—the solid organs swollen and purple with excess of blood content, and, when cut, running out like overfull sponges. The splanchnic nerves, it will be remembered, run down within the thorax.

If paroxysmal tachycardia can be really determined by dilatation of the splanchnic area and the consequent greatly diminished supply of blood to the left ventricle, it is easier to understand the usually sudden manner of its inception and termination.

Other remedies suggested for paroxysmal tachycardia are atropine (by Dr. Herringham) and vascular dilatants (by Sir James Barr). It is interesting to note that, although in this case the blood pressure was low, yet the patient seemed to do decidedly better when on a mixture containing spirits of nitrous ether.

A RED LINE ON THE GUMS.

BY

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DURING the last three years it has been my privilege to examine a very large number of men (about 900) employed at works in which lead ores are handled, and with such an opportunity one would not be surprised at the possibility of finding some new feature associated with the poisoning symptoms classified under the heading of "plumbism."

It is astonishing what a large number of cases have been placed in the category of plumbism since the passing of the new Compensation Act, which, under circumstances associated with fewer temptations, both to the workman and his doctor, would most probably have been put down to alcohol, syphilis, rheumatism, or some such disease.

The workmen are drawn from a stratum of society composed of unskilled persons, who have been brought up amidst surroundings that do not conduce to any mental or moral elevation, with the result that their resistance to temptations of an alcoholic nature is of the feeblest. With a diminished income due to alcoholic excess, this class of workman does not obtain the necessary quantity of proper food to enable him to resist disease, which, if it be associated with the blue line on the gums, is most likely classified as plumbism.

What is this blue line? In a large number of the older "hands" one nearly always see a dark, dirty line, which, with the application of a suitable tooth powder on a good toothbrush would rapidly disappear; this is not the lead line. The typical lead line is seen to be just under the surface of the edge of the gum, and situated in the papillae, and under a magnifying glass presents an interrupted appearance. These papillae in some cases become enlarged almost to the extent of rupture, and the question naturally arises, What is the cause?

The following case is very interesting as bearing on the question:

A. B., aged 29, has been engaged at the same works for about ten years. On August 2nd, 1909, he lost the sight of his left eye, which had only troubled him for a few hours previously by becoming excessively watery. He gave a history of an attack of colic (which may have been due to lead) in May, 1909, but which did not last long.

He is rather anaemic; the left eye is damaged by a haemorrhage into the anterior chamber, which completely interferes with his sight. There is no paralysis. On testing the saliva the potassium sulpho-cyanide reaction is well marked, and to those who have had much experience of this test such a fact will be surprising. On the gum a marked blue line, with which is associated a line of very enlarged papillae, in many places enlarged to the extent of rupture.

Is this engorgement due to the presence of lead in the system? About two years ago, when examining the men at a works in the district, I was particularly struck with the fact that a large percentage of *their gums showed a distinctly red line*, in marked contrast to the men at other similar works, so much so that I drew the attention of Dr. Legge, of the Home Office, and Dr. Collis, one of the inspectors, to the matter, and the latter has corroborated my observation. This red line is, in my opinion, due to an enlargement of the papillae at the edge of the gum, and, peculiarly enough, was seen in lads who had been only fourteen to twenty-eight days at the works, as well as in the outside men, such as masons, whose contact with the fumes occurred only when the latter were diluted.

The works are situated on the bank of a river, and are nearly surrounded by works of many sorts, so that the atmosphere is most probably charged with particles of an irritating nature, and no doubt it is this source of irritation, especially in the case of boys newly arrived at the works, that causes the engorgement, to which the lead finds its way, and on which it leaves its mark.

The conclusion that one naturally arrives at from such facts is that the so-called lead line is no proof of lead in the system, but that it appears in workers whose gums have been irritated by particles other than lead.

The Lettsomian Lectures

ON

THE CEREBELLUM AND ITS AFFECTIONS.

DELIVERED BEFORE THE MEDICAL SOCIETY OF LONDON,

BY

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LECTURE III.

NON SUPPURATIVE ENCEPHALITIS.

NON-SUPPURATIVE encephalitis may occur in adults in association with infective diseases, but has been especially described in children after one or other of the acute diseases to which they are so liable. The mode of development of the acute ataxia in a child previously healthy, with the subsequent gradual improvement, after an attack of measles or some similar disease, are points that are highly suggestive of either encephalitis or acute degeneration, due to the action of some toxin on the nerve elements of the cerebellum. Such evidence cannot, however, be accepted without *post-mortem* confirmation, so that it is satisfactory to find that when some such cases have come to necropsy, although, as a rule, this has been at a remote period from the acute attack, the evidence of sclerotic changes in the cerebellum has been such as to justify the original diagnosis of encephalitis.

Prominent among those who have directed attention to this class of case in children is my colleague, Dr. F. E. Batten, who, in a paper on ataxia in childhood, brings out the main clinical features of the affection as it occurs in children. A child, previously healthy and of good physical and mental development, becomes ill of some acute febrile disease, which may or may not assume a definite recognized type, such as that of one of the exanthems. Loss of consciousness may supervene, but general convulsions are exceptional. After a few days, in which the child appears to be making a normal convalescence, it is sat up, but is found to be unable to maintain itself in this posture owing to loss of balance. The child's hands reveal marked incoordination, and, if supported on its feet, the lower limbs are found to be wildly ataxic. Moreover, some articulatory defect of speech may be noticed. The general health improves in the course of a few days, but the child continues ataxic for a time, after which recovery gradually takes place, and may be completed in a few months, or it may take two or three years before this result is reached.

Although the cases included in Batten's paper are open to the criticism that they are unsupported by any pathological data, in view of the fact that all the patients recovered, it will nevertheless be admitted that they are identical with other cases that have come to necropsy, in which evidences of encephalitis have been established.

No better example of this is to be found than in a case recorded by Clapton.

A child, aged 4 years, who had been previously healthy, was attacked by measles, after which she suddenly became unable to use her limbs or to talk. After remaining in more or less the same condition for about six months she began to improve, but even at the age of 15 her gait was still ataxic, and she could not use her hands properly. Death took place at the age of 33, when the cerebellum was found to be less than a third of the normal weight, and presented neuroglial proliferation, with

almost complete disappearance of the nerve elements of which the normal cortex is composed.

These cases must further serve to illustrate the fact that all cases in which encephalitis affects the cerebellum in childhood do not end in that perfect and complete recovery which resulted in a case published by Dr. Frederick Taylor.

A boy of 4 had fallen downstairs without seeming to be hurt eight weeks before he came under observation, and three weeks before he developed the following symptoms he suffered from whooping-cough. For three weeks his eyes had twitched, he had dropped things out of his hands, and he had been unable to walk. He was unable to stand unsupported, and was unsteady in the sitting posture. Lateral nystagmus was occasionally present. A special feature of the case was a trembling of the limbs and trunk, which resembled that seen in disseminated sclerosis. The tremors of the arms only appeared on attempts at voluntary movement, and were not violent or extensive, but quick and short. Vomiting occurred several times during the first fortnight that he was under observation, but then ceased, and repeated examination of the fundi failed to determine the existence of optic neuritis. During the next two months there was little change, but he subsequently improved, so that about eighteen months later he could walk on level ground, though in a very ataxic manner, his movements resembling exactly those of a drunken man, and not those characteristic of the gait of tabes. His arms were also ataxic. Scarcely any tremor was now noticeable in the head and neck. His speech was hesitating and his voice was not properly modulated, but he looked fairly intelligent, and he was lively and cheerful. Sensation appeared to be normal. In the course of the next two years he had improved so much that he was able to run and to bowl a hoop, although his speech was still a little imperfect, and when seen at the age of 33 so complete was his recovery that he was able to follow the calling of a clerk, and presented no sign of his former complaint, but was an active man, and included hockey among his recreations.

Most of the cases that have come to necropsy have done so at a period so remote from the original illness that sclerotic changes were alone determined, and supply the only justification for the assumption that an encephalitis had existed. A case recorded by Nauwerck, however, affords an illustration of what the acute changes may be in cases of the kind:

A girl of 19, who had been formerly healthy, except that she had previously suffered from otitis media after a transitory cold, with slight pains in the limbs, developed some headache, especially in the occipital region, fainting, and repeated vomiting. Her pupils were contracted and did not react to light, and the discs were normal. There were no cord symptoms, and her urine was free from albumen and sugar. Two days after the onset she had developed jactitations, with constrained positions of the limbs. Respiration became deep and very slow, and ultimately Cheyne-Stokes. There was groaning at times, and she experienced difficulty in swallowing. Death occurred four days after the onset of the illness.

At the autopsy the right cerebellar hemisphere was occupied by a walnut-sized apoplectic focus, consisting of black coagulum, the margins of which were irregular, and the walls formed by a greyish-red softened zone beset by small haemorrhages. The morbid changes extended across the middle line into the left cerebellar hemisphere, where there was a focus the size of a small hazel nut. On the right side the softening infiltrated the cortex over an area the size of a penny. The tympanic cavity was normal, as was the frontal sinus.

Another class of case which may be the outcome of an acute disease earlier in life is that in which there are evidences of chronic meningitis in addition to atrophy and a general sclerotic condition of the cerebellum, and in which epilepsy and mental enfeeblement may form part of the clinical picture. A case recorded by Hammarberg illustrates this:

A child who had previously been quite well fell ill with some form of severe cerebral inflammation at the age of 7. Following on the acute illness, he became ataxic, and presented continuous oscillations of the trunk and limbs, while all of his movements were slow and uncertain. Speech, which was lost for a time, was indistinct and scanning in character after its return. At the age of 24, the patient became subject to fits of mania, developed delusions, had some epileptic attacks, and died. The cerebellum was found atrophied and sclerosed, and the pia mater covering it was thickened all over. The frontal lobes of the brain and the left inferior olive were also the seats of patches of sclerosis.

VASCULAR LESIONS.

In considering the phenomena which characterize tumours of the cerebellum I took the opportunity of referring to the fact that vascular lesions are much less commonly responsible for disorders of function of the

cerebellum than of the cerebrum. It must not, however, be supposed that they do not occur in the cerebellum. Nevertheless, in the vast majority of cases they prove of pathological rather than of clinical interest, because death has resulted so rapidly or under such conditions as have not allowed of cerebellar symptoms being determined, or that the lesions have been of such a character as could not be expected to give rise to a characteristic clinical picture.

The first class of case is supplied by haemorrhage, in which the patient dies rapidly owing to rupture of the blood into the fourth ventricle, or if living longer remains comatose, and does not permit of proper analysis of the symptoms in the way that might otherwise allow of information being obtained which might lead to a correct diagnosis.

The latter class of case is usually supplied by thrombosis, in which foci of softening determined on necropsy are so small and insignificant that they could not reasonably be expected to occasion characteristic symptoms during the patient's lifetime. This is to be accounted for by the free anastomosis of the cerebellar arteries, whilst the sources from which the cerebellum receives its blood supply make the occurrence of embolism altogether unlikely in the organ. These remarks are sufficient justification for my asking you to allow me to remind you of the sources from which the cerebellum derives its arterial supply. Not only does the cerebellum derive its supply from the basilar, but it also does so from the vertebrals before they unite to form the basilar sheath; whereas the superior cerebellar artery, which supplies the lower surface of the organ, and the anterior cerebellar, which supplies the inferior surface, are derived from the basilar, the inferior cerebellar, which also supplies the under surface and the posterior part of the upper surface, comes direct from the vertebrals. It will be readily understood that the free anastomosis of the vessels precludes the possibility of large areas of softening resulting from thrombosis as a rule, and that the cerebellar vessels are so fortunately placed in regard to the main trunks from which they are derived, that there is little likelihood that an embolus would become deviated from one of the main channels of the cerebrum to be lodged in the cerebellum.

The following vascular lesions are met with:

1. Haemorrhage.
2. Thrombosis.
3. Arterial degeneration with sclerosis of the cerebellum.

Haemorrhage.

What has been said in regard to haemorrhage must not be taken to mean that extravasations of blood never take place into the organ under conditions that permit of a correct diagnosis during the life of the patient, for this would not be true. The cases are, however, very exceptional in which the uncomplicated cerebellar syndrome is determined, for the reason that it so often happens that the haemorrhage is as much pontine as cerebellar, taking place in one of these seats and extending to the other. Premonitions that have been insisted on are headache, especially occipital, and giddiness; while, according to Nothnagel, haemorrhage in the cerebellum may be suspected if there be repeated vomiting at the beginning of the attack without the occurrence of any obvious motor paralysis. In an old woman, whose case was studied by Touche, vomiting a month before death was the only symptom, and yet there was a focus of haemorrhage in the centre of the left cerebellar hemisphere.

A case that illustrates what the effects may be in a patient in whom the haemorrhage is strictly confined to the cerebellum, without either involvement of the pons or rupture into the fourth ventricle, is supplied by Boldt:

Each cerebellar hemisphere was found to contain a haemorrhage about the size of a walnut, in a man aged 42, in whom severe general spasms were followed by a dazed condition, in which state the tendon-jerks were exaggerated and continuous clonic tremors were observed in the right extremities and lower part of the face on the same side. Two days later the eyes became divergent, there was definite dysarthria, cerebellar ataxia and Romberg's sign. Aphonia, great difficulty in deglutition, and Cheyne-Stokes breathing, preceded the fatal termination of the case, which occurred four days after the onset of the illness.

Another case which supplies an example of what may happen when the haemorrhage occurs into the middle lobe is supplied by Collett:

An extensive haemorrhage occurred into the middle portion of the superior vermis in a girl aged 19, who experienced difficulty in breathing, whose eyes deviated to the left, whilst the left half of her face was in tremor, and her arms and legs in constant movement. Six days later sudden spasm occurred, with cyanosis and arrest of respiration.

Another case, which is an example of how the cerebellar syndrome may result from haemorrhage into one of the peduncles, is one recorded by Porot:

The extravasation of blood was limited to the first part of the right superior peduncle. A man of 50 was attacked by vertigo and transitory diplopia, followed by the cerebellar gait, dysarthria, hemi-asynery, ataxia and diadochocinesia of the right arm, bilateral exaggeration of the knee-jerks, preservation of the superficial reflexes, and slight nystagmus on extreme turning of the eyes to the right.

The more usual state of things, however, is that the haemorrhage in the cerebellum is complicated by haemorrhage in the pons. Inglis and Fenwick met with a case of the kind:

A woman, aged 26, had old blood clot filling a cavity on the right side of the cerebellum, while a cyst the size of a pea, containing clear fluid, was situated in the posterior extremity of the pons on the same side. In this case, as in others of the kind, there was evidence of affection of the pyramidal tracts, as supplied by the extensor type of plantar reflex, while such partial hemiplegia as existed was on the contralateral side to the cerebellar and pontine lesions, as evidenced by the findings at the necropsy. Moreover, numbness of the left side of the face was one of her symptoms. She had frequently repeated purposeless movements which shook the bed, and she lay on her right side, and, when turned on to her back, turned her eyes and head to the right in a way that is contrary to what we know usually obtains in acute lesions of the cerebellum, in which there is deviation of the head and eyes to the opposite side, as obtained in a case published by Laignel-Lavastine and Halbron, in which haemorrhage into the right cerebellar hemisphere resulted in conjugate turning of the head and eyes to the left.

On the other hand, how haemorrhage may occur into the cerebellum without causing cerebellar symptoms is illustrated by a case published by Spiller, Robertson, and Wadsworth:

A woman, aged 37, was found to have a haemorrhage into the vermis and posterior portion of the fourth ventricle, without having had vertigo, convulsions, or any other symptoms to lead to this condition of things being suspected during life.

In a case published by Camille Lian there was generalized muscular weakness without alteration of the tendon jerks and superficial reflexes, but no characteristic symptoms pointing to the cerebellum, in a woman aged 60, in whom haemorrhage in the anterior and internal portions of the quadrangular lobe of the right cerebellar hemisphere existed, and the blood infiltrated the whole of the cortex of the superior half of the hemisphere and part of the cortex of the superior vermis.

Thrombosis.

While it is true that thrombotic lesions in the cerebellum are usually so small that they are discovered on necropsy without having caused any symptoms that could lead to their being suspected during life, exceptions occur, as may be judged from what obtained in a case published by Négel and Théohari:

In a man aged 50 the disturbance of equilibrium was so great that he was unable to stand upright without support, continuous oscillation of the body resulting on his attempting to do so, and there being a tendency to fall backwards. In this case, however, in addition to the chief focus of softening in the left hemisphere of the cerebellum, involving the white substance and nucleus dentatus, there were similar foci on the inner side of the occipital lobe, two on the right side of the pons and one on the left. He, however, presented no clinical evidence that would justify the belief that the pyramidal tracts were affected in the pons or elsewhere.

M. Thomas has also published a case in which, after thrombosis of the right vertebral and postero-inferior cerebellar arteries in a man of 58, there resulted vertigo, a tendency to fall to the right, ataxia of the right arm and tremors of both legs, slight ptosis of the right eye, the pupil of which was smaller than the left, transient lateral nystagmus, and difficulty in swallowing.

Arterial Degeneration with Sclerosis.

Yet another way in which vascular disease may lead to disturbance of cerebellar function is by the occurrence of a more diffuse process, with a great deal of neuroglial proliferation which leads to sclerosis. Few cases of the kind have, however, been proved by the discovery of histological changes in the cerebellum that can be ascribed to

such vascular disease. One of the best established is a case recorded by Schultze:

The first symptoms were observed at the age of 39; they progressed rapidly, so that the patient died at the age of 43. A typical staggering cerebellar gait developed without Romberg's sign; there was slight nystagmus, intention tremor of his arms, and his articulation became slow and indistinct. Thus a clinical picture was produced which was similar to that which may result from a progressive degeneration of the cerebellum, in which vascular disease cannot be blamed for the morbid changes or the clinical picture to which they give rise. In reality, however, in this case, as in a few others of a similar kind, abundant evidence of vascular disease and its consequences were forthcoming on necropsy.

The cerebellum was found considerably atrophied, and presented patchy sclerosis. The Purkinje cells were greatly reduced in number, and those which had not disappeared were nevertheless atrophied. The molecular and granular layers were only slightly affected. The central nuclei shared in the changes, and were thus atrophic. The white matter was greatly destroyed, the lesions being both focal and diffuse, with a great deal of sclerosis due to neuroglial proliferation, and the vessels were diseased. The superior cerebellar peduncle was degenerated, and atrophic changes, probably secondary to the disease of the cerebellum, were determined in the olives, middle peduncle, and transverse fibres of the pons, whilst the spinal cord was normal.

DEGENERATIONS OF THE CEREBELLUM.

Before dealing with the degenerations which may result in the cerebellum it is necessary for me to remind you of a few points in connexion with the structure of the cortex of the organ, in addition to those anatomical details to which I directed your attention in the first lecture, for these, you will remember, only related to the separation of the cortex from the intrinsic nuclei, and to the connexions which exist between the organ and other parts of the central nervous system. No reference was made to the actual minute structure of the cortex.

Structure of the Cortex of the Cerebellum.

The cortex consists of two layers, an inner or granular, which is best considered first, in that the neurons of the small nerve cells of which it is composed, and which are spoken of as granules, send their axons vertically towards the surface to reach the outer or molecular layer, where they are found to be continuous with horizontal fibres which run between the cells of this layer. The cells of the molecular or outer layer are not so small as those of the inner layer, and they are not so numerous, so that they are more scattered. Between the outer and inner layers the cells or corpuscles of Purkinje are to be seen. These are large flask-shaped cells, whose dendrites branch in antler fashion, forming another constituent of the outer or molecular layer.

The following groups of cases of degeneration of the cerebellar system have been established:

1. Parenchymatous degeneration of the cerebellum.
2. Olivo-ponto-cerebellar atrophy.
3. Degeneration of the spino-cerebellar tracts.

Parenchymatous Degeneration of the Cerebellum.

Since Marie's publication in 1893, when he collected certain cases which presented more or less similar clinical features, from which he constructed a clinical picture that was regarded by him as characteristic of a group to which he applied the term "hereditary cerebellar ataxia" (*L'Hérédo-ataxie cérébelleuse*), many writers have followed his teaching, and it has become an accepted fact that a group of cases exists in which primary disease of the cerebellum leads to a very definite and characteristic clinical picture, which is not to be confounded with that of Friedreich's ataxia, in which the clinical phenomena are to be ascribed to changes in the afferent cerebellar tracts in the spinal cord rather than to disease of the cerebellum itself. Much as we are indebted to Marie for his paper, which called attention to the possibility that such a group of cases exists, and odd as it may seem, in only one of the cases on which his description of the affection was based has any morbid condition of the cerebellum been revealed by necropsies subsequently performed in the cases, and even in that case the morbid changes were not strictly limited to the cerebellum, in that there was optic atrophy.

True as this is, it nevertheless happens that investigations stimulated by his contention have resulted in establishing beyond question the existence of cases in which, with a clinical picture of ataxia, primary degeneration of

the cerebellar cortex has been found to account for the condition without any co-existing degeneration of the afferent cerebellar tracts in the spinal cord. No better illustration of this class of case is to be found than that published by Gordon Holmes:

A man aged 53, from a family of eight, of whom four were similarly affected, and in each of whom symptoms of the disease set in between the ages of 33 and 40 years. Two brothers and a sister were the other members of the family affected, and all of them had been in the Marylebone Infirmary under Dr. Lunn's care, where the patient died at the age of 70, and afforded Holmes the opportunity of making a histological examination of his nervous system.

At about the age of 35 he began to be unsteady in walking, and this became progressively worse, while by the time he was 40 his hands were very unsteady and uncertain in their movements, and soon after this his articulation began to be altered. He was small but well built, and, as in the case of the other members of the family affected, the tonsils were small, as were his genital organs, and beyond his eyelashes he had no hair on his face and very little on his head. He had never required to shave. Nystagmoid jerks were evoked on lateral turning of the eyes to either side. His pupils were equal, and reacted to light and accommodation. His optic discs were normal. There was some tremor of his head when unsupported, and his upper limbs also revealed tremor, in addition to ataxia and uncertainty in their movements. His gait was unsteady and reeling, like that of a drunken man. Romberg's sign was nevertheless absent. No disturbance of sensation was reported; the knee-jerks were brisk. He ultimately became bedridden, and unable to stand or walk without support. His articulation was slow, jerky, and explosive, and there was marked nystagmoid jerking of the eyes, especially on lateral movements. The facial muscles showed considerable excess of contraction when he spoke, as is so often seen in Friedreich's disease. Volitional movements of his limbs were poorly co-ordinated, though not unduly weak, the ataxia resembling that seen in Friedreich's disease. His tendon jerks were brisk, and there was no disturbance of sensation or of the special senses.

Examination of the central nervous system after death showed marked more or less uniform reduction in the size of the cerebellum, which was unduly firm in consistence. On microscopical examination the molecular layer of the cortex was found markedly reduced in breadth, and the majority of its cells had disappeared, while it had undergone some sclerosis, which was most marked in its deeper part, which borders on the intermediate zone in which the Purkinje cells are situated. These cells had entirely disappeared throughout the greater part of the cerebellum, and even where present they were much reduced in numbers. Neuroglial proliferation and sclerosis replaced the Purkinje cells that had disappeared. The cells of the granular layer had also disappeared to a variable extent, which led to marked rarefaction of this layer. Secondary to the nerve cell degeneration was a slight degree of neuroglial sclerosis. The white matter of the folia was greatly reduced in size, and revealed a considerable amount of sclerosis. The intrinsic nuclei of the cerebellum were about normal in size, and their cells were normal for a man so old as the patient was when he died, so that the well-preserved intrinsic nuclei presented a striking contrast to the degenerated cortex in sections in which both parts were visible at the same time. The superior cerebellar peduncle was intact, and did not show the slightest degree of degeneration or atrophy, a description which applied also to the middle peduncle and transverse fibres of the pons. The restiform body (inferior cerebellar peduncle) was, however, reduced to about half its size, a state of things to be accounted for, in part at any rate, by the fact that the inferior olives were markedly affected, so that the olivo-cerebellar fibres were naturally degenerated, and had disappeared from the restiform body. The affection of the olives was secondary, and had nothing to do with the production of the symptoms, so that the case may be looked on as a typical example of what may be expected from the clinical point of view in a case in which degeneration is limited to the cerebellum. No degeneration or other change was detected in the grey matter of the medulla, where all the nuclei, including those of Deiters and Bechterew, were intact. The cells of the tegmentum of the pons were also unaffected, and no evidence of sclerosis was detected anywhere in the pons or mid-brain. The red nuclei and optic thalami were normal, as were the crura cerebri and internal capsule, while the cerebral cortex was also free from degeneration, except that there was a pigmentation of cells, common to people of the patient's age, and perhaps a slight reduction in the size of Betz's giant cells of the precentral area. The optic nerves presented no sign of degeneration or atrophy. The spinal cord was absolutely intact, except for such changes as may be seen in the cord of any old person affected with arteriosclerosis, as were the dorsal and ventral roots and the dorsal root ganglia, and such peripheral nerves as were examined.

It is clear, then, that a primary and progressive degeneration of the nerve elements of the cortex of the cerebellum was responsible for the clinical picture in this case, and that although the nervous systems of the two brothers and the sister were not subjected to the exhaustive examination which was made by Holmes in this case, the

clinical features were so identical in all four of the patients that it is permissible to suppose that the pathological basis was also the same in them all. The familial nature of the disease and the existence of congenital or developmental anomalies in the family are special features to be noted, while these anomalies make it probable that the selective disease of the cerebellar cortex was primarily due to a hereditary defective vital endurance or a tendency to degeneration of these tissues, which began to fail from the middle of adult life.

That such cases of progressive degeneration may date from childhood is suggested by the cases of progressive ataxia met with in children, and is proved by the cases of a brother and sister studied by Fraser.

These patients were affected from early childhood, and became progressively worse as they grew older instead of improving, in the manner seen in so many cases of ataxia in children. They presented a staggering cerebellar gait, with inco-ordination of the upper limbs, tremor of the head, nystagmoid jerking of the eyes on lateral deviation, primary optic atrophy, strabismus that was possibly congenital and articulatory disturbance of speech, while vertigo was a further symptom in one of the cases. Examination of the cerebellum of the male patient showed that the organ was markedly reduced in size, and the cortex was atrophied to about half the normal depth, Purkinje's cells having disappeared in greater part, while the few remaining were shrunken and atrophied. The spinal cord appeared to be normal, but was not subjected to microscopical examination.

Olivo-Ponto-Cerebellar Atrophy.

To Thomas we are chiefly indebted for most of the knowledge which we possess of another type of cerebellar ataxia, which has also been investigated by Déjerine, in which the clinical picture closely resembles that to which attention has just been called, but in which, in addition to degeneration of the cerebellar cortex, there is simultaneous degeneration of the grey matter and fibres of the pons and of the inferior olives. Although there may be slight differences in the clinical pictures of the three cases on which his description of the affection is based, in the main the conditions were the same, and the principal outstanding feature was marked disturbance of equilibrium in the typical cerebellar *démarche ébrieuse*. The individual movements of the lower limbs were not as ataxic, and only one of the cases showed any affection of the arms, intention tremor being the defect noted in this case. Romberg's sign was not present. The articulation was slow and scanning in all of the cases, and two of them presented nystagmus.

The pathological basis determined to account for the clinical picture was the same in all of them, although in one it was complicated by disseminated sclerosis, which was regarded as quite accidental and independent of the other morbid processes. The essential changes consisted in atrophy of the cerebellar cortex, which was severe, though not uniform either in distribution or in degree, practically complete degeneration of the inferior olives, the nuclei of the pons and its transverse fibres, both here and where they constitute the middle peduncle of the cerebellum, while the restiform body (inferior peduncle) revealed only partial degeneration. Nowhere in the degenerated parts was anything but very slight neuroglial sclerosis determined, thus stamping the changes as due to primary degeneration of the nervous elements. The central nuclei of the cerebellum were relatively intact, and the spinal cord revealed no degeneration of the afferent cerebellar tracts nor any other changes, except that in two of the cases the crossed pyramidal tracts were slightly pale. Although, as in the other type already described, the affection does not make itself manifest until an advanced age, and presents no evidence of any hereditary tendency, it differs in further showing no familial tendency, which was so prominent a feature in the other cases.

Degeneration of the Spino-Cerebellar Tracts.

It has long been recognized that the clinical picture of cerebellar ataxia may have as its pathological basis an affection of the spino-cerebellar system instead of disease of the cerebellum itself, and the best established cases of the kind are those included under the title of Friedreich's ataxia, in which the afferent cerebellar tracts, both dorsal and ventral, are degenerated, and in which the cerebellum is intact.

Some observers have, however, suggested that the organ is affected in conjunction with the spinal cord in some of the cases, and there are those who regard this affection as but one variety of a common disease, which may affect the cerebellum or spinal cord. Raymond accordingly considers that various intermediate links can be established between Friedreich's disease and Marie's hereditary cerebellar ataxia.

As has already been explained, however, most of the cases on which Marie based his description of hereditary cerebellar ataxia, and in which defect of the cerebellum was supposed to account for the clinical picture, fall under a group of cases in which the cerebellum is in reality intact, but in which, nevertheless, the dorsal and ventral cerebellar tracts, together with Clarke's column in the spinal cord, are affected in a way that makes the cases more closely linked to Friedreich's disease than to any affection of the cerebellum. The explanation of how Marie's supposed cerebellar ataxia is in reality accounted for by affection of the spinal cord is supplied by the fact that only two of the cases on which he based his description had come to necropsy when his paper was published, and that all of those that have allowed of histological examination since have presented morbid changes in the spinal cord, while the cerebellum has been intact.

In illustration of this I cannot do better than refer you to the well-known cases of Sanger-Brown, as they were included in Marie's description in the type of disease in which the spinal cord was not supposed to be affected, whereas the cerebellum was, and in which in reality those of the cases that have come to necropsy since then and have been examined by Meyer and Barker respectively have all shown the reverse to be true.

Twenty-one cases were observed in four generations of one family, and the clinical manifestations of the malady most commonly appeared between the ages of 16 and 35. Ataxia was the outstanding feature in the symptomatology, and first affected the legs, then the arms, then the muscles of the face, eyes, head, larynx, and pharynx. A reeling cerebellar gait resulted, but Romberg's sign was absent. Attempts to keep the head or limbs in fixed positions by voluntary effort resulted in choreiform movements of these parts. Palsies and subsequent spastic contractures developed, and the tendon-jerks were exaggerated, but sensation was not affected. There were no deformities other than those produced by the spastic contractures. The pupils were defective in their reaction to light, and optic atrophy was present.

In the three cases examined histologically the cerebellum was practically normal, except, perhaps, a slight diminution in the number of Purkinje's cells. Marked changes were, however, found in the spinal cord, in which the dorsal cerebellar tracts and Clarke's columns were completely degenerated, and the ventral tracts of Gowers slightly so. There was, in addition, degeneration of the middle-root zone in the dorsal columns, and ascending degeneration of Goll's columns.

CONGENITAL DEFECTS.

Few morbid conditions of the cerebellum are of greater interest than congenital defects of the organ. These may be conveniently described as falling into one or other of three groups:

1. Cases in which the cerebellum as a whole is small, but otherwise normal in structure.
2. Cases in which the cerebellum is more or less uniformly reduced in size, and in which histological changes can be determined in connexion with its minute structure.
3. Cases in which there are all sorts of gross defects, amounting, it may be, to what is practically absence of the organ, so rudimentary may be its sole representative.

Congenitally Small Cerebellum of Normal Structure.

Cases that fall into the first group are of interest for the reason that a congenital defect of the kind has been held responsible for the clinical picture of cerebellar ataxia, such as is to be accounted for by degeneration of the cerebellum alone or in conjunction with the nuclei of the pons and the inferior olives in some cases, and by degeneration of the spino-cerebellar system of tracts in others. That persons affected in this way should reach adult life without revealing any of the clinical abnormalities by which cerebellar defects are recognized is not in the least surprising, in the light of what may similarly obtain in cases of gross defects of the organ, and in view of the provision for compensation of cerebellar inadequacy to which reference was made in our second lecture. What, however, is difficult to accept in these cases is a congenital inadequacy of the cerebellum which has existed from birth until adult life is reached, should then be held responsible for a

clinical picture in which cerebellar symptoms develop and become progressively worse until a fatal termination is reached without evidence of regressive changes in the organ being detected on histological examination. Nevertheless such is held to be the case, as is illustrated by two examples recorded by Miura:

Two brothers suffered from symptoms of cerebellar defect, which commenced at the ages of 25 and 35 respectively. The gait became reeling, articulation explosive and stuttering, the face showing a vacant and stupid expression. Sight became dim, and in one of the cases there was nystagmus. The limbs became ataxic, tremulous, and weak, while the reflexes and sensation remained intact. Only one of the cases was examined *post mortem*, and beyond the fact that the cerebellum was small (only half the normal weight), as was the brain stem and spinal cord, no abnormality in the shape of degeneration or regressive changes could be detected on microscopical examination.

A similar state of things obtained in three brothers, whose cases are described by Nonne:

The cerebellar symptoms commenced at the ages of 10, 14, and 30 respectively, became progressively worse, and were accompanied by optic atrophy. Histological examination of the central nervous system failed to determine any morbid change in any part of the nervous system, which was, however, small as a whole. The cerebellum and brain stem were less than three-quarters of the normal. In one of the cases the spinal cord was abnormally small, but not in the others.

Congenitally Small Cerebellum with Histological Changes.

That congenital abnormalities of the cerebellum may, however, occasion disturbance of function without the defects being of the gross character which obtains when nearly the whole or a part of the organ is absent, is evidenced from what I was once able to determine in a puppy from a litter in which all were similarly affected. In this class of case, although no gross macroscopic defect is evident, microscopical abnormalities can nevertheless be determined, as contrasted with the cases included in the first group, in which no such structural alterations are evident.

There was marked disturbance of co-ordination in the animal, so that attempts at walking resulted in its falling first in one direction and then in the other. There was almost constant oscillation of the head and trunk when the puppy was sitting or standing. It was killed, and its cerebellum was found to be about three-quarters of the normal, but with no gross external defect apart from the general reduction in size. On microscopical examination, however, pronounced changes were found in the cortex of the organ, where Purkinje's cells were absent in a large part of the cortex, and when present were heaped up into masses which extended into the granular layer. The molecular and granular layers were very irregular in breadth, being increased in places and diminished in others, and the appearances generally were difficult to explain, except as the result of a congenital defect.

Southard has met with a somewhat similar condition of things in an imbecile who was epileptic, which he also regarded as a congenital defect. His patient does not appear to have presented cerebellar symptoms during life.

In the absence of pathological evidence it is, of course, impossible to say whether any of the cases described by Batten as congenital ataxia were to be accounted for by such anomalies, or whether a mere delay of development of the cerebellum without structural changes is responsible for the clinical picture. The condition is characterized by marked unsteadiness of the head, trunk, and limbs, with in consequence inability to perform movements, except in an ataxic manner, and as a result inability to stand, but falling at first, while, when able to walk, the gait reveals marked ataxia. Articulatory disturbances of speech, with slowness and difficulty in swallowing, complete the clinical picture, which improves in time, so that a child who at the age of 2 is unable to walk owing to ataxia, can nevertheless do so at the age of 4, although probably not perfectly even then.

Gross Congenital Defects of the Cerebellum.

Cases that fall into the third group, in which there are gross defects of the cerebellum, are chiefly of interest as illustrating how pronounced may be the congenital defect without notable clinical symptoms, as evidenced by the fact that some defects have been discovered *post mortem* when no suspicion of the condition had been entertained during the life of the patient.

I cannot better illustrate this than by reference to a case, the notes and photographs of which Dr. Shuttleworth has very kindly placed at my disposal. He writes that, stimulated by my lectures, he has been induced to look up the records of a remarkable specimen of defect of the cerebellum which he met with at the Royal Albert Asylum twenty-six years ago, and which is described by Dr. Ferrier in his book on *The Functions of the Brain*.

The case was that of a female imbecile, aged 15, the subject of phthisis, of which disease both of her parents had died. There was nothing peculiar in her appearance, and nothing remarkable in the shape and size of her head. Her palate was very narrow and deeply grooved, but not cleft, and to this condition was ascribed an indistinctness of her speech. She had never menstruated or shown any sexual tendencies, and her generative organs, though present, were like those of a child. There was general feebleness of intellect, but her imbecility was of a slight character, and there was every reason to suppose that she would have been capable of considerable education had she lived. The special senses were unaffected. There was no paralysis, and she could walk well and steadily. Moreover, she could sew and knit, although there was some tremor of her hands and inability to thread her needle. She could not carry a vessel containing any liquid without spilling some of it, attempts to pour water from a jug were attended by a similar result, all of which showed that there was some, though very slight, inco-ordination in her upper limbs.

Cerebellar defect was unsuspected during life, but on autopsy an exceedingly rudimentary state of the organ was found. No abnormality could be detected in the cerebrum, which was well developed, but the cerebellum was of the most diminutive character. The left lobe was a mere papilla, the vermiform process a minute nodule obscurely marked with laminae; while the right lobe, which constituted the main portion, was only half a square inch in superficial area and only a quarter of an inch in thickness at its base.

The pons was indicated only by a few transverse fibres, which barely concealed the pyramidal tracts in their course from the foot of the cerebral peduncles. The corpora quadrigemina had a normal size and appearance. The olivary bodies of the medulla oblongata were only obscurely indicated. With the exception of the cerebellum and its peduncles, which were reduced to insignificant dimensions, the rest of the brain and all the cranial nerves were perfectly normal in appearance.

Other cases have, however, presented very distinct cerebellar symptoms, which have permitted of a correct diagnosis during the lifetime of the patient; but even in these they have not been of such severity as we should have been warranted in expecting had we not been prepared for the compensation of cerebellar defects with which we are so familiar. Many such are on record, but the following from more recent literature will serve in illustration:

The case is that of a child 6 years old, who presented disturbances of equilibrium and diminution of vigour of all movements, but Anton, who records the case, considered the functional disturbance but slight when compared to the defects met with on necropsy, and thus concludes that other parts of the brain perform a compensatory function, becoming anatomically hypertrophied to permit of this. In the position of the cerebellum was merely a gelatinous membrane covering the fourth ventricle, microscopical remnants of cerebellar tissue existing only at its margin. The inferior olives were absent, and there were only traces of the red nucleus and superior cerebellar peduncles. The restiform body was very rudimentary, and the direct tracts between the cerebrum and cerebellum in the peduncles were absent. There was excessive development of the funiculi gracilis and cuneatus and the gelatinous substance of the trigeminus, while the cerebral cortex was disproportionately wide and the optic thalamus comparatively large.

CEREBELLAR SYMPTOMS IN GENERAL DISEASES OF THE NERVOUS SYSTEM.

There remains for our consideration what may be described as the colouring which defects of the cerebellum may give to the symptoms of some general affection of the nervous system, and which may materially alter the usual clinical picture of the malady, even to such an extent as to lead to errors in diagnosis.

Cerebral Diplegia.

An example of how this may come about under circumstances that are unimportant is supplied by cerebral diplegia, in which the ordinary clinical picture of the affection may be materially modified by the addition of cerebellar symptoms. Two cases recorded by Freund as examples of cerebral diplegia illustrate this, for prominent among the symptoms were those which have been described by Batten as characteristic of congenital ataxia. It seems reasonable to suppose, as Batten has suggested, that the cerebellar symptoms that can be determined in such cases

are in reality due to the concomitant affection of this organ in addition to the cerebrum, and not to affection of the latter without involvement of the cerebellum. We must, however, be content to wait for pathological proof of this before we can permit ourselves to arrive at any positive conclusion on a subject of the kind, although the cases recorded by Batten—in which, from pure cerebellar ataxia to cerebral diplegia, a variety of transitional forms are traced, in which evidence of affection of the cerebellum and cerebrum can be determined in varying degree—are very suggestive.

Disseminated Sclerosis.

Disseminated sclerosis supplies another example of a general disease of the nervous system whose clinical picture may be considerably modified by the addition of cerebellar symptoms. Indeed, so pronounced may be the cerebellar symptoms that the clinical picture of the malady becomes readily confounded with the progressive ataxia that is due to degenerative disease of the cerebellum or to the spino-cerebellar system. That the opposite mistake of regarding as disseminated sclerosis what is in reality a disease of the cerebellum or of the spino-cerebellar system has been often met with, is abundantly evident from a review of the diagnoses in cases of these affections that have come to necropsy. Except for the importance of scientific accuracy, however, errors of diagnosis are not material in so far as these conditions are concerned, but it is nevertheless most important that we should make a careful study of all such cases, as well as those which present a more typical picture, and are more easy to recognize as produced by one as opposed to the other of these diseases, for it sometimes happens that disseminated sclerosis is diagnosed when cerebellar tumour exists, or vice versa. The importance of arriving at a correct diagnosis under these circumstances is too evident to need further emphasis in the light of the brilliant achievements of modern surgery, where tumours of the cerebellum are concerned.

No lengthy discussion of the differential diagnosis of these two conditions is possible in the time at our disposal, but it may be said that, as in the case of pontine tumours, which were contrasted with those which occur in the cerebellum, the out-standing feature which should guide us is evidence of affection of the pyramidal system in disseminated sclerosis, as revealed by spastic paralysis, ankle clonus, absence of abdominal reflexes, and alteration of the plantar reflex to the extensor type. It is to be admitted, however, that in its early manifestations disseminated sclerosis may affect the cerebellar system to the exclusion of the pyramidal tracts for a time, so that it is fortunate that there are other manifestations that may serve to guide us to a correct diagnosis, of which none is more important than affection of the sphincters, notably that of the bladder, which is liable to occur so early in the course of the disease, and which has no part in the uncomplicated cerebellar syndrome. Subjective and objective defects of cutaneous sensibility must be similarly regarded, while the remissions followed by relapses, so common in the course of disseminated sclerosis, do not occur in cerebellar affections, although we have seen that there are some of the morbid states that affect the organ that permit of marked improvement, even up to complete cure in some cases. Although optic atrophy has formed part of the clinical picture of some cases of cerebellar disease, this is not the rule, except when gross lesions, such as tumours, have occasioned optic neuritis which has resulted in consecutive atrophy, so that the amblyopic defects of vision, with contraction of the fields and central scotoma, with or without optic atrophy, as determined by the ophthalmoscope, must be regarded as in favour of disseminated sclerosis.

When the possibility of cerebellar tumour is under discussion, optic neuritis must be allowed to favour the probability that this, rather than disseminated sclerosis, accounts for the clinical manifestations in general, for although optic neuritis has been determined in disseminated sclerosis by competent observers, it is so uncommon that in a large experience of this disease I have not yet met with a case. Headache of the severe and paroxysmal type, met with in cerebral tumours, is not likely to occur in disseminated sclerosis,

and the same may be said of vomiting, but it is otherwise when we come to consider vertigo, for this symptom may be most pronounced and distressing.

In conclusion I must ask you to allow me to offer an apology to Dr. Charlton Bastian for not including the results of his observations among those which I quoted in support of Dr. Hughlings Jackson's view in regard to the cerebellum, for Dr. Bastian supplied the first proof that muscular tonus is derived from the cerebellum. (Bastian: *Med. Chir. Trans.*, 1890, pp. 207-217; *Paralysis, Cerebral, Bulbar and Spinal*, 1886, pp. 222-228.)

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Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

SERUM DIAGNOSIS OF SYPHILIS: FLEMING'S METHOD.

IN a paper published in the *BRITISH MEDICAL JOURNAL* of August 14th, 1909, on the diagnosis of syphilis by some laboratory methods, particularly relating to the complemental fixation method by Wassermann, I brought forward the results of some preliminary trials of the easier methods of Hecht's modification, elaborated by Dr. Fleming—namely, the principle of using both haemolysin and complement for sheep's red blood cells normally present in human blood.

In this first series the results were not very satisfactory, a rather large proportion of bloods normally failed to haemolyse the sheep cells completely, thus reducing the number of positive results that should have been obtained, but at the time I distinctly stated that the numbers were too small to dogmatize upon.

The method, however, appeared to be very valuable, and called for further work; I have therefore made weekly examinations from over 500 cases, controlled by the more elaborate original method, obtaining far more favourable results. This series gave indefinite reactions in only 10 per cent. A positive result was always reliable—in fact, it was obtained in some cases earlier in the disease than with the other methods tried, as shown by several observations taken at different dates from the same case. Consequently I look upon the results given by Dr. Fleming's method as a most valuable check, and if the reaction is definite consider that it should be accepted as final.

The test was almost always applied about six hours after the blood was taken from the patient, so that no deterioration of the complement would have occurred, for it was found that the greater the delay between the time of taking the blood and the test the larger the percentage of indefinite reactions.

I feel that it is due to Dr. Fleming to make this further statement, as some adverse criticisms of his method have been based on my original reference.

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A CASE OF FETAL PURPURA.

IN August last I was summoned by a midwife to see a newly-born child of unusual appearance. The mother had always enjoyed good health, and beyond weakness and sleeplessness, and the fact that she was a "shocking, terrible size," she had not suffered from any disease during this pregnancy. She had experienced, however, more discomfort since the date of quickening than with her previous six children. Early in its course, one of her children died from tuberculous broncho-pneumonia. She had a fall six weeks before labour set in, and did not notice fetal movements during the last fortnight. The labour was associated with excess of liquor amnii (about 2 gals.), and there was somewhat excessive haemorrhage during the separation of the placenta. The child was born naturally, and lived for twelve hours. Apart from the appearances to be described, it was a well-formed male child weighing 8 lb. and measuring 23 in. in length. The cord measured 30 in., and showed numerous ecchymoses along its course. The placenta was unusually large. The child's appearance was remarkable. Its skin was dusky, mottled, and covered by ecchymotic areas varying in size from petechial spots to large rounded areas. To the touch they imparted a lumpy feeling, and had a pale raised centre and dark periphery. The colour did not alter on pressure. There were also ecchymoses on the buccal mucosa. On *post-mortem* examination it was found that most of the morbid appearances were confined to the skin. The purpuric areas cut like hard nodes, and showed pale injected surfaces. There were no haemorrhages into the brain substance. Some excess of fluid was present in the right pleural cavity, and petechiae were visible under the pericardium and peritoneum; there were small haemorrhages under the capsule of the right kidney extending into its cortex. The left kidney, spleen, and suprarenal capsules appeared normal. The bladder contained clear urine. Nothing could be found to explain the cause of the condition described except perhaps a history of tubercle in the family of the father. He himself, though stated to be a sufferer from epistaxis and winter cough, was a big healthy-looking man, but his great-grandmother, grandmother, three of his sisters, and one brother had died of phthisis. One of his children had phthisis and one had died of tuberculous broncho-pneumonia. On the mother's side there was no history of tubercle.

Datchet.

NORMAN GLEGG, M.D. Edin.

A CASE OF DIABETIC COMA AT THE AGE OF TEN.

ON December 27th, 1909, I was called to see A. T., a boy a few months over 10 years of age. I found him very restless and complaining of severe epigastric pain; he was also inclined to be drowsy at times, and his mother said he was very thirsty. The boy was very emaciated, and further inquiry elicited the fact that he had been thirsty for some months, and had been passing large quantities of urine. An examination of the urine showed a specific gravity of 1030, a large quantity of sugar, and the presence of acetone was revealed by perchloride of iron. Rectal injections of normal salt solution, and large and frequent doses of sodium bicarbonate in milk were ordered, but the drowsiness rapidly became more pronounced; coma developed on December 29th, and death took place on the following day. Curious to relate, although some neighbours afterwards told me that they had noticed the boy's gradual emaciation, the mother did not seem to have observed it, nor did she set much store on his continued thirst.

Diabetes at this early age is comparatively rare. Amongst 1,360 private patients who consulted Dr. Pavy, diabetes began under 10 years in 8, between 10 and 20 in 57, between 20 and 30 in 97, between 30 and 40 in 224, between 40 and 50 in 339, between 50 and 60 in 428. Even between 60 and 70 the number was 182, very large considering the fewer number of persons living over 60 compared with those between 20 and 40. There were 35 patients in whom the disease began at ages over 70, and 1 over 80; the youngest patient Dr. Pavy had under his care was an infant 12 months old.¹ The proportion of males to females affected with diabetes was 2.5 to 1.

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¹ *BRITISH MEDICAL JOURNAL*, December 5th, 1885.