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A CONTRIBUTION

TO THE

Pathology of the Cerebellum.

BY

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A CONTRIBUTION TO THE PATHOLOGY OF THE CEREBELLUM.[1]

By E. C. SEGUIN, M.D.

I VENTURE to publish this contribution, because it seems to me that a certain value attaches to cases which, fairly well observed during the patient's lifetime, are completed by autopsy. Especially is this true of cases of somewhat rare lesions, often of difficult diagnosis, as are tumors of the cerebellum. Even if the publication of such cases brings no immediate brilliant result, it may be of help, later on, to other workers in the same field.

The first case which I shall relate tolerably well fulfils these conditions: it was observed frequently, almost constantly, during eight years by several competent general practitioners and by myself; frequent notes of the symptoms were recorded; the patient was made the subject of clinical lectures at the College of Physicians and Surgeons in several successive winter sessions; and a correct diagnosis of the location of the lesion was made early in this period, being fortunately recorded in a letter written to the attending physician in 1878, seven years before death. Finally, a reasonably complete autopsy was made, which brought to light the lesion which had caused symptoms for so many years, besides recent lesions not at

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all connected with the almost pathognomonic symptoms upon which the diagnosis had been based.

Three other cases of tumor of the cerebellum are also briefly related and their specimens presented. They are not as instructive as is the first case, but they also seem to illustrate, in a somewhat useful manner, I trust, certain points in diagnosis, prognosis, and treatment. The four cases together substantiate in many ways the diagnostic laws first formulated by Nothnagel.[1]

CASE I.—P. M., a single man, 37 years of age, a retired officer U. S. Navy, was first brought to me by Dr. D. C. English, of New Brunswick, N. J., in the autumn of 1877. From numerous notes taken from that time until May, 1883 (when the last entry was made in my case-book by Dr. Amidon), I condense the following history of the case:

Illness began in 1867 (eighteen years before death) by much headache and an attack of partial loss of consciousness or powerlessness. Had several such attacks, never falling in them. In 1868, consulted Dr. Agnew[2] because of failing sight. Had no diplopia. A seton was put in the back of his neck, notwithstanding which headache continued as the chief symptom of the disease. He had no symptoms in the legs. Later, in 1869, the headache diminished.

In 1869–70, began to notice a gradually increasing loss of power in the legs; no pains in them.

Was in Europe from 1871 to 1874; legs and head troublesome; sight impaired, but not growing worse.

Returned home and was retired in 1874; has since been in *statu quo*. The head is perhaps less painful than in 1870.

In the last few months pain in whole of head, more in front; mostly in front of head and in orbits (none in face). This pain is not nocturnal, nor is it influenced by weather changes. No dizziness. Never diplopia or hemianopsia. No neural pains in limbs; hands skillful. Denies changes in speech and loss of memory. No difficulty in micturition. Most positively denies chancre and syphilitic symptoms.

Once in a while he has had a bilateral feeling of numbness of hands and feet, never permanent. This was not a numbness from position of limbs. Less headache. Has become pale and thin.

Examination.—The head is held stiffly and vibrates a little. The eyeballs show great horizontal nystagmus, especially when they are directed to his left. Pupils equal, normal, and of medium size. Ophthalmoscopic examination shows marked atrophy of both optic nerves (the left more), choroidal atrophy and abnormal

[1] "Topische Diagnostik der Gehirnkrankheiten," p. 78. Berlin, 1879.
[2] No record of this examination can be found.

pigmentation. O. D. V. = $\frac{10}{100}$, O. S. V. = $\frac{10}{200}$. There is no facial or lingual paresis or tremor. Speech is not perfect, but rather slow and hesitating; not exactly syllabic. The hands are steady and strong: R. 50°, 49°, 45°; L. 37°, 38°, 36°, in three successive trials on dynamometer.

There is no ataxia in the gait, but the walk is peculiar: stoops much, staggers with feet apart and very active. With eyes closed the steps are smaller and more cautious, but the gait is not aggravated. Stands almost perfectly with eyes closed and feet slightly separated; but cannot stand with feet held together. The legs, in walking, are not jerked outward and forward, nor are feet brought down as in ataxia. Strength (resistance power) at knees normal. When walking with bare feet it is seen that the toes are abnormally active, clawing the carpet, as it were, to get support.

The sensibility of face, fingers, and legs (one tested) is normal.

There is slight ankle clonus (patellar reflex not noted).

I then made a diagnosis of disease of the cerebellum, excluding posterior spinal sclerosis.

After seeing the patient a third or fourth time, in May, 1878, I wrote as follows to Dr. English:

"*Dr. D. C. English, New Brunswick.*

"My Dear Doctor:—I have re-examined Capt. M. with great care, and can make out no material changes for better or worse. He seems not to be in a good general condition. I am now more certain in my diagnosis. I feel quite sure that he has disease of the cerebellum. According to very recent studies of cerebellar lesions by Prof. Nothnagel, of Jena, the disease in Mr. M.'s case is very probably central or bilateral and involves the vermis superior. I see no new therapeutic indication. It would be well to build him up for a while before resuming the former powerful medicines.[1]

"Very sincerely yours,

"E. C. Seguin."

In the notes of May 14th, 1878, it is stated that lately there has been occasional diplopia for distance: that the patellar reflex is normal. The heart is healthy. The walk is again minutely described as *supra*.

Oct. 16th, 1878. Claims great improvement over last year's condition. No headache worth mentioning. Speech is perhaps better. Eyes unchanged. Walk is as before characteristic of cerebellar disease; with feet wide apart, arms used to keep equilibrium, toes clutching the floor. Co-ordination of hands perfect. Stands almost perfectly well with eyes closed. Speech thick and slow.

April 11th, 1879. Certainly no worse. Tongue deviates to right. V. as before. Grasp: R. 50°, L. 40°. Has a complex nystagmus. In central fixation, finger held directly in front of patient, it is downward ↓; in outward fixation it is in either direction according to direction of the strain → or ←. Has taken about one hun-

[1] Courses of iodide of potassium and of nitrate of silver had been given.

dred grains of nitrate of silver since Feb'y 1st without discoloration of skin or mucous membranes.

October. No material change. Speech the same. Occasionally has had a feeling of impending loss of consciousness (petit-mal?). The patellar reflex is supra-normal on both sides. Slight diffused headache.

May 21st, 1880. No change except that nystagmus is different: constantly obliquely to left and downward ↙.

Oct. 27th, 1882. Last July noticed a peculiar sensation in epigastrium, not nausea. One day that month fell forward unconscious; there was no spasm and he did not bite his tongue. Since has had a "light" feeling in his head several times. This is first distinct attack since 1868. The walk is less good, but has same characters, groping, feet separated, hands reaching out, body bent forward. No true ataxia in hands or in feet (tested lying). Stands as well with eyes closed as with eyes open. Patellar reflex greatly exaggerated on both sides. Pupils active, of medium size; both optic nerves much atrophied, with small vessels. Pulse of normal rate, but over-tense. Urine recently re-examined and found normal. Manner slow, looks haggard. Sleeps well. At this time Dr. English thought that there had been no material advance in the disease.

In May, 1883, had nausea one day, ending in projectile vomiting lasting all evening. The next morning he was found semi-comatose, and became conscious the next day.

I did not see Mr. M. in the last two years of his life. He was attended a part of this time by Dr. D. C. English at New Brunswick, and by Dr. W. Elmer at Trenton during the last year.

Dr. Elmer has very kindly given me the following statement as to the course of events in 1885:

"On the 23d of February, after complaining of headache a few days previously, he was taken suddenly in the library with an epileptic convulsion lasting, if I remember rightly, about half a day before consciousness was regained. I do not think there was absolute paralysis of the left side at this time, but partial immobility; his power to move or lift his leg was quite limited, and he complained of pain in the leg and foot, and also in the hand, desiring friction of both members. The limb was not stiff, and yet motion was rather painful. The attack affected also speech and deglutition; the former being very indistinct and the latter performed with effort. I cannot say that the tongue was actually shrivelled, though I am sure there was no enlargement of that organ. Without any test by the æsthesiometer, there was hyperæsthesia of the affected parts."[1]

There was afterwards no sudden change in the patient's condition. New symptoms gradually appeared, such as increasing dulness, stupor, drawling, greatly impaired articulation, difficult deglutition, and polyuria; in brief, many bulbar symptoms. Con-

[1] Letter dated Trenton, Jan'y 20th, 1887.

vulsions occurred on the 20th of April, and death took place by coma on the 22d.

AUTOPSY.—On April 24th, 1885, forty hours after death, I made the autopsy in the presence of and with the assistance of Drs. English and Elmer. The following notes were taken on the spot by one of the gentlemen present:

The skull and dura mater are normal. The convexity of the brain is pale and rather flabby ; here and there are opacities along the superficial vessels, and a number of these show streaks and patches of yellowish new-formation. In some, an old thrombus is demonstrable. Base of brain : olfactory bulbs are normal ; the optic nerves small and of a dull yellowish color ; the third nerves are also yellowish-white and dull-looking, but not small ; the fourth, sixth, seventh, and eighth nerves appear normal. The same is true of the filaments of the hypoglossal nerves and of the lateral bulbar nerves. The carotids are stiff, gaping, and are the seat of marked arteritis of the patchy or nodose form (not calcareous). The branches of the carotids, the posterior communicating, and the posterior cerebral arteries show the same lesion. The basilar is more diseased, the arteritis occupying its whole length, making it a stiff, whitish tube with fair calibre. The vertebrals are less diseased, about as much as the carotids. No thrombi or emboli in any of the principal vessels.

Transsections of the cerebrum show dilated lateral ventricles. The nucleus caudatus is normal on both sides, but the section at the level of the chiasm shows the outer segments of both nuclei lenticulares in a state of softening ; yellowish gray. The external capsule and claustrum are, however, normal. The caudal part of both thalami shows yellow softening, the left more than the right. The internal capsule looks white and normal.

Spinal cord : Its dorsal veins are extremely injected and varicose ; the cord itself seems small for the size of the subject. A section through the cervical enlargement shows only a doubtful grayish appearance in the left postero-lateral column.

Cerebellum : On inspection, the caudo-dorsal aspect of the cerebellum reveals a large cyst, apparently two inches (50 mm.) in diameter, occupying the site of the vermis superior (middle lobe) as far as its frontal third, extending caudo-ventrad as far as the fourth ventricle. It appears that the vermis superior is destroyed, except its frontal third. The floor of the fourth ventricle is apparently not diseased. The cyst extends at least one inch (25 mm.) into the right lateral lobe of the cerebellum. The caudo-dorsal boundary of the cyst is simply a transparent membrane (arachnoid ?). Its basal and lateral walls are thicker and somewhat gelatinous, but it is apparently a smooth membrane (no sarcomatous basis). The cyst is strictly monolocular and contains only a clear fluid without floating bodies.

The medulla oblongata and pons are both softer than normal, and the projection of the anterior pyramids and olives is somewhat

effaced. No sections were made, and the cerebellum attached to the pons and medulla was placed in a solution of bichromate of potassium for hardening. Other specimens were also preserved for future examination.

The heart showed marked hypertrophy without valvular disease. The aorta bore non-calcareous patches similar to those in the encephalic vessels.

The following is a fair summary of the case:

Beginning eighteen years before death with headache and one or more epileptiform or apoplectiform attacks. Impaired vision, with atrophy of optic nerves; nystagmus; continued headache, mostly frontal; *typical cerebellar titubation;* slight dysarthria; no distinct paralysis, ataxia or anæsthesia; patellar reflex exaggerated; foot-clonus; partial left hemiplegia without contracture. Death after cerebro-bulbar symptoms (stupor, greater dysarthria, dysphagia, salivation, and polyuria).

Lesions: Cyst of the cerebellum, destroying the middle lobe (or vermis superior), except its frontal third; penetrating deeply into the right lateral lobe, but not destroying the nucleus dentatus; exerting some pressure upon the floor of the fourth ventricle. Also, extensive arteritis obliterans, causing multiple foci of softening in the cerebrum. There was found, later, on making sections, a hæmorrhagic focus in the right ventral half of the pons (in the midst of the pyramidal fasciculi), causing descending secondary degeneration of the usual type.

ANALYSIS OF SYMPTOMS.—This long clinical history may, I think, be advantageously divided, for the purpose of analytic study in connection with the lesions found, into five periods:

1. The period of onset or of initial lesion. The account of this is very meagre, and we have no professional memoranda to guide us. Two or three attacks of an epileptiform or apoplectiform nature, preceded and followed by headache, marked the occurrence of the primary lesion or lesions. Quite certainly there was no marked paralysis or inco-ordination then, for the patient continued to perform his duties as paymaster, U. S. N. Without being at all positive in my assertion, I would suggest that these attacks represented hemorrhages in the cerebellum, probably in the right hemisphere dorsad of the nucleus dentatus, and not far from the vermis superior. These hemorrhagic foci, instead of cicatrizing, became fused in one active cystic formation, which gradually enlarged and

encroached more and more upon the medial part of the right lobe, ultimately involving the vermis, and exerting some pressure upon the floor of the fourth ventricle. The next year, 1868, the patient consulted Dr. Agnew for failing sight. In all probability, there was then the condition of neuro-retinitis passing into atrophy, so well known as a symptom of distending lesions in the brain. There were then no symptoms in the legs, so that if we rely upon cerebellar titubation as the special symptom of lesion of the vermis, the cystic or pre-cystic formation had not then encroached upon the middle lobe of the cerebellum, although it was capable of impairing the nutrition of the lobi optici, to such an extent as to produce partial loss of vision.

2. The period of impaired equilibrium, which extended from 1869–70 to within a few months of death. This was ushered in by "weakness" of the legs, followed in a year or two by staggering. In 1874, this disability had become so great that the patient was retired from the service. During the five years, 1869 to 1874, there were no new symptoms, and the eyesight, though impaired, did not grow worse.

It is very probable that in the years 1869–71 the cyst attained its maximum size and permanent relative position, as shown in the specimens.

3. A third and very long period of *statu quo:* the lesion quiescent, and no fresh cerebral lesions formed; from 1874 to within two months of death, let us say about twelve years. During eight years of this period, from 1877, Mr. M. was under my immediate observation. I saw him repeatedly, and three times at least made him the object, with his intelligent co-operation, of clinical lectures in winter sessions at the College of Physicians and Surgeons. He then presented the following symptoms: More or less headache, occipital and frontal, partial atrophy of both optic nerves, nystagmus of variable type, slight slowness and thickness of speech, and typical cerebellar titubation. Repeatedly was it demonstrated that there was no ataxia, strictly speaking, no paralysis or

anæsthesia. The mental functions were normal, and the special senses, except sight, active. The absense of fulgurating pains and the presence of good knee-jerks, excluded posterior spinal sclerosis, of course.

4. The attack of convulsions followed by partial left hemiplegia, which, according to Dr. Elmer, was purely motor and unattended by secondary contracture. This corresponds, I believe, to the hemorrhage in the right ventral area of the pons Varolii, and to the secondary descending degeneration which can be traced from it even with the naked eye.

5. The terminal period, characterized by many bulbar and cerebral symptoms. These symptoms were all due, in my opinion, to various localized ischæmias produced by the generalized arteritis, and not to any increase in the original cerebellar lesion. The arteritis, affecting as it did most of the basal and cerebral arteries, produced the various foci of softening in the cerebrum noted in the autopsy, and also led to impaired nutrition of the medulla oblongata. It may be a question whether the bulbar symptoms, dysarthria, dysphagia, salivation and polyuria, were all of strictly bulbar origin, or whether some of them may not have been the result of softening of the outer segments of the lenticular ganglia. Against this view, that we had here symptoms of pseudo-bulbar paralysis, may be urged the fact that the external capsules, claustra, and cortical substance were normal (to the naked eye, at least). It seems to me that the symptoms above named were truly of bulbar origin, though not due to pressure from the cyst. Sections through the medulla do not show any distortion of the floor of the fourth verticle, such as would have been produced by severe pressure downward by the cyst. The stupor and final coma, lastly, represent the more extensive softening in the brain, and its general ischæmia due to the arterial degeneration.

PATHOLOGICAL ANATOMY.—As described *supra*, this cyst was monolocular, filled with transparent fluid, and bounded by a definite limiting membrane all around. At the caudal end of the cyst, the membrane and pia mater

were united. A piece of the membrane from the basal part of the cyst appears firm, homogeneous in texture, and about the thickness of thick drawing-paper (about ½ mm.). It looks, as in the piece passed around, not unlike dura mater which has been hardened in bichromate solution and alcohol.

Fig. A represents fairly well the appearance of the cyst *in situ*, as seen from behind. It replaced the greater part of the caudo-ventral vermis, destroying more especially the pyramid, uvula, and the right tonsilla. Frontad, portions of the ventral vermis remained, overlying the fourth ventricle; this was made up of the lingula, as far as I could determine.

Figs. B and C are both made frontad to the greatest development of the cyst, and show it bounded, dorsad and ventrad, by more or less altered, foliated cerebellar cortex. The left nucleus dentatus is absolutely intact, the right almost normal in size, the lesion just reaching it. The gross specimen and the thin transsection passed around show the level of the floor of the fourth ventricle undisturbed; there was not, probably, any great degree of pressure upon it.

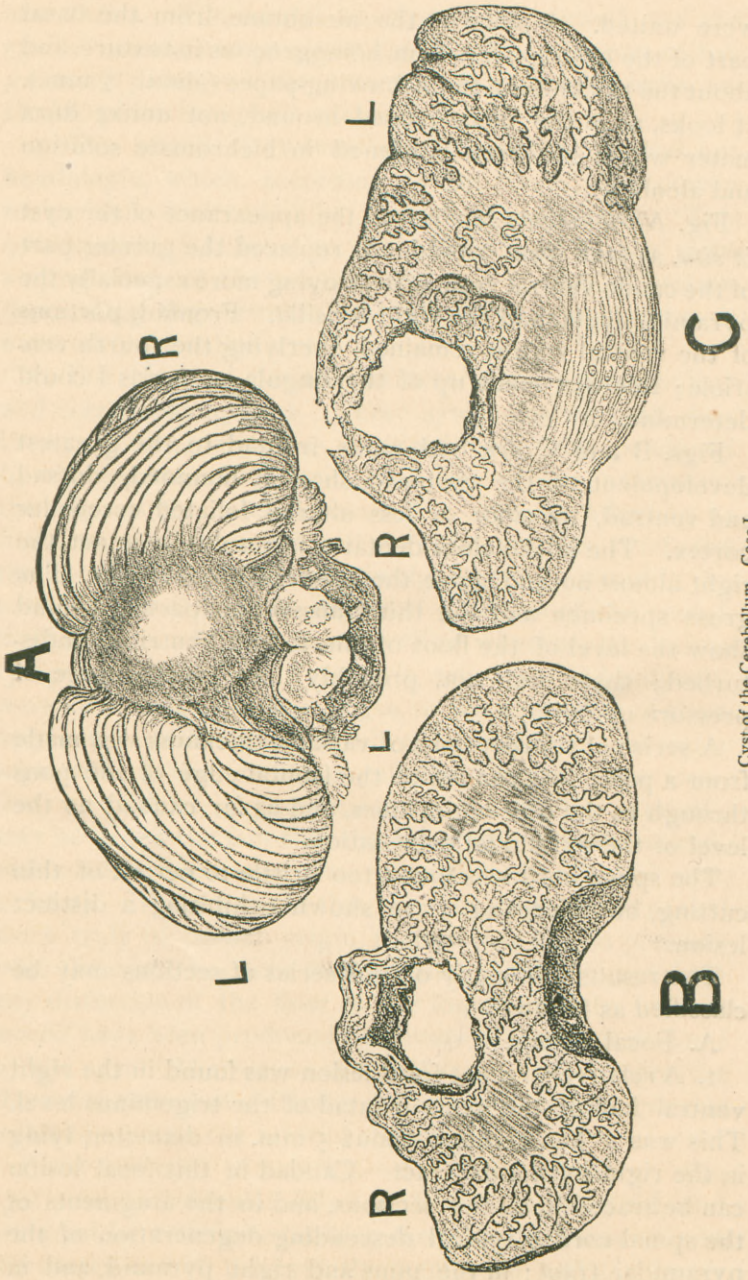
A series of transsections of variable thickness was made from a point just caudad of the frontal edge of the pons through to the post-optic lobes, and as far caudad as the level of the pyramidal decussation.

The spinal cord was found too brittle to permit of thin cutting, but pieces of it are shown exhibiting a distinct lesion.

The results of a study of this series of sections may be classified as follows:

A. Focal lesions.

1. A relatively recent focal lesion was found in the right ventral half of the pons frontad of the trigeminus level. This was a hemorrhage about 5 mm. in diameter, lying in the right pyramidal tract. Caudad of this focal lesion can be traced in all the sections, and in the fragments of the spinal cord, a typical descending degeneration of the pyramidal tract; in the pons and right pyramid, and in



Cyst of the Cerebellum. Case I.

the left crossed pyramidal fasciculus of the cord. The right anterior column also shows a tractus of degeneration near its edge. This lesion fully accounts for the attacks of convulsions and partial left hemiplegia which appeared two months before death. It is interesting to note that there was not a positive contracture of the paretic extremities to match this well-marked descending degeneration. I believe that occasionally degeneration of the pyramidal tract may exist without contracture, but in this case it might be said that death occurred too early to allow of the development of the contracture, as only eight weeks elapsed between the seizure and the fatal issue. The time necessary for the establishment of contracture in hemiplegia varies, from four to twelve weeks; still the fact that here we have fully developed degeneration of the pyramidal tract without contracture is interesting, and serves to throw additional obscurity upon the mechanism of hemiplegic contracture.

2. Changes in the medulla oblongata caused by disease of the arteries. There are histological changes in the nuclei of this region, but I have not had time to study them.

B. Secondary degenerations from the cerebellar lesion. This, it will be remembered, destroyed the caudo-ventral vermis and a part of the right hemisphere of the cerebellum, extending ventro-laterad as far as the nucleus dentatus.

1. The processus ad cerebrum (anterior peduncles of the cerebellum) appear normal and are equally developed as far frontad as their decussation point, just caudad of the nuclei tegmenti.

2. The processus ad pontem (middle peduncles of cerebellum) are normal; though their fibres pass through atrophied bundles of the pyramidal tract in the right half of the pons.

3. The processus ad medullam (posterior peduncles of cerebellum, more especially the corpus restiforme) appear somewhat reduced in volume in the bulbar sections on the right side.

4. The left olive is undoubtedly smaller, flatter, than its

fellow, and contains ganglion cells which seem smaller and ill-developed.

This atrophy of the olive is not at all extreme, not comparable with that shown me by the late Prof. von Gudden as resulting from removal of one-half of the cerebellum of a rabbit.

5. As far as can be determined in the imperfect sections obtained from the dorsal spinal cord, the ascending cerebellar fasciculi (direct cerebellar tracts) and the columns of Clarke are not atrophied or degenerated.

Consequently it may be concluded that in an adult man destruction of the caudo-ventral vermis (pyramid, uvula, and one tonsil), with some injury to the hemisphere of the cerebellum reaching to the nucleus dentatus, existing for about sixteen years, produces atrophic (degenerative) changes only in the corpus restiforme of the same side and the olive of the opposite side, both these organs being only slightly affected.

CASE II.—J. J., aged fourteen years. Seen first on July 29th, 1880. Had been a healthy boy. At three years had whooping-cough severely, with several convulsions. Parents deny convulsions or petit-mal since.

About January 1st, 1876, J. fell heavily on a stone walk, striking his head so hard as to make him unconscious; did not vomit. In April of that year he began to have curious vomiting spells in the early morning, followed by violent occipital headache. The patient describes the vomiting as not preceded by nausea, and the rejected matters contained no food. After having had these attacks for several days, one afternoon J. fell unconscious and had a general convulsion, repeated in the night. After this J. carried his head inclined to the left shoulder, his occipital headache continued, and he had a stiffish feeling in the neck. The vomiting did not return, and there was no delirium.

At the end of May he had gradually become paralyzed generally, but more on the left side. He had pain in his eyes with rapid failure of sight. Drs. Agnew and Knapp found white atrophy of the optic nerves. No recovery of sight since. (It is very probable that during April there had been choked disks, with fairly preserved vision.) Speech was never affected.

Spontaneous improvement occurred, and in July J. was able to sit up, and gained rapidly in all respects except sight. Some disability in use of hands and walking remained. He grew well, and was taught at a school for the blind. Has been very intelligent. No special symptoms occurred for nearly four years, viz.,

until May of this year (1880) when he began to have attacks of occipital pain and vomiting; occasionally had pain in left mastoid region, and numbness in left side of chin, and around left corner of mouth. A few days ago was found unconscious; probably having had a convulsion. Admits occasional dizzy or unconscious spells of momentary duration. Is still able to be up all day, dressed.

Examination (July 29th).—Eyes in left conjugate deviation; sightless; pupils wide; nerves bluish-white. Tongue straight; right hand 20°; left 25°. Left leg stronger than right. Consequently has right hemiparesis; no tendon reflex at knees; walk is staggering, more off toward his left. There is no distinct ataxia, and the walk is not of the type called cerebellar; no anæsthesia. I gave him a mixture of bromide and iodide of potassium, of each salt about 1. at night; quinine, sherry-wine, and food.

Sept. 29th.—Patient improved wonderfully in first month of above treatment. Early in September had a sort of convulsion, and since more or less occipital pain; objective symptoms as above.

Nov. 14th.—Poorly of late. Occasional attacks of occipital pain and vomiting (without nausea); rather frequent attacks of petit-mal, or perhaps more strictly speaking syncopal attacks, usually associated with headache. In last twenty-four hours has been semi-comatose, at times vomiting.

Nov. 16th.—To-day better, and is ordered ten drops of a saturated solution (equal parts) of iodide of potassium three times a day, to be increased each day by two drops at a dose. The small dose of bromide heretofore given (about 1.) stopped.

Dec. 6th.—The iodide has been gradually increased to forty drops three times a day, with the best results: no headache or vomiting or syncope since beginning iodide. No bromide. Rich food and sherry.

Examination shows a new symptom, viz., occasional twitching and distinct ataxia of the right upper extremity; none in the legs; perhaps a trace of ataxia in left hand. Absolutely no tendon reflex at knees. Right hemiparesis; no anæsthesia; face not paralyzed. Is up all day, and walks out-of-doors occasionally. Iodide to be gradually reduced.

Several times during the winter and spring of 1881, J. had a return of occipital pain and syncopal attacks; more recently of cervical pain also. These attacks were invariably cut short by blistering the nape of the neck or the mastoids, and by giving at once the full doses of KI, viz., from forty to fifty drops three times a day. Previous to Dec. 21st, the blisters had not been used, so that we may conclude that the more potent agent, in affording relief to the very distressing and threatening symptoms, was the iodide of potassium. The relief usually appeared in two or three days. Between the exacerbations the dose of iodide was from ten to twenty drops; and he had a variety of tonics.

The summer of 1881 was exceptionably favorable for J. He

was very well and happy. Though blind and slightly ataxic he enjoyed life, and was very cheerful. He had learned to do many delicate manipulations with his hands.

Oct. 12th.—J. was seized with convulsions, vomiting, and a gradually increasing pyrexia. Died comatose on 14th at midnight, with axillary temperature of 103° F.

Autopsy showed a tumor involving a large part of the inferior portion of the right hemisphere of the cerebellum, compressing the underlying portion of the mesocephale. The upper three-fourths of the same right hemisphere of the cerebellum was occupied by a cyst containing a clear fluid. The bottom of this cyst is the solid tumor referred to above. The cyst has disintegrated the upper and middle portions of the vermis superior.

The cerebral convexity showed abundant heavy patches of purulent sub-arachnoid meningitis, chiefly along vessels. The microscope showed in fresh serum preparations tubercle-like masses round about vessels, and at their bifurcation. This meningitis was the cause of death.

A microscopic examination of the cerebellar tumor showed the sub-cystic tumor to be mainly sarcomatous, cellular, and vascular, with foci of amyloid degeneration.

The family are all unusually healthy. Besides J. there are seven living children who are pictures of health. The father and mother are perfectly well, and always have been. The teeth of patient were normal, and he was a well-developed lad of rather hydrocephalic aspect. No suspicion of specific disease could be entertained in this case.

Case III.—Paul K., aged eight years. Seen in consultation with Dr. Malcolm McLean, of Harlem, on Nov. 17th, 1879.

In the past eight or nine months has suffered from diffused headache, attacks of vomiting, double exophthalmus, and staggering gait. Has been seen by many physicians, most of whom attributed the symptoms to "malaria." Child grew steadily worse in spite of treatment on this theory, and in August was taken to the Catskill Mountains. While there seemed worse; headache severe; staggered and vomited; was very weak. In September came under Dr. McLean's care, with above symptoms; no paralysis or impairment of intelligence. Parents stated that there had been no epileptiform seizures and no fever. Small doses of iodide of potassium caused improvement. Treatment suspended in October.

In last two or three weeks again worse; severe headache, much of it occipital and frontal. Great enlargement of the head and separation of the sutures. Marked exophthalmus—staggering gait and pseudo-paraplegia. A few days ago, there occurred sudden recession of the exophthalmus, and simultaneously there appeared a soft, fluctuating tumor or swelling in the right occipital region.

There is no history of injury to the head, and no evidences of tuberculosis.

Examination.—Child pale and intelligent; speech normal; vision seems good by finger and color tests, but the ophthalmoscope shows double neuro-retinitis (choked disk) of moderate degree. No facial or lingual paralysis. Co-ordinates perfectly well. All the cranial sutures are wide open; anterior fontanelle closed; forehead not very prominent; no exophthalmus now. In the right occipital region, in the location of the lambdoid suture, is a soft, compressible subcutaneous tumor, walnut size, whose contents beat synchronously with the pulse. The appearance of this swelling caused a relief to all symptoms except debility. It might be supposed that this swelling contained fluid derived from the hydrocephalus, but from its location I felt considerable doubt as to this.

Patient walked feebly in a staggering way; no paralysis or ataxia. Not the typical cerebellar "titubation."

I made the diagnosis of internal cerebral hydrocephalus, probably from tumor of the cerebellum compressing the aqueduct of Sylvius. I advised against puncture and aspiration of the newly-formed sac, and recommended larger doses of potassium iodide.

Dr. McLean wrote me Dec. 30th of this year:

"We immediately increased the iodide of potassium from ordinary doses (5 to 10 grains) to 25 and 40 grains; so that he received amounts of the medicine varying from 90 to 150 grains per day. The medicine never disturbed his stomach, and his symptoms were certainly ameliorated by the larger doses, which were continued for four months without interruption. The pains in the head were undoubtedly controlled by the medicine."

The child died in the early spring of 1880. The autopsy was made by Dr. McLean, under difficulties; the examination being followed watchfully by relatives, so that it was hurried and no notes were taken at the time. As the tumor was enucleated for future microscopic examination, its relations can only be given approximately, as recalled by Dr. McLean, who has very kindly written me as follows: "It was situated in the left hemisphere of the cerebellum, bulging out toward the anterior and middle inferior lobes, and pressing upon the aquæductus Sylvii. It seemed to have made appreciable pressure on all portions of the left hemisphere, but was buried in the tissue of the lobes I have mentioned. There was considerable softening around the tumor. . . ."

The growth was a fibro-sarcoma. It was well that the externally protruding sac was not punctured, for it turned out to be the extruded lateral sinus.

CASE IV.—Doctor F., aged 28, seen at Morristown, N. J., with Drs. Stephen Pierson and Frankenheimer, Nov. 6th, 1878. An enthusiastic hard-working practitioner, who left Charity Hospital last year. Had suffered from some "pulmonary trouble" during the past two years (probably extensive pleuritic adhesions).

In September of present year (about two months ago), was attacked with repeated causeless nausea and vomiting. Soon after a severe pain, paroxysmal in character, showed itself in the range

of distribution of the right occipitalis major nerve. This was aggravated by movements. After a few weeks, the pain became occipital and non-neural (*i. e.*, not within the course of any nerve-trunk). The pain has been the chief symptom, though occasionally absent for a week or ten days. The present attack or recurrence of pain is the fourth ; it is excruciating. Vomiting has continued more or less all the the time, and emaciation has progressed. No fever.

"He exhibited the ataxic gait for several months before he gave up practice at . . ., at times staggering in the street, and laying hold of fence-posts to keep him from staggering. He feared people would accuse him of drinking too much." [This is quoted from a letter Dr. S. Pierson was kind enough to write me Feb. 4th, 1887 ; it would thus seem that staggering was the *first* symptom in this case.]

Movement of the body and sitting up in bed aggravate pain. In the last two weeks there has been paresis of the right external rectus (sixth nerve), and diplopia. No hemianopsia. Has required a great deal of morphia and chloral to secure partial relief. There has been no fever, cough, or expectoration. No stiffness of neck or opisthotonus. Sight unimpaired. Has had no ordinary headache, and no dizziness. No bulbar symptoms. In last ten days both hands have been the seat of partial numbness (though not exactly as if parts were "asleep"). On 1st inst. (five days ago), Dr. Chas. S. Bull, of New York, found no lesion of the fundus ; there was only a congenital excavation of the disks.

Syphilitic infection and injury to head positively denied.

Examination.—Patient a little sluggish from narcotics. The head is not tender. Paresis of right sixth nerve ; third nerves normal. Disks distinctly choked with very tortuous vessels and minute hemorrhages in retina. Left cheek is perhaps paretic, but tongue points straight. The æsthesiometer shows marked anæsthesia : two points distinguished only at 40 to 50 mm. on the left side of forehead and left cheek. Finger tips are likewise affected, viz., on the right side points are differentiated at about 3 mm.; on the left at 5 or 6 mm. Simple contact and pinching are, however, well felt on face and fingers. Strength of grasp not tested. Patellar reflex absent. The patient was too feeble and wretched to be asked to rise and walk.

Diagnosis.—A tumor, probably tubercular, in the pons Varolii near the medulla oblongata. I was led into this error by the partial left hemianæsthesia.

The autopsy, performed by Dr. Pierson a few days later, revealed a solid (sarcomatous) tumor, occupying the greater part of the right cerebellar hemisphere to the median line ; much pressure on adjacent parts.

I am indebted to Dr. Pierson for additional points of value in the history of this case.

DIAGNOSTIC CONCLUSIONS.—The symptoms presented by these four cases varied somewhat in their grouping, yet were singularly harmonious. Let us consider them in order of constancy:

1. *Lesion of the optic nerve*, either as choked disks or secondary atrophy, was present in all the cases. This is in striking contrast to my experience with strictly cerebral (hemisphere) tumors, in which the optic nerves were affected only three times in ten cases. There is reason to believe, furthermore, that choked disk is usually an early symptom of tumor of the cerebellum.

2. *Headache* was present in all cases. It was distinctly occipital and paroxysmal in cases II., III., and IV. Occipito-frontal and never severe in case I.

In case IV., the pain was at first for several weeks a neuralgia of the occipitalis major nerve on the same side as the tumor. This is rather difficult of explanation.

3. *Vomiting.*—This was a very early symptom in cases II., III., and IV. It was a causeless vomiting, occurring almost always in the early morning; and was usually accompanied by severe occipital, or occipito-cervical pain. Indigestion did not occur. I have recently seen two other cases (in children) presenting the unmistakable symptom-grouping of cerebellar tumor, in which inexplicable vomiting was the only symptom (eyes not then examined) for months. One little fellow had had his stomach washed out for supposed gastric catarrh for a period of at least two months, even after some distinctly cerebral symptoms had appeared.

In case I. vomiting was never (?) present.

4. *The walk* was affected in all cases.

In cases I. and IV., there was cerebellar titubation. This was typical in case I. In case II. there was simply staggering with tendency toward the left.

In case III. a diffused staggering, somewhat like that of alcoholic intoxication, was observed. It is to be observed that case I. is the only one in which the lesion destructively affected the vermis in its caudo-ventral parts. In the other cases the vermis was more or less compressed.

Consequently, case I. goes to support Nothnagel's law that cerebellar titubation is characteristic of a considerable destructive lesion of the vermis.

5. *Motor Eye-symptom.*—In case I. there were various types of nystagmus. In case II. there was conjugate deviation (without vision) to the left; that is, away from the lesion, contrary to what occurs in hemisphere lesions.

6. *Paralysis.*—Slight, but distinct right-sided paresis (not of face or tongue) was present in case II. The other cases presented only a diffused loss of power.

7. *Ataxia,* strictly speaking, was present only in Case II., affecting the right upper extremity. This, as well as the paresis, was on the same side as the cerebellar tumor.

8. *Anæsthesia* was found only in Case IV. It was demonstrable on the left side of the face and on left fingers (opposite the tumor).

9. True *vertigo.* Subjective or static vertigo was not present in any case. It has always seemed to me that this was not a strictly cerebellar symptom, but one indicative of irritation, direct or indirect, of the acoustic nerves. Probably static vertigo also occurs from disturbances in the hemispheric circulation, as in galvanization of the cervical sympathetic. It might be added that, in the two living cases of cerebellar tumor above referred to, vertigo is absent.

10. The *bulbar symptoms* shown at the close of life in Case I. were evidently due to malnutrition (ischæmia) of the medulla by reason of the arteritis obliterans which affected the vertebral and basilar arteries and their branches.

11. *Psychic symptoms* were wanting in all cases.

THERAPEUTIC CONCLUSIONS.—Three of the cases (I. II. III.) teach us that many of the symptoms of cerebellar tumor may be controlled, and the disease retarded materially by the free use of iodide of potassium.[1] The

[1] Since the reading of this paper, a remarkable case of cerebral tumor cured by iodide of potassium has been published by Dr. B. L. Milliken, of Cleveland, in the Medical News, February 12th, 1887. All the symptoms dis-

remedy should be given in full doses, irrespective of the patient's age, from 100 to 300 grains a day, largely diluted. Even the vomiting is sometimes controlled by this, as shown in my living case whose stomach had been previously washed out.

PROGNOSIS.—This is not necessarily fatal. Case I. died of an entirely different pathological process, viz., general arteritis obliterans and multiple softening of the encephalic mass. Case II. died of tubercular meningitis. In both these cases, the growth had been completely checked, and gave rise to few symptoms beyond impairment or loss of vision.

It is, therefore, reasonable to hope, especially in children, for an arrest of the cerebellar disease, with some residua, such as blindness and slight motor disability.

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appeared except atrophy of the optic nerves. Dr. Milliken makes no attempt to locate the tumor, but it seems to me quite clear that it was a cerebellar tumor, with another lesion of the right temporal bone (above ear).