell's paralysis,
he should make the diagnosis of two lesions. Since experi-
mental and physiological evidence is no less conflicting
than clinical evidence (for the intimate connection of the
nerves involved, with the facial nerve, 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 mechanisms of this
part even in health; we cannot attribute to any diagnostic
value to the affection of the uvula and palate—nor at least in
the case of localization of this cause of deafness. And precisely
the same statement may be made with regard to the affecting the
nervous, unilateral paralysis: the second attacks left its mark
in this respect; but what significance it is impossible to say.
But it was safe to draw the general conclusion that the higher
ventures of these symptoms indicated a deeper involvement in para-
lysis. The mental faculties also now began to suffer, and the
aphasia was now, I think, not only a palsy of articulation
but a failure in memory as well. Indeed, it had been
the time ha had once when we could no longer hesi-
tate to render a decided prognosis, though unfortunately an unfavor-
able one; both regarding to his paralysis, at any rate, the condition
of the patient was incurable. He might have recovered from his
first seizure, because of the compensatory power still in the unin-
jured portion of the upper structure; the effects of this attack
were rapidly passing off, and (at the time of the second attack)
were limited to little more than in ability to fix muscular
adjustments (as in pretending small articles with the fingers)
and the symptoms of the second seizure largely, I should imagine
were due to brain tissue than would admit of recovery; for,
I quote Dr. Todd "nervous tissue is one which is never regenerated
quickly and seldom completely, so that a great a long continued loss of it, if it occurs, is not likely to he recovered from."

* Alexander Apopleg. Reynolds' System of medicine

II Ibid.
As to 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 pares- 
sis affected (1) the limbs, and (2) the eyes. First as to the limbs, 
both arms were more affected but with a difference. The attack was 
characterized by an epileptic onset. The quite unaccountable fact of seizures 
of eyes, or cecatix, should produce an epileptic 
attack without any further hemorrhage of blood. But from the 
fact that his paralysis in question seemed gradually to deepen 
as the case progressed, and that I never observed this transient focal 
epileptic bising, forming at the month to which characterize the pure 
epileptic hemorrhage, I should imagine that the occurrence of the 
fits pointed to a renewed outflow of blood generally, perhaps 
the site of the old lesion. Still bearing as under, in a well 
stroke (quoted on page 23) one cannot help being impressed by the remark 
of Dr. Todd, then lecturing in a case of shaft, vicinity in which fits 
had occurred, "the retention and accumulation of aery and blood 
elements of the brain as the blood in an already much diluted state 
of that fluid were quite sufficient to create the irritation of 
the brain archists have fitted depend." Dr. Hills testimony of 
Dr. Jackman is of that great significance, and to day 
I know of no means of distinguishing with certainty, the cerebro-
spinal fluid from cerebral hemorrhage and again, "a man hemiplegic for 5 years, with renal disease, liable to haemorrhage, single or multiple, limited to nearly 80 to the palpated 
side, and then, do not necessarily mean usually point to a 
false opinion." I cannot but think that these words sound 
strange as coming from D. Jackman, who as well known 
attaches some the haemorrhage and cerebral hemorrhage, to denote 
perhaps as certain no vestige of the true disease. All that can 
be said as to the condition of the limbs resolves itself into a
Tod., op. cit. vol I p 235

†. *Rigidity and Implegmus* - Medical Examiner, Sep. 5th, 1877.

‡. *Tod.,* op. cit.
discussion of the nature of clonic and tonic spasms. I found a somewhat similar phenomenon mentioned in Todd's clinical lectures on hemiplegia. The paralysis in his case was on the left side, and the result of repeated attacks was epileptiform. The arm, he says, would be jerked about uncontrollably while the leg was at the same time affected with a tonic spasm. In my case, the leg was involved, while the arm was stretched out in a tonic spasm, but the other arm seemed also to be engaged in tonic contractions, 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 any violent movements in either arm, nor in the leg that was involved but I detected any spasm preceding this convolution. The rigidity did not, in fact, last long enough to allow of any ascertainments whether there was any dissipation of energy in the form of heat, as has been observed in this condition. This implies a theory that the theory advances, I think, by Dr. Jackson, with regard to the muscular rigidity or tonic spasm occurring in hemiplegia. It is that it implies a state of cerebellar obstructions, superposed as to maintain the muscular fibre in state of continued tension, similar in fact to those induced by the passage of sluggish current on the chorda, an "unpedaled cerebellar flux", an unpedaled, because of the cerebellar influence being destroyed by a paralyzing lesion elsewhere in the cerebrum & Dr. Todd isspeaking of "early" and "late rigidity", agued that this phenomenon indicates "a cause which excited at once a paralyzing and irritating influence on the brain." But he did not draw any distinction between cerebellar and cerebrospinal influences. I think however he means to say that both were "irritative", used by two 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 authors of Dr. Jackson are to be commended. I see, recently, that Dr. Roes, another writer on cerebal pathology, has come to the same conclusion, and independently.
that the etiology of acute encephalitis is as obscure as its "modus iniitio" - to say that it shows the muscles have "accepted this situation," is, of course, a description and not an explanation. In a pathological point of view, it is generally regarded as characteristic of encephalitis "varioliformis." But as this case quoted on page 23 as a "negative instance" its 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 tetanus. Speaking in general terms, this occurs frequently, as in any case which occurs from the state known as "early" and "late rigidity." But 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.

It was 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 close union of the left corneal striatum. We may at any rate believe that the paralysis which fell upon the right side towards the terminal period of the case, was never compared as severity with that on the left, as in some degree due to the last-mentioned lesion. But this point in question is, whether the peculiar convulsive seizures which engaged the limbs at this time (and the instance, of which) could point to a similarity of causation? I am not sure to a similarity of causation. I am not sure how much place the region of the fest-effecting (right) corneal striatum of this were to, we may remark that the tetanic spasms occurring in these late attacks would indicate (and the above hypothesis) a further extent of involved action than when both arms were engaged and the right during the second attack.

If such considerations as the foregoing, excite the criticism of over-refinement, I would answer, this where all is so obscure and uncertain, we can ill afford to have the feeblest conceptions, which at first sight, may seem patent. At the same time, we must temper our acceptance of any proposed hypothesis by bearing in mind the unexplained residuum of negative.
James' "Clinical Lectures on Medicine" 1864, pp. 399-5.

instances. As an illustration of this, I think it well while to refer to a case which has been put on record. In those days of the localization of cerebral disease, toably worked out by the faithful labours of such men as Ferre and Heisterbach, we are "kept to turn away from ingrafted instances," as he then says. There must have been some causal lesion, though undetected at the "post-mortem" (and again comes in my difficulty with regard to what is "organic" and what "functional") but when an 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 terminated the death. "This," he says, "combined with the hemiplegia, seemed to furnish indisputable evidence of some local affective of the opposite side of the brain, and yet, "to his great surprise and with a very confident assertion as to this case taken at the autopsy, i.e. p. 293, none such was detected." I will leave this case to speak for itself, only adding this remark, a word of Dr. Jackson, "that whilst we see nothing, we are believes that there is nothing to be seen." In this first attack, it will be remembered, trouble of both arms was observed, this would imply, in the hypothesis of opposed cerebral and cerebellar influences, a minor degree of tetanic spasm, which was more fully developed later on. It is interesting perhaps, acknowledging the same cause as the hemiplegia of Sydenham-Lefranc. It may be mentioned that a similar hemiplegia was observed in the Dublin case just quoted.

Before quitting the subject of tetanic spasm, I should like to add that I have seen a well-marked, even to slight hysterics in a case of pure hypertonia, and to quote Dr. G. Lee's case, who says he has never known it further to extend, as exceptions which have been either proved or
believed to the cerebellum.

With regard to this type, what the significance of the
conjugate deviation to the right side? In the first place it is
a symptom betraying gravity; it shows that the paralysis of
the affected side is then complete. It probably analogous 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. In such a case,
indeed, that the patient was more than once in danger of falling out
of bed. This lateral deviation of the eyes is often prostrated by a
violent twitching of the ocular muscles, generally occurring
simultaneously with the twitching in the paralyzed limbs,
and when this happens the eyeball is forced to the paralyzed side
and that side which was engaged in the violation. Then
it is, in short, a temporary extraneous viscerotonia, and it
is to be explained as the sign of the attack, as this convulsion which
attack a hemiplegic limb. The symptoms against is "locally" are,
"ictal" patient is said "look to his lesson." Nor as
a deviation and rotated, in this case was always to the right.
I could not doubt that the later hemorrhages (or, actually rate,
whetned the causes of this symptom might be) were in favor
at the hemiplegia of the first attack, as the right
side of the brain.

--- Autopsy ---

On removing the brain and the dura mater was observed to be
affected adherent to the situation of the pachymeningeal bodies,
and these were unnaturally large. The aneurisms of the case
had a number of transparent, opaque appearance, frequently
seen in the case of hemiplegia, but no signs of inflammation
or deposit were discerned in this regard. The basilar artery
without branches, even externally engaged in the brain. The
blood vessels.
funeral cinder were [very conspicuous], and a proplectic clot or the indications of there were found as the following situations.

**Right Inferior Surface:** Anteriorly, and approaching its upper surface a shallow cutaneous depression, 1/2 inch in diameter, containing the remnants of a decaying blood clot, which did not fill the cavity. The posterior portion of this "epiglottis" had been extensively destroyed; a thin exudate, the size of a hazel nut, tenderly undersurface almost this large, surrounded by activities of yellowish and dark colored central hues. This lesion involved the contiguous an velar and, being injured or cut, did not involve the anterior threatened as of that side.

**Left Epiglottis:** 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 its cavity.

**Right Posterior Thalamus:** In the center, an excavation, in which lay a somewhat firm and yellowish exudate, sized a pea; just above this and rather posteriorly, and smaller in size, a more recent clot was to be seen.

**Central Hemisphere, right side:** A recent clot, the size of a small ball, lying immediately above posterior horn of its right lateral ventricle. **Left side:** A recent clot, of size rather less than the preceding, situated, half an inch from the surface of the posterior lobe, about the picture of Rolandic.

There was no refection around there were two clots.

**Heart:** in Systole; large and loaded with gas, the left ventricle was moderately hypertrophied, the right heart was dilated, no marked vascular disease, except some indications of th vessels, which did not effect their competency. Scleral of arteries as the ascending Aorta.

**Kidney:** typical of interstitial nephritis, the right was advanced, both much enlarged, surface pitted, capsule thick, blood vessels wasted, and in parts, apparently, quite diminished, a few cysts among the tubules.

**Liver:** in weight 4.98, deeply congested ("nutria"). It fell.
somewhat firmer under the fingers, than natural, but there was hardly any true anesthetic indication to be seen.

Spleen: engorged and considerably enlarged.

Stomach: billiaries; mucous membrane highly engorged. I could not say whether there was any sign of inflammation in the stomach.

Lungs: engorged and somewhat ephymatosus.

The next case shall give us an idea as to the association of symptoms clearly pointed to some organic disease within the head. The interest of a discussion of its symptoms lies more in their nature is 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 a family history, which could throw much light on the case. The patient was a carpenter by trade; raised, as far as one could make out, a temperate life; there was no reason to suspect he was the subject of Syphilis, or had any hereditary taint of a dyspepsia. 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. weakness of right ear
3. staggering gait
4. Vomiting, and some headache
5. Diplopia (double) and mydriasis.
6. Anesthesia of right side of face and 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 nature 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
and a few seems necessary with regard to this arrangement.
It is to be seen that such a list, if made complete, would appear
at first sight so far from being a priori; whereas it is my intention to illustrate quite the opposite method of acquiring knowledge, viz., the inductive or true scientific method. I wish to put the case as it would present itself as actual life to the mind of the
observer as clinical study. I am aware of the danger of verifying facts to suit a theory (a proceeding, only too easy, where too much,
as in medicine, rests upon conjecture). But if anyone are chagrined that in this arrangement of symptoms I have adopted a preconceived idea, it is owing not to supposing that this prevents me using the true scientific method—a method of induction. This is hardly the place to enter into discussion such a matter at length, but to raise the question of dialectics than anything else. But I would point out that, 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 if we know other things. That anything which we are pleased to call a "fact," really implies a host of notions or theories, and is in itself a generalization. For 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 one fresh 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 simile from another department of science)"faith" and "writings" should stand opposed. All life is problematical to the wise man; and it is to be hoped that as the world
progresses, it will lay aside, as it is laying aside, the fatal duel
which has been the greatest menace and is this too often the
fetter of those who penetrate the secrets of nature.
time. I do not deny the great value of diagnosing these diseases for practical purposes: but solely, be it understood, as a matter of ability, and not as implying an essential difference in making his diagnosis then, a physician must form some idea, theory in general notion (this in medical language is called præliminal diagnosis) which serves as a sort of mental scaffolding towards upon. The above list, 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 sleep well, he can't hear well in one ear, he says he feels sickly, 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 (perhaps the physician has already seen this while he is talking) and probably this is all he cares to tell you. Paul already the physician has made a first guess. He then proceeds to examine his patient's nervous system, the results of this examination he notes down in his memory as his note book, and while doing this he has formed a general notion of the case (he has made a præliminal diagnosis) in fact, he cannot help drawing some self-evident conclusions, and this, as inevitably, will guide him in his arrangement of the symptoms for further consideration. But he never carefully go over the case, he has got the so-called "facts," but they of themselves give no guide him to the truth, he has yet to lay his thoughts side by side with these facts, before they will yield proof. He has it in mind to bring all his available knowledge to bear upon the symptoms singly and collectively, when this is done, "hit us tell these, and often not even then to be justified in pronouncing his diagnosis.

Double Ocular Hemorrhages. I put this as its head of sickness as being the most valuable symptom of cerebral lesions, e.g., tumors. The opthamologic appearances here are, sine irune, and I will simply confine to a "tumor" idea. The retina was also, and extensively involved. It was bluerd (see annexed fig.) with patches, scattered in the fundus.
Double Optic Neuritis and appearance simulating Albuminuric Retinitis in a case of Encephalitis, believed to be, Insus Carabelli, and in which there was no Albuminuria.

in the right eye there was but little loss of vision, the eye being perfectly clear. In the other, the vision was equally perfect. There was no appearance of the retina, and there was no lesion of the disc. In this case, there was no albumen detected in the urine at the time the patient was under examination, and this seemed to be a case of chronic Bright's disease. It was known from the absence of the functional and structural changes which accompany that disease, whether the history of this case, and the history of the eye, was of the same order. As to the general question, if the ophthalmologic appearances were as described, is this case, brain disease such as a tumor, in what way did it disfigure it?

The grave matter of conjecture, as to the reason why a large lesion of the brain, whatever its situation may be, should cause changes in the optic disc. When a tumor is far back and inward in position, increased intracranial pressure by interfering venous return through the "tumor space" might account for a "choked disc." But then as Mr. Goodwin says it is not unusual to see a disc that is not distinguishable between ischemia of disc and a neuritis disciform. If this is so decided we cannot as much pass the threshold of the inquiry as to the mode of production of optic changes in diseases of the brain. Although the statement conflicts with certain views of Dr. Ogston's with the quotation from the same authority given on page 17, it does not necessarily indicate our ignorance on the subject. Mr. Hinglaj's statement never uses the term "ischemia" or "choke disc," but regards all 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 in cephalgia) acting
*Orlocal Times v Gazette 1872.*
in some way reflexly in the optic nerves. It is very seldom, he thinks, that a tumour causes this symptom by direct pressure.

But when symptoms occur in general have intense pain in the head. There was one such symptom here, however. This is a difficult question and it will require further investigation.

The defect in vision, which the patient complained of, I should attribute to the state of the retina. Anamnose is due to the change in its function, only when they have progressed through these previous instances. Such patients do not often become totally blind before death, and this is due to optic atrophy, a sequence of the retinas. Anamnose may also occur in tumour cases from anatomical disturbances of the cerea, when the tumour involves the trigeminal nerve.

The optic neuritis then could tell us something more than the probability of some gross lesion somewhere in the brain. But the patient complained of right eye vision. Now if this were to be attributed to the nervous system, i.e., if the right temporal lobe were involved, it would furnish a clue to the nature 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 these (although they were present to a certain extent) of such a kind as to reach a degree as to be of value.

Now headache of unilateral type is a usual symptom in cases of intracranial disease, and according to, perhaps, our best English authorities, in such matters, Dr. Bright's doctrine, "never results from disease of any kind in any part of either hemisphere." Dr. Bright goes on to say, in contrasting these two symptoms, it is his impression that "headache does not arise from intracranial tumors, or other abnormalities present, unless the part involved is actually involved or directly pressed on. If so this significance is altogether different from that of anamnose from optic neuritis, as in optic neuritis the abnormality is present elsewhere and involves any part of the nervous system." Again, if the head is supposed to indicate a localizing symptom, whereas so far as
"S. Etruria in Auditory Vertigo"
Medical Times Gazette vol 1 1877
yet Thurne, after the neuritis occurs from tumors in many parts, probably, in any case, of the cephalon. The causes of the deafness, where the patient walked was involved as no cause of exclusion, is by a method of exclusion: the cause adopted is that of Forbes Cells’ personal audition, i.e. making the bone to the height of the bone or the bone of the bone rather than the internal ear or the nerve itself. That was check, and then it first the<br>point to be made out. The hearing can be tested in this manner by placing a watch, for instance, on a tuning fork against the forehead or some bone in the median line of the head, or in the way as against the test. Now if you can be blocked (i.e. if there is disease of the tympanic cavity) the sound is heard better than 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 as the blocking of disease. Now in applying this test, in the case in question, and it was done twice, but several times, and with ease, the patient (who was an intelligent man) always said it was the W to that he could not hear the ticking of the watch for whatever I thought it in the right ear. This being so, and there being more of the aden of the tympanum of ear disease, e.g. discharge, Ear ache, neuritis (with the exception of vertigo), indeed, which will include pain because it was reasonable to conclude that there was something coming out this main right parties another; and if it was suspected that there was some in the ear of disease, this symptom would help to localize it. And this is not quite true, as far as localization goes, which was shown in the case of urticaria, 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, right, his movements as well put down to these? What does a staggering gait imply, as seen in instances, in this case
†. Physiology 1878 p. 517

x. Hermann's Physiology 1878. p. 504
of a lunatic 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, it may mean, that the centre of coordination, if such there be, is at fault. Now the general result of experiments and observations incline us to credit the cerebellum with the function of coordination. Feeling of loss of coordination being referred to lesion of this part of the brain, and remembering on the discordance of clinical evidence, St. Michael Hale, goes on to say, "Still experimentally, 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 functions more exactly." And this opinion is also advanced in the latest and perhaps the best physiological text-book in the language (Kerr, 1871). In his consideration of this symptom, I may mention the case in question, with that of abetolical rheomolism made very notice about the same time. There was a history of a severe fall, after which some kind of convolutional seizure had occurred; the head also began to enlarge more rapidly than was natural, and the right side to fail, so that the girl was taken to an oculist. At this time, she had a descent of the right eye, the right disc became atrophic. There was a strong hereditary history of struma, and judging from the age of mother and the appearance of the head, the diagnosis pointed to a growth of this kind probably located near the symphysis, sometimes described in the cerebellum. This symptom was the peculiar kind of walk; it was unsteadily staggering of the balance, with the shuffling of the rheomolistic, it was more an attempt to broaden the base of support, a stardy, soft, unbalanced suspicious of a tendency to fall backwards, reminding one in character though not in degree, of the "rheomolistic" symptom of Young. In the present case there was only loss of coordination here, and the actual injections might have been due to alcoholism. Indeed this
so-called "cerebellar reel," is cause the reel of the brain.

that, from it reported us to London Medical Record that

one of Nishinagai's patients (suffering from cerebellar ataxia) had several times been locked up by the police, as drunk, only to his table.

The diagnosis suggested in this little girl's case was, then, tumor in one of the cerebella, whether we could locally further to questionable; there was no evidence of a second localizing symptom; and therefore doubt.

a symmetrical enlargement of the head, would point to the middle one of the cerebella, or about the tentorium. By the "vena Galeni," would he obstructed and the hemisphere, there fore unduly expanded. In cases are on record, I have been two such, in which there was symmetrical enlargement with hydrocephalic distension, and yet the tumor was found after death to be in one or other of the hemispheres.

Now to return to this 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, the cerebellum may have different localizations, all of which pointing to the cerebellum as the "origo," but not necessarily implying a cerebellar in that situation. Recking may have (as Dr. Jackson has shown) from cerebellar, but this was included here. Might it be due to the diplopia, for the patient's perusal nerves in 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 the eye symptoms would not account for it here, indeed to an extent much from the same when both eyes were closed.

And as before noted, it was different from the suffering of the paralysis.
and then his hemiplegia did not amount to much. Might it not be due to the vertigo? Let us discuss this symptom. Our patient complained of being giddy. Now this is a symptom of very various import. The association between giddiness and staggering is so obvious that I cannot doubt that there is a causal nexus, or that between them there is 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 wavelike movement like nystagmus, and the body inclining in the opposite direction, and if these currents be strong enough, the sensation of giddiness passes into an actual felt vertigo. And again, all these phenomena are best explained by supposing that the current interferes with the cerebral coordinating mechanism which results in different effects, the compensatory movements of the body and eyes, the changes in the mechanism at the same time affecting consciousness as to produce a feeling of vertigo. But all this may be known by observation in the influence of any individual - viz. the association of giddiness and inaction of the eyes when rapidly turning round. Or say this movement makes us giddy and the eyes resting for a brief moment on each object presented are rapidly hurried on to the next in succession, causing a natural nystagmus. The phenomenon is known as the "laying off of equilibrium", but to speak more particularly and scientifically, it is called, in French, a vertigo. It is, then, the muscular sense. The sense which is the most important means we have for adjusting the body to its environment. As Dr. Livingstone says, "Vertigoous sensations are usually, seen but felt, but in both muscular sense is involved." After speaking of the ton of coordination in animals deprived of the cerebellum (organ of coordination), Dr. Forbess says, "Since the peculiar movements characteristic of vertigo may take place in the absence of consciousness, whereas the vertigo being actually felt we may with security assert the
* Plutarch, op. cit. p. 497.

Also ibid., p. 517.
No failure to stand upright and the feeling of giddiness are both reminiscent effects of the same disarrangement of the co-ordinating mechanism. The causal nexus is then not between the vestibule and the incoordination, but they both acknowledge a common source. Vertege, in fact, itself, a disorder of co-ordination, it is on physical evidence as Dr. Jackson has pointed out, a motor symptom. That is the common cause implied here: we have spoken of an afflicting of the muscular sense, and we have referred to the cerebellum as a centre of co-ordination. But, to quote Dr. Foster again, "It is probable that its functions (i.e., the cerebellum) are especially connected with affinal impulses proceeding from the semicircular canals. Fibres of the posterior roots have been traced into the substance of the cerebellum (particularly in particular) and the latter of Mr. Pickering has been found beyond doubt that this nerve (probably, of all the peripheral nerves) is in connection with the cerebellum. If it were not for this we should have been forced to explain why Ear disease (apart from propaganda through public opinion) should cause such symptoms of loss of equilibrium. Dr. Vulpian believed that the cause of the association of deafness, tinnitus and vertigo, in this disease which bears his name (and in which also there occurs sometimes obvious motor symptoms) was some disarrangement of the semicircular canals. He arrived at this conclusion by reasoning from the well-known experiments of Yerewod. And the speculation of Goitz was that injury to this part would explain the symptoms of vertigo and incoordination. In this view Dr. Jackson's diagnosis coincides, and mentions cases of deafness from injury, as already said was a prominent symptom. One in particular in which the patient always felt giddy and unsteady, looked like a drunken man, so that it might be supposed that there was also cerebellar disease." Dr. Foster "has no doubt that the semicircular canals are centres of equilibrium; when one
Holt, op. cit. p. 877.

ampulla is irritated the impulsion is away from it; thus, rotation of the left-posterior one will cause the patient to fall towards and to the right. In this view the ampullae form a sort of peripheral sense organ for the centripetal cerebellum of the muscular sense.

In Meniere's disease (Auditory Vertigo), there is generally, as before stated, an obvious motor symptom; 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 after falling the patient will often turn in the 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 leaned, but there were always deviations, when he walked, to one side or the other. It would appear from experiment as well as from clinical observations that the deviation, falling, a rotation is as a rule towards the side of the lesion. Section of the middle peduncle of the cerebellum on one side almost invariably gives rise to rotary movements, but instead 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 - Out there are many clinical observations. However, there is a certain general authority (Dr. Nottigall) 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 after cerebral hemorrhage, it may be mentioned in this relation (p. 24).

In many cases of brain paralysis with epileptiform onset where this symptom is observed, it is considered safe to
conclude that the disease (e.g., a clot) on the side of the brain toward 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 inconceivable and false. In the case there is an organic lesion, whereas in hearing disease it is put down to functional disturbance; but putting aside the question whether one may well ask, and which haunts one the query into the Epileptics for instance, as to the real relative meaning of the things we call "organic" and "functional." We may remark that in uncles can occur in the paralyzed side of the body the same lesion (which, roughly, I proceeding with bodily and emotional); there being at the same time what Dr. Dust would have called, a "destruction" and an "irritation" lesion; what Dr. Trowell would call, a "destruction" and "destruction lesion."

But to take a more analogous example viz. that of two Epilepsy with an auditory aura (and cases of this sort are very apt to be seen in the same locality), the reverse seems to hold. An Epileptic of this description has instance noises in the right ear, immediately, preceding, before the head comes to the right, "so as to look almost over his back," and then he falls insensible. Now, if auditory vertigo is as Dr. Carse described, an Epileptiform neurosis in the visceromotor tracts, a center of equilibrium, are supposed to be in a state of instability; one would have expected that the unpleasant scenes have occurred, not the reverse way, but the same way, as in Epilepsy; and auditory

which the auditory apparatus is also evidently involved. It must be emphasized that this kind of Epilepsy is very rare. If I had not myself seen the case quoted above, I should have hesitated to refer to it.
To true was Féré's in his experiments produced the
impulsion in the same way of which it is alleged by such
depil observers as St. Guven. to take place in auditory
Vertigo
But according to Dorsouvre the impulsion was always to the
side of this lesion.
This conflicting evidence which is interesting in a clinical
point of view, strikes more at the possibility of localization
in these cases than of the hypothesis of the connection between
the cerebellum and the aortic apparatus.
Amuse serious objection to this view is the fact that "often
section of both-nerved auditory lobe animal does not lose the
power of controlling its movements in space, and when
subjected to passive movements a to the action of a current
passed through the head, it reacts as if unimpaired. Various
persons who accidented in accident exhibit no other ab-
normality but death." Then also certain considerations of
Naturhistoria, as well as the physiological fact that the Ferri-
ocular canals do undoubtedly, because an aortic function,
are against this hypothesis in question. In recent
infirmities of ophthamology, he always observed that disad-
ussed to the nerve to doubt its accuracy, or rather the modi-
ification of it, which it is not necessary to mention further.
Indeed it would be out of place to prolong the discussion
of this question in a paper such as this and I must
confess to 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, where vertigo and loss of equilibrium
as symptoms.
Paul, 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, he himself closed one eye and
only looked with one eye and incline his head.
His diplopia taken in connection with the following symptoms must be put down to the same cause of his motor troubles. Dr. Living says, "Generally there is diplopia from disarrangement of muscular sense." And in its commonest instance of incoordination of vision, double vision is an early symptom. He, I presume, exactly analogous to the staggering of the locomotor apparatus, the Stannumning of his organs of speech, and the helpless twitching of the muscles of expression. Indeed, Sir James Paget, in his remarks upon paralysis,istinguishing the bladder's use of the expression, "Stannumning Stannumning unto other organs than those of speech." Diplopia is then, I suppose, a "Stannumning" of the eye, and we are safe in putting it down to some affection of the ocular motor nerves, the first in particular: (Another localizing symptom), but it is more difficult to assign the cause of the palsy to the brain. In the present case the palsy becomes worse in all directions. That it is intimately connected with the symptoms of loss of equilibrium has been noticed in the foregoing remarks. Tyrer produced it in his experiments, and he describes the palsy of the eyes which is associated with gloominess and unsightliness as an "Eye = leptotomy affection of the cerebellar or sub-cerebellar centres." 
As to the central position of what he calls the auditory centre, in the cerebellum, Tyrer produced movements of the eyes by the electric shocks: and it is interesting to observe that Lyon has also found that experimental irritation of the semicircular canals produces ocular deviation, each canal acting its own deviation; another proof of the association of the labyrinth with the muscular sense, as well as the sense of equilibrium. But then it must be remembered that my patient's case is a rare case in which we do not expect any affection of the cerebellum. In the case, therefore, I have seen a boy who died of tumours,
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were cerebral hemiplegia: the nystagmus was very marked but nothing amiss with the cerebellum could be detected as noted above, although it was carefully looked for, as tabetic facial had occurred before death. Then again we are met with persons who have nystagmus, perhaps from birth, and yet there is no giddiness, or other associated symptoms. But these cases may well be instances where the organism tries to adjust itself to altered abnormal conditions. Although we cannot explain the symptoms of nystagmus more accurately, it is, at any rate, suggestive and of some diagnostic 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 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 its paresis, and hemiplegia of the limbs, all occurring on the same side viz. 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 of medicine (Bristow) we find his 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 on one side; its loss of sensation was not so marked. There was no distortion of the corner of the mouth, at least not when at rest.

In contradicting Dr. Todd’s erroneous opinion, that it was the fifth and not the seventh that was involved in facial hemiplegia, Prof. Tanderlos says: “Dr. (Dr. Todd) says nothing of falling in obliquity of the lower jaw, which that
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Roth, "Exposition of the Hebrew System" 1824. p 164.
paralysis would certainly occasion. Now this was not
seen in this present case; we should not expect it. In this
former case does not seem its appropriate 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 linking the parts 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 it "expression") that the
jaw loses its opposition while at rest, to some extent, to
muscular action alone with decay; 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 so
closely) mentions the diagonal of the cheek and angle of the
mouth in cases of some, hemiplegia paraplegia, believing
the symptoms to be due to affecting the trigeminal nerves.

It seems strange that it never occurred to this great observer
who made himself master of the mechanism of expression, that
that is done by disease, and not counted fault in health in
certain states of mind. As we are apt to say, for instance,
of a person in trouble, as Jones is the receipt of some bad
news, that "his countenance fell." This change in facial
expression, not, would have been more ready to ascribe
to the facial nerves than to Sir Charles Bell. But then it may
be said he drew his conclusions not only from clinical
evidence but from experimentation on animal as well.
There are two or three remarks to be made here. Firstly,
this well known that where symmetry is native and to the
human being is often also evident, and this might indicate all
the difference in the case in question. I can imagine
that we long projections being in the...
chiefly used us his experiments by Sir Charles Bell, * would be much more likely to fall on the removal of muscular support than in the case of man, and this again depends on the fact nerve on one side is a much more serious affair for the muscles supplied, than ever happens, I inquire, in central disease which affects cranial nerves. Then if it must be remembered that Sir Charles Bell resorted to circumciseal experiments after his amputation 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 its fifth, in man, to produce anything more than weakness of the temporal, masseter, and zygomatic muscles of that side, and which would not affect the power of the face while it rest; and only to be ascertained when the muscles were put in strong action. It was only when I made my patient clenche his teeth firmly that one could detect the difference in the stiffness of the muscles, of the two sides. But it was clear to observe that no one could mistake it.

It seems strange that Dr. Todd, 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 naturally confined to involuntary and forced action of the muscles of expression : the usual incomplete paresis of facial paralysis being neglected: the above is half the amount seen nearly, if at all affected. Of course in such slight palsy there was no alteration in the natural tone of the face, no falling of the cheek, from this cause. I did not observe any trigeminal deviation.
Jan. 1876, m. II. f. 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 paralysis of the fifth would certainly vitiate any conclusions drawn as to the special nerve apparatus of these senses; and in the case of taste, where there is this triplex association of paralysis, the problem becomes too complicated to be of any value in diagnosis.

The affecting of the fifth would suggest some impairment in common sensation in the tongue, the pharyngian nerves, heart, 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 seemed to corroborate the conclusion that the lesion was in the right side of the 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. Seguin mentioned a case in which, at the post-mortem inspection, a tumour was found to occupy one side of the brain; extending also into the case crania! of that side; during life the following symptoms were observed - hemiplegia and facial paralysis and sensory, all on the same side as the lesion of the brain. In this respect I may quote the authority of Dr. Hughlings Jackson (which is always trustworthy) who gave out as his opinion that a lesion in this...
situation viz. in the region of the middle crus of the cerebellum on one side and not only accounted 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 experiments laid on this part of the brain — and Dr. Wortley's testimony, quoted on page 36. I concluded that the lesion was, some adrenalin product, probably a tumour in a cyst, of, or pressing upon, the right crus cerebelli and flocculus, involving the trigeminal, facial, trigeminal, and abducens nerves, and perhaps the other motor, as their intracerebral 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 the patient died the 8th after he had passed from my observation, and no post-mortem examination was then allowed. There was therefore no verification of an inductive reasoning. But I think the facts detailed above favour this last claim. No convulsions occurred throughout the case.