The lesion may be vascular, such as an aneurysm or a thrombosis, it may be a tubercle or possible luetic or may be one of those rare conditions that occur rarely with migraine, a migrainous ophthalmoplegia, and whose pathology is unknown.

The most probable diagnosis is aneurysmal dilatation of one of the posteromesial branches of the posterior cerebral artery. Such lesion best explains the symptoms and their conduct. Were the lesion in the peduncle itself the syndrome of Weber would be present, *i. e.*, alternating superior hemiplegia: complete paralysis of one side of the body, including the face and paralysis of the third nerve of the opposite side. We may be right in assuming that the weakness of our patient's left side is the expression of a slight encroachment on the peduncle by a lesion such as aneurysm, tubercle, or gumma even, and that her symptoms constitute therefore the Weber syndrome not fully developed. In view of the fact that the Wassermann reactions are negative even after the administration of what has been called a provocative dose of salvarsan (0.4 gm.) and that there are no somatic evidences of syphilis that diagnosis may be excluded. A local tuberculous deposit cannot, however, be so safely excluded. The existence of 30 lymphocytes to the cubic millimeter in the cerebrospinal fluid suggest tubercle and the patient's history of exposure to the infection must be kept in mind.

There are many reasons why the case was not believed to be an example of ophthalmoplegic migraine. In the first place, it is improbable there are any cases in the literature since Charcot first called attention to the remarkable occurrence, *viz.*, oculo-motor palsy occurring in the course of an attack of migraine, and recurring, in which the paralytic accompaniments of the attack came on when the patient was as old as this one is. In the second place her present symptoms were not ushered in by phenomena of migraine, and in the third place the symptoms other than the third nerve symptoms, namely, the reeling, the hemiparesis, and the hemianopsia are not features of ophthalmoplegic migraine.

CONGENITAL CEREBELLAR ATAXIA

By Henry K. Marks, M.D.

The patient, from the First Division, is 5½ years old. His father is healthy. His mother for the past few months has been complaining of headache and constant dull pain in the right shoulder and arm. Her serum Wassermann has been reported positive and she is under treatment in the hospital. There is no history of hereditary or familial diseases. No consanguinity exists between the parents. A maternal aunt has paralysis agitans.

There are three other children besides the patient, all older than he. They were full-term babies and are healthy and thriving. The eldest is now 18. The mother had one miscarriage after the birth of the patient. The patient himself was a full-term 7½ pound baby. Labor was prolonged and difficult. After thirteen ineffectual hours it was discovered that he was a transverseling. Instruments were resorted to and the child was born asphyxiated. For two hours he gave no sign of breathing. Soon after he began to breathe, he was fed by spoon and swallowed naturally without regurgitation. From the time of birth till two weeks later

the child made no sound but lay as if dead. Then he began to cry, crying day and night. His cry was that of a normal child. From now on he took the breast for the first time.

To be noted in his subsequent history are the following: His first tooth appeared at the age of five months, the second at seven months. When about a year old he began to take things in his hands but his movements were weak and awkward. The mother is certain that there was at no time any disturbance of sight or hearing. The child appeared to understand but was not as smart as her other children.

What gave the parents chief concern and what now brings the child to the hospital is a so-called paralysis. Not until he was two years old was he able to sit up, and then so unsteadily that the fearful mother preferred to keep him flat. He would suddenly jerk from side to side and fall, commonly backward. At the age of two and a half years he started to creep. He began to stand when he was three and after a year or so attempted gradually to walk, but very unsteadily. He first attempted to talk at the age of three. His speech, however, was very defective. He has a vocabulary of four or five words—mamma, papa, cake, stairs; but the sounds only remotely suggest the originals. From birth he has been obstinately constipated, and his bowels have never moved spontaneously, most of the time fecal discharge has been hard, scyballous masses, the size of small marbles. For a year past he announces when he has desire to urinate. Since birth he has drooled but now he is gradually ceasing to do so.

The child has had no infectious diseases and a slow, gradual improvement is being noted in the disturbances enumerated.

On physical examination a child of average size for his years is found. His face is thick and heavy, his general nutrition only fair. His tonsils are hypertrophied, the submental glands palpable; diastasis of the recti exists. General examination reveals no abnormality of his internal organs nor any definite signs of congenital lues.

His right pupil is a trifle larger than the left and neither is quite circular. The reactions to light and accommodation are, however, prompt. His fundi are normal, the cranial nerves are intact.

His muscles are small but their strength is quite proportionate to their development. Gross range of movement at all the joints is normal. There is no true hypertonia, though variable voluntary spasm occurs on attempts at passive motion. As to his reflexes, his arm reflexes are not definitely obtained. No Hoffman sign exists. The epigastrics and abdominals are active and equal. His knee jerks are feeble, his ankle jerks not obtainable. Clonus and Babinski absent.

What rules the picture is a general disturbance of equilibrium, a disturbance in the exact performance of voluntary movement of all sorts. This refers not only to his body and extremities but to his eye movements and speech as well. There is no paralysis of the external eye muscles, but on forced deviation there is seen the impossibility of sustaining a given position. True nystagmus is absent but a constant bulbar unrest occurs. His speech is indistinct, scarcely intelligible. At most he makes simply approximate sounds. A general choreiform unrest is evident—a difficulty in maintaining static attitudes.

Examined more closely, a general awkwardness and insecurity are seen in his arm movements. There is no dysmetria, no asynergia in a

strict sense. Prehension is very fair. Finer movements of the hands, however, such as holding a pencil to draw or write, are grossly affected. Signs of peripheral ataxia are absent. *Adiadochokinesis*, however, is extreme and about equally distributed, right and left. The outstretched hands display a moderate hyperextension at the metacarpo-phalangeal joints.

In the legs with the child in dorsal decubitus this same general awkwardness of movement is evident. Well-defined dysmetria exists. In the heel-knee test the heel is thrown high up on to the thigh, then gradually slid on to the patella. Occasionally his legs perform wide, coarse, irregular movements as the knee is being attained or after. There is no *asynnergia*, no peripheral ataxia in the conventional sense, no cerebellar *cataplexy*. Aside from a variable amount of choreiform unrest he sits as a rule fairly quietly. Occasionally, however, his body may suddenly swerve laterally and even cause him to fall. He stands on a moderately broad base. The choreiform unrest of his trunk and arms becomes now in general more marked than when sitting. Occasionally he will suddenly sway laterally and he seeks then to improve his equilibrium by broadening his base. *Rombergism* is absent. His gait is typically titubating with a marked propulsive tendency. At times this latter becomes so pronounced that he appears literally to plunge forward rather than to walk. He holds his trunk habitually over-flexed on the pelvis. In bending backward no compensatory flexion of the knees or rise on the toes occurs, but this must be accepted with reservation.

His superficial and deep sensibility as far as can be determined and his special senses are intact.

There is then a history of marked disturbance of equilibrium dating from birth, the history of a child who did not for some reason or other learn the coördination of movement, the function of sitting, standing, walking, etc., at an age when in healthy children it should normally occur, the history of a child in whom these functions were delayed but are being slowly but gradually acquired.

The child presents the picture of a cerebellar syndrome practically pure. As to the involvement of the cerebellum there can be no doubt. We have in other words to do with a case of congenital cerebellar ataxia. This point is easily settled. The mode of onset, the course of the disease, its tendency toward gradual improvement are classical and immediately rule out that large obscure group of cases under which *Friedreich's disease*, the hereditary cerebellar ataxia of *Marie* and *olivo-ponto-cerebellar atrophy* are included. The presence of a considerable mental retardation, even omitting the fact that the child has never had systematic instruction—his mental age by the way is only 2.8—does not militate against this conception. Of importance almost equal to the mode of onset and course of the disease are certain negative data—the fact that there are no signs of pyramidal involvement, that the special senses, cranial nerves and general sensibility are essentially intact.

Granting then that this is a case of congenital cerebellar ataxia, it is left to decide what is the etiological factor, what is the essential pathology. Is it to be sought in a defective development of the cerebellum and *agenesis*, *aplasia*, *hypoplasia* or may it perhaps be found in a secondary condition, an inflammatory process occurring in utero or some vascular accident occurring either intrauterine or during the process of birth? In other words, have we a cerebellar infantile palsy comparable to *cerebro-infantile palsy* due to *encephalitis* or *hemorrhage*?

Granting syphilis in the mother at time of conception, granting congenital syphilis in the child—unfortunately neither examination of his blood or spinal fluid has been done as yet—we have a favoring etiological factor for any one of these three conditions—agenesis, vascular accident or cerebellar encephalopathy. The prolonged difficult labor, the transverse position of the child, the use of instruments might well have produced hemorrhage with secondary porencephaly. Even the report of positive syphilitic findings in the spinal fluid cannot allow us to differentiate with absolute certainty. Further, whether congenital cerebellar ataxia in the strict sense of agenesis or cerebellar infantile palsy, the course of the diseases need show no particular differences.

POLYNEURITIS OR POLIOMYELITIS

By J. L. Joughin, M.D.

This patient, from the First Division, is a bright young Jewish girl, aged 12. When very young she had measles and whooping cough. She had mumps three years ago and either two or three years ago suffered from an attack of grippe. She has never had sore throat, her health for the past few years has been excellent, and she has regularly attended school, during that period never having absented herself on account of illness.

On Wednesday, eleven weeks ago, she complained of severe pain in the calf of the right leg which occurred only on walking. This was the only symptom characterizing the onset. On Thursday the intensity of this pain diminished, but she noticed a rapid decrease of the motor power of both legs. In spite of this, however, she walked eight blocks to school and ascended four flights of stairs to gain her class room. Near the top of the fourth flight her legs gave way entirely, she fell and was unable to regain her feet without assistance. At this time she noticed that she could not get up from her chair without placing her hands on the arms of the chair and thus partially supporting the body weight while in the act of arising.

Friday she went to school in the morning but in the afternoon became unable to walk and since that time she has never been able to resume attendance at school. By Saturday all pain had left her. On Sunday the motor loss increased to complete disability when she entered the Jewish Hospital, Brooklyn, and remained there for six weeks. She says at this time she could not stand alone even momentarily, nor walk, and any movement of the toes or of the foot as a whole at the ankle joint was quite impossible. The mother of the child confirms these statements. There were not at that time nor had there ever been any paresthesias nor does she recollect that she ever felt pain on deep pressure of the muscles of the calves or thighs.

A day or two after entering the hospital she began to fumble in picking up objects and this difficulty became very marked, though it never increased to complete impotence. A definite loss of power developed in the upper extremities, the fingers became partially flexed and it was only after passing approximately two weeks in the hospital that she became able to extend them. Apparently there have been no subjective sensory