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.

CASE E. T.

Year.	Jan.		Feb.		March.		April.		May.		June.		July.		August.		Sept.		Oct.		Nov.		Dec.		Total Fits for Year.
	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	
1903	—	—	—	—	—	—	—	—	—	—	2	—	5	—	4	—	4	—	1	—	0	—	0	—	16
1904	—	0	—	4	—	5	—	4	—	5	—	4	—	19	—	5	—	0	—	4	—	17	—	5	72
1905	—	2	—	13	—	3	—	5	—	10	—	9	—	6	—	5	—	10	—	10	—	10	—	16	99
1906	—	14	—	9	20	3	40	0	40	0	40	0	40	0	40	0	40	1	40	0	40	0	40	0	Since Br. started 4
1907	40	0	40	0	40	0	40	0	40	0	40	0	40	0	40	0	30	0	20	0	30	0	20	0	0

CASE A. M.

Year.	Jan.		Feb.		March.		April.		May.		June.		July.		August.		Sept.		Oct.		Nov.		Dec.		Total Fits for Year.
	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	
1904	Gr. *	4	Gr.	31	Gr.	33	Gr.	36	Gr.	47	Gr.	29	Gr.	55	Gr.	20	Gr.	50	Gr.	0	Gr.	10	Gr.	2	277
1905	60	0	60	0	60	0	60	0	60	0	60	0	60	0	60	1	60	0	60	0	60	1	60	0	2
1906	60	0	60	0	60	0	60	0	60	0	60	0	60	0	60	0	60	0	60	0	60	—	—	—	0

* Admitted January 13th, 1904.

CASE H. V.

Year.	Jan.		Feb.		March.		April.		May.		June.		July.		August.		Sept.		Oct.		Nov.		Dec.		Total Fits for Year.
	Br	Fits.	Br	Fits.	Br	Fits.	Br	Fits.	Br	Fits	Br	Fits.	Br	Fits.	Br	Fits.	Br	Fits.	Br	Fits.	Br	Fits.	Br	Fits.	
1904	—	—	—	—	—	—	—	—	—	44	—	24	Gr. 20	44	Gr. 20	18	Gr. 20	8	Gr. 40	1	Gr. 40	0	Gr. 50	1	133
1905	50	0	60	1	60	0	60	1	60	0	70	1	80	0	80	0	70	0	60	0	60	0	60	0	
1906	60	0	60	1	60	0	60	0	60	0	70	2	70	0	70	0	70	0	70	0	—	—	—	—	3

CASE B. B.

Year.	Jan.		Feb.		March.		April.		May.		June.		July.		August.		Sept.		Oct.		Nov.		Dec.		Total Fits for Year.
	Br.	Fits	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.	Br.	Fits.			
	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—		
1904	—	—	—	—	—	—	—	—	—	—	18	—	71	20	15	40	8	50	3	60	1	60	3	119	
1905	70	0	70	0	70	0	70	0	70	0	70	0	70	0	70	0	60	0	50	0	50	0	50	0	0
1906	40	0	40	0	40	0	40	0	40	0	40	0	40	0	40	0	40	0	40	0	—	—	—	—	0

Is the condition we recognize as epilepsy a disease or is it only symptomatic? Is there such a malady as idiopathic epilepsy? Epilepsy must be regarded as the result of a disease or the perpetuated sequel of one, a vicious habit of brain being established. The only point on which there seems to be unanimity of opinion is that one fit predisposes to another, the oftener they occur the oftener they are likely to occur, until they become in a way self-perpetuating. Much has been written as to the relative importance of predisposing and exciting causes. For practical purposes it would be well to ignore predisposing causes, and, after all, their presence is a supposition. It is supposed that convulsions would not occur during teething unless there was a predisposing cause such as heredity, rickets, or nutritional instability. Yet in my cases rickets and heredity have been conspicuously absent. I can trace 33 per cent. of my cases to their origin during teething, and one of these cut teeth so early as the third month, and in others of which I have had personal knowledge teething was by no means delayed. Moreover, one has to think of the hundreds of eminently rickety children who never have a fit. In my experience the only constant factor in infantile convulsions has been the evidence of pain and unrest, and the number of cases of infantile convulsions that have gone on to epilepsy has been large.

Pain and nerve tension seem to so disturb cerebral

harmony that there is a furious outburst of energy giving rise to a display of sensory, vasomotor, and motor function constituting the fit. The fit is so obviously an outcome of cortex function that idiopathic epilepsy is regarded as a disease of that area. Epilepsy is a malady that lasts for many years, it is a malady that is very prevalent, it has afforded the fullest and best opportunity of exhibiting its pathology. Thousands of *post-mortem* examinations must have been made by the best pathologists on cases that ought to have demonstrated the lesions in its completed stage. Yet no single constant unvarying condition has been found. Pigmentations and vacuolation cells, changes in the perivascular spaces, induration of the pes hippocampi have been given up as the causes of epilepsy; degeneration of the cells in the second cortical layer has not been verified. The tendency now is to assume that an unseen and invisible lesion is present, a chemical one—one adduced by Gowers and accepted generally because Gowers has said it, and that is, "an 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." If this is the cause of epilepsy it is not surprising that pathologists have been unable to see it. Briefly, the reasoning seems to be that epilepsy is, from a study of its symptoms—aura, loss of consciousness, tonic and clonic spasm, convulsions, coma, drowsiness and sleep—so obviously a

display of the functions of the cerebral cortex, that the disease must be seated there, and this is supported by experimental investigations, especially those of Bechterew. He proved that convulsions with tonic and clonic spasm followed by coma just like epilepsy are produced on electrical stimulation of the brain cortex. But does it follow that the disease must be in the cortex because the symptoms are those of cortical function and they have been produced by stimulation of the cortex? I think not; Bechterew's experiment amounts to this, that a healthy brain gave epileptic symptoms on stimulation. This is significant; there are two factors—healthy brain and stimulation—and they give epilepsy. No need of heredity, no need of rickets, no need of abnormal chemical nutrition, with detaching molecules and yielding atoms. I am of opinion that we have in Bechterew's experiment the explanation of epilepsy as we find it in everyday practice—a healthy brain bursting out into fury owing to its over-stimulation by sensory impressions poured into it from one or more or varying sensory areas, the pain and tension of teething acting like the electrical stimulation: the brain gets teased, less tolerant, almost vicious in its habits, so that it will reply, long after teething has passed away, to other sensory disturbances with an epileptic fit, thus explaining how so many cases of infantile convulsion go on to what is called idiopathic epilepsy. Epilepsy thus becomes a reflex act in its origin and a cerebral vice in its fuller development and perpetuation.

This view of epilepsy in its origin being mainly, if not wholly, a reflex disease, brings into harmony many apparently hopelessly divergent facts regarding its etiology and treatment; for there are undoubted cases on record where correction of errors of refraction has cured the epilepsy, and of epilepsy ceasing after the nose and throat have been cleared of adenoids. I have had a case where constipation was the cause and cascara the cure. Osler mentions a case where an undescended testis was the cause of epilepsy; pressure on it produced an attack; its removal cured the disease. Gynaecologists have seen the origin of epilepsy in ovarian irritations. Oculists have found it in eye-strain. Dietetic specialists have traced it to the Sunday dinner, while the genito-urinary surgeon accounts for it all by a floating kidney. They are each and all right in their observation if they would only allow that causes might be found outside their area requiring treatment outside their routine.

TREATMENT.

Treatment of epilepsy is to remove the cause where this can be done, and, where it cannot, render it inoperative. The need of classification is again obvious; there are some cases that must be ruled outside the hope of much betterment by any treatment—developmental cases, cases arising in imbeciles, in the microcephalic, in inoperable tumour cases, and probably the confirmed epileptic, the individual who has had severe epilepsy for twenty or thirty years, and whose mind, brain, and body are hopelessly wrecked by repeated fits. His time for treatment is past. Peripheral, traumatic, emotional, and toxic causes give rise to about 60 or 70 per cent. of all cases in childhood and youth, and are, in my opinion, curable if treated early and long enough.

Bromide treatment is necessary and curative in peripheral, traumatic, emotional, and toxic cases. I think it was Sir T. R. Fraser who first demonstrated that the action of bromide was one of allaying reflex action. A stimulus of given strength was applied to the leg of a frog, general muscular contractions followed; under bromide the same stimulus produced less contraction, while more bromide limited the contraction to the side of the body that was stimulated. A larger dose of bromide limited the contraction to the leg only.

Epilepsy being due to severe, prolonged, or repeated sensory stimulation of brain, I use that amount of bromide that is necessary to stop the sensory stimulation. The amount required to do this varies exceedingly. Eighty grains a day is the average, but so small a dose as 60 grains has been sufficient to arrest epilepsy of an intensity that was to be estimated by the occurrence of 267 "grand mal" seizures a year. Among the boys at the colony now, 130 grains a day has been the highest dose given so far; but I have given 320 in private practice and arrested the disease. The method is to give 20 grains night and morning, and increase by 10 grains

per day as often as fits recur. Epileptic boys stand bromide very well, 80 or 100 grains per day are taken without them showing any sign that they are taking a drug; 150 or 200 grains per day begins to interfere with the equilibrium—their walking becomes uncertain, and they ought to be kept in bed. With larger doses, swallowing and organic reflexes are interfered with. Adults do not stand bromide nearly so well. The boy that had 320 grains had sordes on his lips and gums, his breath was foul, swallowing was difficult, there was slight hæmatemesis and melaena, respiration was shallow, and râles were heard. The fits, which were numbering eight and ten a day, stopped, the bromide was gradually reduced, and the boy had no more fits for two years. I detected no toxic action on the heart; it remained regular throughout, no dilatation, little loss of clearness about the sounds. The toxic action of potassium salts on the heart to me seems a myth. I use citrate of potash in large doses—60 to 90 grains per day—for long periods for gravel, migraine, and some eczemas, and fail to find the slightest evidence or hear patients (often old) complain of any symptom of heart weakness. On the other hand, with bromide I find respiration does get embarrassed, slow, shallow, or at times halting and deep, and accompanied with râles and rhonchi. I find in Martindale's *Extra Pharmacopoeia*, 1906, Dixon is quoted as follows: "Potassium salts administered per os are non-toxic; they are excreted faster than they are absorbed."

Bromide rash may well be ignored. I use the best English-made bromide, and rashes have never given me any trouble. Of 40 cases taking bromide now, only 3 show any rash; in 2 it is very faint, a few papules on the forehead. In 1 it is severe—takes the form of boils, forming ulcers with purpuric-looking surrounding skin. This particular boy had an attack of the same thing about a year ago, and it disappeared in three weeks without the bromide being knocked off, and without anything in the way of treatment except a soothing antiseptic ointment. Other objections to bromide are that it makes them look blue and blotched, and miserable, starved, dazed, and ugly. This is a gross libel; such appearances are not due to bromide. I had an epileptic under my observation, but not under my treatment, who very well illustrated this description, but he never took bromide. He died a suicide, and the coroner's jury returned a verdict of death due to temporary insanity arising from business worries.

For those few cases that may have their origin in absorption of toxic products from over-eating, from unsuitable food, or from constipation, diet is of importance, but for the bulk of cases diet might be summed up in—three meals a day, everything fresh, everything limited, flesh never oftener than once a day and three times a week is ample. In the salt-free or purin-free diet I see no likelihood of good being done.

A few more years of Starnthwaite experience will prove whether epilepsy in the young, when taken early and treated assiduously, can be permanently arrested. The outlook at present is very hopeful, and, if we succeed, then the question becomes a very large one, and one, I think, for the State to deal with.

A CASE OF HYSTERICAL SOMNAMBULISM, SHOWING ABNORMAL ACUITY OF VISION IN THE SOMNAMBULISTIC STATE.

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A GIRL, aged 21, a teacher, typewriter, and now a student of music, was sent to me on July 29th, 1907, complaining of sleep-walking of a month's duration. Having at that time temporary charge of beds in the Birmingham General Hospital, with the consent of her doctor I took her in for the sake of observation, and it was during her stay in that institution that the events recorded below took place.

Family History.

The patient's mother died aged 29, but the cause of death was not known. Her father, at the age of 26, committed suicide by shooting himself in her presence. Two brothers are living; one is in good health, the other