affection of the nervous system, and in which there are no special signs, such as may obtain in some functional cases, to establish the diagnosis. The following is a good illustration of what is meant:

A woman was seen in the out-patient room at the Queen Square Hospital, with a history from her doctor that there had been two attacks of pneumonia, one of which had been complicated by pericarditis, and that subsequently to this she had developed certain choreiform movements, with inability to speak. The patient was unable to stand or walk, and was apparently so weak that she could not even sit up. Severe spasmodic movements affected all of her limbs, together with her neck and trunk. This was clearly not the kind of case in which I could look for assistance from one or other of the signs of organic disease to which I have called your attention, and physical examination did not reveal any particular signs that may exist in hysteria. My suspicion as to the real nature of the case was, however, aroused when I tried to induce the woman to speak, for, instead of the articulatory difficulty so characteristic of chorea, with the rolling about of the tongue and other unsuccessful efforts at articulation, the patient made a grimace that did not recall to my mind anything that I expected in chorea, and spoke in a whisper without any of the articulatory defects for which I was prepared. With this clue to the real nature of her condition, I was next able to persuade her to control the movements of one or other limb, so as to enable me to test her tendon jerks and plantar reflexes, and with isolation and strong faradism, combined with suggestion, she rapidly recovered in a way that would have been quite impossible had true chorea accounted for the clinical picture.

The following is another case in which the diagnosis of functional disturbance had to be made without any one positive sign which could be regarded as pathognomonic of hysteria:

A young man was sent to University College Hospital because his illness had proved obscure and had not yielded to treatment in a provincial hospital. The history of his illness did not strongly suggest any known organic affection of the nervous system, but there was just that in it which made it possible that disseminated sclerosis might be found to account for the symptoms. I accordingly examined him in detail, having clearly in mind the possibility that this organic disease might be present, and, beginning with the cranial nerves, I reviewed the whole of his nervous system, with negative results, until the knee-jerks were tested and found to be exaggerated. This did not, however, prove that there was organic disease, as you will recognize from what has already been said on the subject. I next examined him for ankle clonus, but the result was negative. I was, nevertheless, still prepared to find evidence of organic disease, for when his lower limbs were uncovered my attention was attracted by the position of his great toes, notably by the left one, which was drawn back towards the dorsum of the foot in a manner that suggested the possibility that the extensor type of plantar reflex might be present. This did not, however, prove to be the case, for on testing them both plantar reflexes were found to be flexor in type, and not even on the left side was there any suspicion of the abnormal extensor type of response. The complete absence of any sign of organic disease of the nervous system made the diagnosis of hysteria certain, although there were none of what I may perhaps be permitted to call the positive signs of hysteria. On making the patient get out of bed to attempt to walk, the awkward character of his station and locomotion was such as one has never seen in any case of organic disease of the nervous system, and was so bizarre as to baffle accurate description. Moreover, on making him attempt to stand with his feet together and his eyes closed, the way in which he swayed and recovered himself, first in one direction and then in another, without opening his eyes or taking hold of any of the objects around him, made the diagnosis still more certain, although it cannot be said that it was ever in doubt after the physical examination in bed had been completed.

In this patient, as in the woman with the general choreiform movements, the assurance that the faradic current would cure his malady enabled his treatment to be conducted with complete success in a very short time, so that he also left the hospital able not only to walk, but to run.

In conclusion, let me ask you to accept two apologies, one for the general imperfections of the lecture, of which I am painfully conscious, and the other for my failure to present the subject to you without appearing to be egotistic, a state of things that is particularly distasteful to me, but which circumstances have made it difficult to avoid.

A Clinical Lecture

ON

SOME OF THE COMMONER SYMPTOMS OF CEREBELLAR ABSCESS.

DELIVERED AT UNIVERSITY COLLEGE HOSPITAL,

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In the last few weeks I have had under my care in the hospital two cases of cerebellar abscess which illustrated some of the clinical aspects of that disease well enough, perhaps, to justify me in saying something about them now. I propose first to tell you, avoiding as well as I can tedious detail, about the more instructive of the two cases, then rapidly to remind you of some of the symptoms of the disease, and finally to give you a short summary of the second case.

CASE I.

A. W., male, aged 25, was admitted July 25th, 1907. He had had a discharge from the left ear for three months. A month before admission a polypus had been removed from the ear. For the last week he had had severe earache and headache; the discharge continued.

The left meatus was filled by a large polypoid mass; there was tenderness over the mastoid but no swelling. There was a profuse discharge of offensive pus from the ear, no facial paresis, and the optic discs were normal. No abnormal nervous physical signs. During the day the temperature rose to 104° F.

The case was manifestly one of chronic middle-ear disease complicated by acute mastoidal suppuration. There was no evidence to suggest any intracranial complication. The same evening the radical mastoid operation was done in the usual way. The tympanum, the antrum, and numerous mastoid cells were found to be full of granulation tissue bathed in foul pus. The operation afforded practically no relief. The temperature continued to range between 100° and 103° F.; the patient felt ill and weak, and from time to time suffered much from headache. On the second and third days after the operation he vomited, but not again. The pulse from the first had been rather slow in relation to the temperature, varying between 65 and 95 per minute.

It was obvious, therefore, that some infective process was still going on. There was, however, nothing to indicate, with a degree of precision great enough to justify action, where the source of the symptoms lay. Moreover, it is not uncommon for quite severe symptoms to persist for some days after the mastoid operation has been done for acute disease, and then to subside spontaneously. There was no course open to us, therefore, but to wait, and to watch with the greatest care for the development of any sign which would localize the seat of the lesion.

The condition above described persisted unchanged for nine days, and then an ominous development took place. The temperature, which had scarcely been below 100° since the operation, fell abruptly from 104° to normal, with no accompanying improvement in the headache or the feeling of illness. Still no nervous physical signs were present. After a slight irregularity for a couple of days the temperature settled down to the normal line, and remained steadily there. This disappearance of the fever was in no sense an encouraging change, for the headache was still severe at times, the patient still felt very weak and ill, complained of giddiness if he sat up, and was definitely though very slightly drowsy.

The third stage of the slow unfolding of the symptoms began on the sixth day after the temperature had reached the normal—that is, on the fifteenth day after the mastoid operation. The first evidence of the change was that early in the morning the patient vomited. About 10 a.m. he was drowsy, and complained of occipital headache; the drowsiness did not seem more marked than on the day before. A note was made that the eye movements were good. Two hours later conjugate movements of the eyes to the left were noticed to be a little impaired, and were accompanied by slight nystagmus.

At 6 p.m. lateral movements of the eyes showed distinct nystagmus, and conjugate movement to the left was distinctly weak.

The optic discs were normal. The right knee-jerk and the

Fig. 1.—Temperature Chart of Case I.—A, Stage 1: symptoms: Headache and feeling of illness; average minimum pulse-rate, 70 per minute. B, Stage 2: Headache, feeling of illness, giddiness, drowsiness; average minimum pulse-rate, 62. C, Stage 3: Headache, feeling of illness, giddiness, drowsiness, ocular signs, vomiting; average minimum pulse-rate, 57.5. D, Stage 4: Rapid disappearance of symptoms; average minimum pulse-rate, 83.5. X, Mastoid operation. Y, Cerebellum explored; result negative. Z, Cerebellar abscess opened. O, First appearance of ocular signs and onset of vomiting. P, Appearance of cerebellar paresis.

left ankle-jerk were brisker than those of the opposite sides respectively.

The next day the temperature had fallen to 97.2°, and the patient had vomited again. For the first time the pulse-rate was below 64—namely, 56 per minute.

The weakness of leftward conjugate movement was more distinct; the leftward nystagmus was still more marked than the rightward; the reflexes were as before. There was, however, one additional sign—downward movement of the left eye was jerky and weak.

The next morning (August 11th)—that is, eight days after the temperature had come down to normal—the drowsiness and giddiness were more marked, the headache and the vomiting persisted; the temperature was 98.6°; the ocular signs were, perhaps, a little more marked, and it was noticed that when the eyes were at rest there was a tendency at times for the left to wander a little upwards and outwards, and a less distinct drift of the right down and out. The right knee-jerk was still brisk, the left distinctly diminished. The plantar reflexes were less easily obtained than before, but still flexor. There was no ataxy of the arms.

The nature of the lesion and its seat were now evident enough to justify further interference. That suppuration within the substance of the brain was present was shown by the classical and highly characteristic form of the temperature chart. A period of continued high fever had been followed by a period of normal temperature, which had passed in turn into a period of subnormal temperature; in each case the transition had been a rather sudden one, and the passage from the normal to the subnormal had been accompanied by a distinct fall in the pulse-rate and by the reappearance of vomiting. That the abscess was in the cerebellum was shown by the presence of the well-known triad of ocular cerebellar signs—namely, nystagmus, weakness of conjugate movements, and skew deviation. The first two were precise and well-marked, the last merely an elusive tendency, although definite enough to be of value in connexion with the others. That the abscess was in the left lobe of the cerebellum was shown by the nystagmus and weakness of conjugate movement being more marked when the patient looked to the left than when he looked to the right.

The most striking deficiency in the signs was the complete absence of optic neuritis, ataxy, and paresis. Of these, the absence of neuritis was by far the most serious, as ataxy and weakness seem to be relatively late cerebellar manifestations compared with the ocular signs. Negative evidence, however, is notoriously unreliable in diagnosis, as indeed might be expected from a very little consideration of general principles. The left lobe of the cerebellum was explored. No abscess was found.

Naturally enough, no relief followed, except a slight temporary diminution of the headache, due probably to a partial relief of the intracranial tension, which was very high.

For the next four days the patient's state remained much as it had been before the operation, except that there was a gradual worsening of the general condition. The temperature remained subnormal, the vomiting continued, the drowsiness, headache, and the ocular signs persisted. On the evening of August 16th, that is the fifth day after the second operation, there was a sudden increase in the severity of the headache, and the drowsiness became much more marked.

The following morning, August 17th, the patient was very drowsy, and resented any examination on account of the extremely severe headache. To the previously existing ocular signs it was clear that paresis of some individual muscles had been added, but, on account of the patient's mental state, no exact observation of them could be made. There was no optic neuritis. A very striking new feature was present in

the existence of a marked paresis of the left arm and leg. These limbs could scarcely be raised from the bed, and movements of them were clumsy and disordered. There was marked flaccidity of the paretic limbs, and the left knee-jerk was, as it had been for some time, much reduced. There was no trace whatever of any weakness of the face. It was clear that some considerable extension of the lesion had taken place, and the addition to the previously existing signs in the form of a typical cerebellar paresis with flaccidity, absence of ankle clonus or plantar extension, and with immunity of the face, made it practically certain that we had to deal with an abscess in the cerebellum, which had been overlooked, on account of its smallness or deep situation, at the previous operation. The temptation to suspect a temporosphenoidal abscess on the right side, where the patient also had middle-ear disease, was readily resisted, in view of the character of the paresis and the known ease with which a small deeply-seated abscess in the cerebellum may escape discovery.

The left lobe of the cerebellum was therefore explored again. It was found to be bulging into the opening in the skull and was tense and fluctuating. An incision allowed about 1 oz. of offensive pus to escape from a large cavity in the left lobe.

The next day the patient was very much better, the headache and drowsiness had practically gone, and the paresis was very much less.

After about a week very few physical signs remained. The ocular signs persisted longer than the paresis, of which practically no trace was present three days after the operation. The day after the operation the temperature rose to normal and the pulse-rate, which had been 56 per minute, rose to 100.

No further complications ensued, and convalescence progressed to complete recovery.

The chart (Fig. 1) shows very well the different periods into which the progress of the case was marked by changes in the temperature—first, continued high fever, then normal temperature, then subnormal temperature; and, finally, a return to the normal after the evacuation of the pus. I have recorded on it the time of appearance of some of the principal physical signs, so as to bring out the correspondence of the temperature changes with the extension of the disease.

The case afforded one the comparatively rare opportunity of watching from stage to stage the formal and systematic development out of obscurity of one of the most characteristic clinical pictures to be met with in medicine. It does not, however, of course, show by any means all of the physical signs which may be produced by a cerebellar abscess. It is, therefore, necessary to say something in general about the commoner and more characteristic of these.

Cerebellar abscess produces or may produce (1) general evidences of an infective process, (2) evidences of a lesion in the substance of the brain spreading with variable rapidity, but as a rule subacutely, (3) evidences of a lesion of the lateral lobe of the cerebellum.

1. GENERAL EVIDENCES OF AN INFECTIVE PROCESS.

When the case is seen from the beginning there will practically always be a period of onset during which the symptoms are those of an acute infective process—a rigor, high fever, headache, loss of appetite, wasting. It is well known that during this period, which in the case I have just related lasted under observation for ten days, there may be no evidence calling attention to the brain, and the case may come to be regarded as a septicaemia. Indeed,

occasionally the patient may succumb in this stage without any suspicion of the seat of the disease having been aroused. This is, perhaps, the rarest and most puzzling of all the clinical forms under which encephalic abscess may present itself—the septicæmic type—and when it is well developed it almost always escapes diagnosis.

Like certain other infective processes, those for example in bones and in closed mucous cavities such as the gall bladder or appendix, suppuration in the brain is apt to undergo a spontaneous arrest or quiescence after the acute onset. Thus arises a second very important clinical type—the latent abscess. On account, however, of the concentration of function in the brain such an abscess is very rarely so absolutely latent as for example an empyema of the gall bladder may be; it is true the infective symptoms may to a great extent disappear, but usually some evidence or other of impairment of function will be present, or some vague implication of the general state. The clinical behaviour of cerebral abscess is thus closely similar to that of pneumonia followed by empyema, and it is a very valuable rule in any case in which an acute illness has been followed by imperfect recovery with vague residual symptoms, to suspect empyema or abscess of the brain.

This form of latency—which we might call true latency—rarely occurs with cerebellar abscess; it must, however, be distinguished from the kind of pseudo-latency which occurred in Case I, where the fever disappeared without any other improvement whatever. This is in no sense due to quiescence of the infective process, but rather to the transformation of one of the symptoms by the progress of the disease. In Case II, presently to be related, partial latency of general symptoms was present, as will be seen, and would have given rise to difficulty had the localizing signs been at all obscure.

2. GENERAL CEREBRAL SIGNS.

Of the three familiar signs of increasing intracranial tension—headache, vomiting, and optic neuritis—very little need be said. Optic neuritis is the most significant when it is present. In spite of its great frequency in the more chronic cerebellar lesions it is often absent in cases of abscess. In neither of the cases reported here was it present; and its absence is of no value as evidence. Vomiting is a more common symptom, so that its complete absence throughout would be distinct evidence against cerebellar abscess. Of the headache we may say that it is by far most commonly occipital and practically never lateral, so that the substitution of occipital headache for the lateral headache so common in acute mastoid disease is good ground for suspecting the development of intracranial suppuration. Giddiness is a sign both of intracranial pressure and of cerebellar disease. In the former case, and sometimes in the latter, it is quite unsystematized, but when it is due to cerebellar disease it sometimes shows constant characteristics, objects during the attack seeming to the patient always to move in a definite direction. Wasting may possibly be regarded as an evidence of cerebral involvement rather than a product of the infective condition, for in some of the latent cases it is the only symptom. Apparently causeless profound wasting in an adult should always make one canvass the possibility of intracerebral suppuration.

The mental state is one of the most important of this group of symptoms. All stages of impairment of consciousness from the slightest to profound coma may be seen, the latter, however, usually only in very advanced cases or in the very acute fulminating varieties.

Perhaps the commonest early change is a very slight dullness, a disappearance of spontaneity, in which all the

less important matters of life are allowed to slide as not being worth the painful effort of attending to them. This painfulness of any kind of mental or physical activity is the keynote to the situation, and as the case advances it becomes more and more accentuated. In the early stages one may often notice a certain dazed expression which is no doubt partly the product of pain and partly that of drowsiness; one comes to recognize it as a rather characteristic feature. While the lethargy and indifference gradually deepen, as long as the patient can be made to respond it will be found that the intelligence is quite normal; it is quite unusual for any true delirium or confusion to develop, as is so common in meningitis or in other conditions which produce a diffuse involvement of the surface of the hemispheres. More important probably in the production of confused mental states is the rapidity of the process, for in the stage of acute onset of abscess there may be confusion which ultimately disappears.

3. LOCALIZING CEREBELLAR SIGNS.

This is by far the most important group, for even in the most latent cases, when the first two groups may be almost completely absent, the local signs are usually to be obtained, as was to be seen in Case II. The localizing signs of cerebellar disease may be divided into those

which are due to the involvement of the structures outside the cerebellum and those which are due to the involvement of the latter itself—the former may be called extrinsic, the latter intrinsic signs.

Extrinsic Signs.

In the posterior cranial fossa the structures other than the cerebellum which may be involved are certain cranial nerves and the pons and bulb.

Cranial Nerves.

The last six of these are those which have a more or less considerable part of their course in the posterior fossa. It is clear that they fall naturally into three groups (Fig. 2)—an anterior or superior, including the fifth and sixth nerves; a middle group, including the seventh and eighth; and a posterior, including the ninth, tenth, eleventh, and twelfth. The posterior group is very rarely involved by abscess of the cerebellum in such a degree as to be of diagnostic value. Some degree of defect in articulation is, however, not uncommon, but naturally enough it is difficult to decide how much of this may not be due to the mental state. The second group is of practically no value in diagnosis, seeing that involvement of it is much more likely to be due to the middle-ear disease which has caused the cerebellar abscess than to the latter itself. We are left, therefore, with the anterior group.

It might have been expected that affection of the fifth cranial nerve would not be uncommon in cerebellar abscess. The sixth nerve, which is so near, is very commonly involved; the abscess is usually far forward in the lateral lobe; indeed, probably farther forward than is often thought, since we are now coming to know how frequent is infection of the cerebellum through the internal ear, yet as far as present experience goes it would appear that the fifth as a rule escapes. It is probable, however, with more exact methods of determining sensory disturbances of slight grade we may ultimately find that changes in the cutaneous area of the trigeminal are not uncommon in cerebellar abscess.*

*It has been shown by Mr. Morriston Davies and the author that very slight changes in cutaneous sensibility are demonstrable and definable in lesions of sensory nerves, so slight as to have produced no other physical signs. Such modifications can, for example, be shown in the area of the trigeminal with sufficient accuracy to be of diagnostic importance long before any insensitiveness to the usual tests for cutaneous sensibility is present. (See *Review of Neurology and Psychiatry*, October, 1907.)

Fig. 2.—Diagram of anterior wall of posterior fossa to show grouping of cranial nerves. VI, V, Anterior group of cranial nerves. VII, VIII, Middle group of cranial nerves. IX, X, XI, XII, Posterior group of cranial nerves. LS, Lateral sinus. SPS, Superior petrosal sinus. IPS, Inferior petrosal sinus.

The case is quite different with the sixth nerve. Paresis of the external rectus on the same side is the commonest isolated sign of cranial nerve involvement met with in cerebellar lesions. The frequent combination of it with weakness of conjugate movements to the same side makes it, however, sometimes a little difficult to detect. Unfortunately, the diagnostic value of weakness of the external rectus is but small. It occurs not uncommonly in acute mastoiditis, and even otitis media (Gradenigo's sign), and is, therefore, of much less significance than is a weakness of conjugate movements, in which are implicated the external rectus of the diseased side and the internal rectus of the other.

Pons and Bulb.

It is not possible here to deal in detail with the various signs which may result from pressure on these. It is enough to state that such pressure may cause a hemiplegia of the upper motor neuron type, usually implicating leg and arm only, and accompanied by exaggeration of deep reflexes, ankle clonus, and extensor plantar response. Such a hemiplegia is much less likely to occur in abscess than in tumour, and it implicates the limbs on the side opposite to the lesion, as the pressure is above the decussation of the pyramids.

Intrinsic Signs.

The fact of the cerebellum being an organ concerned with the regulation rather than with the origination of movement, gives to the signs of lesions of it a quality characteristically different from lesions of the direct motor path. The contrast is well brought out when we remember in cerebellar lesions the great rarity of convulsive attacks, the absence of rigidity, and the fact that paresis is a late sign of advanced disease and scarcely ever amounts to paralysis.

We must now try to get some broad general outline of the motor effects of cerebellar lesions, leaving aside all confusing detail and dealing only with the common, obvious, everyday features.

The motor effects of a cerebellar abscess may be classified as follows:

1. Ocular signs:
 - Nystagmus.
 - Weakness of conjugate movements.
 - Skew deviation.
2. Signs affecting the limbs:
 - Inco-ordination.
 - Paresis.
 - Hypotonicity.

Ocular Signs.

It is not possible to enter into the discussion of this group, and I can only allow myself to refer to two points in connexion with it. You will have noticed in the description of Case 1 that skew deviation is described as occurring upwards and outwards on the side of the lesion and downwards and outwards on the other, and that this differs from the deviation usually found in cerebellar tumours, which is upwards and inwards on the side of the lesion. The deviation, such as occurred in Case 1, I have seen in other cases of cerebellar abscess, but never as more than a slight and transitory manifestation, or in any way approaching the marked and persistent deviation sometimes produced by tumours. Unless the case, therefore, is constantly under observation the phenomenon may escape notice altogether.

The second point I wish to call your attention to in connexion with this ocular group of signs is the matter of the diagnosis of suppuration in the internal ear. This condition may produce headache, nausea, vomiting, vertigo, disturbances of equilibration, and nystagmus, and is therefore an important cause of difficulty in the diagnosis of cerebellar abscess. As you know, a great deal of work has recently been done upon the diagnosis of labyrinthine suppuration, and many details have been made out whereby cerebellar nystagmus and labyrinthine nystagmus can be differentiated.* I shall only trouble you with one—namely, that while the former is most marked when the patient looks towards the side of the lesion, the latter is in the great majority of well-marked

cases in the opposite direction. The frequency with which suppuration in the labyrinth is now known to occur justifies me in reminding you how important it is when carrying out the radical mastoid operation, which is the preliminary to the treatment of intracranial complications, to inspect with great care the inner wall of the attic in order to detect, if such be present, the characteristic sinus leading to the external semicircular canal.

Signs Affecting the Limbs.

Inco-ordination.—Disturbances of the normal co-ordination of movement may be demonstrable in several ways:

- (a) During pointing to a given object.
- (b) In the gait.
- (c) During rapidly reciprocating movements, such as alternating pronation and supination of the forearm.
- (d) During complex habitual movements involving several joints.
- (e) By the effects of the sudden removal of resistance to movement.

(a) When a patient with cerebellar ataxia of the arm is asked to touch the tip of his nose the movement is carried out with a characteristic uncertainty. The tremor involves the limb on the same side as the lesion; it does not increase towards the end of the movement; it is usually fairly coarse, but is rather variable in this respect; it is present only during movement and disappears when the patient is asked to hold his hand out in front of him.

(b) The cerebellar gait can rarely be observed in cerebellar abscess on account of the grave general condition, but when the patient is well enough to walk he shows the characteristic wide base of support, staggering towards the side of the lesion, with, perhaps, exaggerated recovery towards the opposite side, difficulty in turning, etc.

(c) A rapidly alternating movement of pronation and supination of the forearm was called "diadokokinesia" by Babinski, who described the disturbance of it which occurs in cerebellar lesions. This disturbance, as all the other intrinsic motor signs of cerebellar disease, occurs on the same side as the lesion. The patient should be asked to carry out the movement on the sound side first, so as to give some standard of the normal smoothness and rapidity of the rotation, and to ensure that he knows what is wanted. When he now tries to perform the same movement on the other side, the rotation is slow and extremely irregular, and he tries to compensate for this deficiency by movements of the elbow and shoulder, which on the normal side show no tendency to take part. This disturbance is called "dysdiadokokinesia"; it is a fairly common cerebellar sign, and is of considerable localizing value, as it is a delicate test for defective co-ordination.

(d) When a normal person lying down is asked to straighten the lower limb from a position of flexion of both knee and hip, the movement executed is single, the extension of the knee and hip occurring together. Sometimes in cerebellar lesions this synergic movement is interfered with, and the patient straightens the knee first and then the hip.

(e) If when a normal person is asked to flex the elbow strongly against resistance the resistance is suddenly removed, there is a sharp, short jerk of further flexion, and then the movement stops. In a limb showing cerebellar inco-ordination a similar manoeuvre often shows a very striking abnormality. When the resistance is removed, flexion of the elbow is continued until the hand touches the shoulder. In some cases there is at first a rapid increase of the flexion, then a slight pause, and finally completion of the movement. The abnormality always occurs on the side of the lesion.

Hypotonicity and Paresis.—I have already mentioned the characteristics of cerebellar paresis in describing Case 1, so that I need do no more than enumerate them. Such a paresis is on the same side as the lesion, does not involve the face, is not accompanied by rigidity, tends to involve the larger movements rather than the finer, and is not associated with ankle clonus or extensor plantar response. It is, however, not enough to say that there is no rigidity; there is, in fact, very often a striking flaccidity of the paretic limbs, as can be shown by passive flexion of the hip with extended knee, or by the possibility of producing slight hyperextension of the knee (knee angle sign).

* Valuable information on this subject is to be found in the writings of Bárány and of Neumann, especially in an article of the latter in the *Archiv für Ohrenheilkunde*, Band 67, and in the recent monograph of the same author entitled *Der Otitische Kleinhirnsabszess*. (Deuticke: Leipzig and Wien, 1907.)

Reflexes.

The deep reflexes are often affected by intrinsic cerebellar lesions in such a mixed way as to produce a picture almost characteristic in its irregularity, but very difficult to generalize. We may say in a broad way that any reflex may be exaggerated or reduced, and both changes may occur with different reflexes at the same time or with the same reflex at different times; and that, therefore, the localizing value of such changes is often slight as regards the side of the cerebellum involved. For example, the knee-jerk on the side of the lesion is often exaggerated, but it may equally well be reduced. It is possible, however, to be more definite as to the absence as a sign of intrinsic cerebellar disease, of the Babinski toe phenomenon and its variants, the "fan sign," Schäfer's, Oppenheim's, and Remak's reflexes.

I may now shortly relate to you the symptoms and signs of a second case, as they show very well how some of the features we have been describing may be combined in an actual example.

CASE II.

E. G., a married woman of 29, was admitted September 5th, 1907. She had been ill for six weeks, but no very definite account of the illness could be obtained. She had had a discharge from the left ear since the beginning of the illness. For the last three weeks she had had much headache, and had taken to her bed. At the beginning of this period she had had much shivering. At the time of admission a large subcutaneous abscess was present behind the ear. This was immediately incised. Some hours later systematic examination gave the following results: The patient was decidedly lethargic, and spoke with a blurred articulation; she answered questions intelligently, and understood all that was said to her. The temperature was normal, and the pulse 90.

Ocular Signs.—Conjugate movements to the left were distinctly weak, those to the right normal. The left eye did not move to the left as well as to the right, so that there was probably some special weakness of the left external rectus. There was no deviation of the optic axes when the eyes were at rest, but the left eye moved upward more readily than the right, and downward less readily. Nystagmus was present during all movements except downwards, and was most marked when the patient looked to the left. There was no optic neuritis.

Reflexes.—Left knee and Achilles jerks exaggerated on the left side. Plantar reflexes very feeble, and showing slight flexion. Other reflexes normal.

Motor.—There was a very little weakness of the left arm and leg, so slight, indeed, as probably to be without significance. Movements of left arm showed distinct ataxy, movements of right normal. Marked dysidiadokokinesis was present on the left side. Flexion of elbow against resistance with sudden release showed, on the left side only, a marked continuation of the movement.

It was, of course, obvious at once that a lesion of the cerebellum was present, and operation was carried out. A large extradural abscess was found in the posterior fossa, suppuration in the lateral sinus, and a large abscess in the left lobe of the cerebellum. These were drained, and the patient did well for ten days, when she began to develop general suppurative meningitis.

The case shows well how the general and cerebral symptoms may be latent, and yet the intrinsic cerebellar signs be definite enough to establish the diagnosis with certainty. It also affords an interesting contrast with Case I in the distribution of the signs. In both the ocular signs were well marked, and in neither was there optic neuritis. But whereas in the first case the motor signs in the limbs were those of *paresis* alone, in the second case the motor signs were those of *inco-ordination* alone.

I shall make no attempt to deal with the treatment of cerebellar abscess, but confine myself to a short remark on each of two points in connexion therewith. In the first place, it seems even yet not to be fully recognized how extremely urgent is the need for prompt operation in all cases of cerebellar abscess. In fact, in any case of increasing intracranial tension in the posterior fossa, so great is the risk of sudden death from respiratory failure, that as soon as the diagnosis is made the case should be looked upon as quite as urgent as one of perforated gastric ulcer or of rupture of the spleen. This danger bears no relation whatever to the severity or mildness of the symptoms and signs.

As regards actual operative technique, the only point I wish to draw your attention to is that the chance of failing to find a small and deep-seated cerebellar abscess is considerable, and that in such a case unquestionably the best course to pursue is to remove a considerable area of

bone and incise the dura freely, so that if an abscess is actually present it can extend more freely towards the surface than elsewhere, and the appalling danger of sudden pressure on the medulla thus be minimized.

THE COLONY AND BROMIDE TREATMENT OF EPILEPSY.

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DURING the past three and a half years I have had charge of C.U.S.S. Colony and Epileptic School for boys at Starnthwaite, and the results obtained from what I might call the radical use of bromide are, I think, worth publishing.

The treatment is based on the assumption that early epilepsy is far more frequently the direct result of peripheral irritation than of Gowers's "abnormal state of chemical nutrition, whereby the molecules are more easily detached and their atoms yield with undue facility to the attraction of the adjacent oxygen," and that the liberal use of bromide prevents this peripheral reflex from disturbing the cerebral harmony and saves the brain from contracting the bad habit of epilepsy on the slightest sensory disturbances. The dose of bromide given is that amount which is necessary to control the fits. This is done in some cases by 40 grains a day; oftener 100 grains is required, and as much as 300 grains has been necessary.

During 1903 we treated four boys—two of them we have discharged as having their epilepsy arrested, though still under observation. One has been removed by his father as cured. He has had no fits and no bromide for a year. The other boy was considered at first unsuitable for treatment, but was put on bromide in March of this year, and has had his fits reduced from ten a month to one in eight months.

	Fits in 1903.	Fits in 1904.	Fits in 1905.	Fits in 1906.	
W. C. L. ..	84	11	1	0	Removed by father.
A. M. ..	267	0	2	0	Working on farm.
A. B. ..	25	0	1	1	Working on farm.
E. T. ..	32	72	99	27	

The case of E. T. shows average fits for 1903 to be 32. During 1904 he had no treatment and the fits numbered 72; in 1905 he had 99 fits without treatment, but during 1906 he was treated and his fits were reduced to 27, and of these only 4 occurred during the time he was taking bromide. The bromide was not started until March 24th.

Of the 10 cases admitted during 1904, 6 were placed on bromide with the following results:

	Fits in 1904.	Fits in 1905.	Fits in 1906.	
H. V.	197	3	4	Still in school.
F. S.	60	5	3	Still in school.
A. Y.	130	1	0	Working on farm.
R. B.	238	0	0	Working on farm.
F. W.	47	11	0	Working on farm.
F. B.	72	16	27	At school.

Two were discharged as mental cases and 2 considered unsuitable for treatment.

Mr. Colyer, our Superintendent at Starnthwaite, keeps most accurate daily, monthly and yearly records of the dose of bromide given, and the number of fits that occur in each day, month, and year. I give here a few cases from his year-book. The first is most instructive in several points. I did not consider the case a very suitable one for bromide, and although he was put on it for a few months it was stopped. Why, I am not quite clear. Bromide was resumed in March, 1906, and his case now affords us three periods—1903-4 partial bromide, 1905 no bromide, 1906 with bromide. The records of the other cases explain themselves.