these retarded pupils suitable training and education in special school classes.

This is a flank movement for the benefit of the feeble-minded of the state. In a few years all such people will be recognized, for all children come under the control of school authorities between the ages of 6 and 14. This will eventually mean that every feeble-minded child may receive proper education, habit training and industrial training during the formative period of youth. The idiots and imbeciles not cared for at home, and the highly potential hereditary defectives with sex tendencies, and those with criminal tendencies, will gradually be drafted into the institutions. We know now that some mental defectives are inherently bad and vicious, and that others are tractable and may be made industrious and harmless. We no longer believe that all mentally defective persons need to be permanently confined in an institution.

The army tests revealed thousands of technically mentally defective drafted men whose mental defect had not prevented self-support and practically blameless lives. Much of the rough and disagreeable manual labor of the mines and fields and forests is performed by persons who could not pass the modern intelligence tests. The lack of 100% efficiency alone does not call for institutional care, or for the taking away of the "right to liberty and the pursuit of happiness."

Original Articles.

A CASE OF MULTIPLE SCLEROSIS*.

BY FLORENCE L. MEREDITH, M.D., NEW YORK.

THE following case has been under observation, although not directly under the writer's care, for nine years. The main portion of this history was written four years after the onset, and the appendix today:

Male, 43 years old, lawyer. Family history: Father died at the age of 60 of chronic nephritis. Mother living and well, now 75. One brother living and well, although he had neurasthenia five years ago. Patient is married and has two children, one perfectly normal, the other of highly nervous temperament, having been obliged to miss a whole year out of school at the age of 16 on this account. There

is no history in the family of any organic nervous diseases, tuberculosis, syphilis, or cancer. Neither were there any mental troubles. Patient's mother is said to be almost pathologically awkward in gait and all motions.

Past History: Patient had most of the children's diseases, but was otherwise considered well. Never had convulsions. Was of athletic type in school days. Had a peculiar habit of lying down flat on his back on the floor instead of sitting in a chair, whenever opportunity offered, all through his youth. The first neurological symptom appeared at the age of 19, when he had a unilateral strabismus of sudden onset, lasting a few months and disappearing as quickly as it came. He was always a heavy smoker, consuming sometimes a dozen strong cigars a day. Very moderate use of alcohol, tea, and coffee. At the age of 20, had a severe attack of malaria. No history of venereal disease or even exposure. At the age of 33 had serious business trouble, causing much worry and anxiety for several years after. At the age of 35 had a fall backward on the ice. Hit on the back of his head and was unconscious for about an hour. No results were observed and recovery was apparently complete in a few days. The following winter he had an exactly similar fall with unconsciousness for a shorter period, and prompt recovery.

Present Illness: Four years ago patient first noticed that his legs were getting weak, especially the left. No pain. Family also noticed that his speech was slow and exact. This gave the impression of increased dignity, which, however, was in contrast to sudden bursts of almost uncontrollable laughter at slight provocation. At the same time he began to walk unsteadily. None of these symptoms were sufficiently marked to be noticed by anyone except his family. He frequently said jokingly that he was a sick man, but no one took him seriously at that time.

About six months later he had a tooth drawn under nitrous oxide anesthesia, and was peculiarly affected by it. He became cyanotic and remained so for some time after recovering consciousness, and was so weak muscularly that he was unable to walk home, nor was he able to walk alone for a week or more, and then only with a cane. He has never gone out without a cane since. From this time on, he became progressively worse for several months. Frequently fell down, always either forward or back, but never hurt himself. A labyrinth test revealed nothing pathological. Strabismus reappeared, this time of the left eye only. He was then taken to a neurologist, who made the diagnosis of multiple sclerosis and offered him no encouragement. At the advice of friends he took up massage treatment, and after two weeks was so greatly improved that he was able to run for a car on one occasion. The masseur was then obliged to go away for two weeks, and

* The first part of this paper was read at a meeting of an association of women doctors in Boston (Hotel Victoria), in 1915.

the patient went promptly back to his previous condition. Subsequent massage had no effect. Added symptoms also appeared in the arms, which were practically unaffected for a year after the onset. The arms grew very weak and a tremor appeared whenever he tried to use them, so that he was unable to write without tearing the paper, or to feed himself without spilling the food. Severe constipation for a time and some retention of urine were added later to his symptoms.

No real pain at any time throughout the disease, although he complained for a time of a girdle sensation around the lower thorax, and also around the head. He had a period of several weeks when taste was almost lost, and later a period of several months of partial amblyopia, when reading was impossible. At another time, for several weeks, he complained of hemianopsia. In spite of the strabismus he never had diplopia. Twice during the past four years he has been confined to the bed for periods of two and three months respectively. Each time it seemed to be because of great muscular weakness and a feeling of exhaustion. On at least four occasions he has had attacks resembling apoplexy, which passed off quickly, leaving no paralysis and the patient in the same condition as before. These all came on during the day, after some special exertion. Has not smoked for about a year. Closed his law office at the beginning of the disease, although he continued to see clients at home for a few months. Has made no attempt to conduct any business for three years, on account of some mental enfeeblement of which he is conscious, but which is not apparent to his family. They declare him to be in his usual mental condition except that he is a little overexcitable, laughs and cries easily, and occasionally repeats himself, as an old man will do. Has always slept well, has good appetite, and does not, apparently, worry about himself. He is possibly a little too care-free. Spends his time reading in an unsystematic way.

Physical Examination: Well developed and nourished man. Trunk better nourished than limbs. Sits all day in a large chair with feet propped up. Is moved there, with very little coöperation on his own part, by his wife and son. Makes no attempt to rise in greeting. Extends his hand, which trembles violently, and says, "How-do-you DO," with a pause between each word and extra emphasis on the last. His voice is not modulated, but is all on one tone. This is his invariable method of speaking. The enunciation is uncommonly distinct, possibly because of special effort. In answering questions he delays some seconds before commencing a reply. Facial expression lacking in mobility. Absence of lines in the face, even while talking. Lips rather full and relaxed. Both these characteristics are newly acquired.

Examination of Cranial Nerves: Pupils small, equal and react to light and distance normally. Palpebral fissures equal and normal. Marked internal concomitant strabismus. All movements of the external ocular muscles can be made, including those of the external rectus, but are made weakly and cannot be sustained. There is slight lateral nystagmus, not present constantly, but only on extreme lateral deviation of the eyeball, not on vertical deviation. No diplopia or hemianopsia can be determined. Vision about normal with glasses, slightly myopic without. Visual fields concentrically slightly narrowed. Slight loss of discrimination of colors. Ophthalmoscopic examination showed optic disc pale, outline slightly cloudy, vessels not tortuous. All movements of the facial muscles are possible, but weak, some tremor induced. Sensation normal on right side of face and head, but diminished to touch, pain, and temperature on the left. Tongue protruded fairly steadily in the median line, not far beyond the teeth. Taste normal to sweet and sour substances. Palate moves normally. Hearing normal, but there seems to be delayed perception of sound. No hyperacusis or tinnitus.

Head is held steadily without tremor on the trunk. Can be turned in all directions with normal strength, but is almost invariably held in the median line. No stiffness of the neck. Arms are rather thin, although muscles feel quite firm. No spasm on passive motion. All movements made weakly, however, and accompanied by a violent tremor involving the whole arm from the shoulder. Oscillations are at the rate of five to eight per second. The usual tests for ataxia are both impossible and unnecessary as the ataxia is very apparent. Marked adiodocinesis. Triceps, biceps, and radial reflexes about normal in right and slightly decreased in the left arm. No Jacobson. In both hands the interossei are greatly wasted. Grasp is fairly strong, but intermittent. No tremor when the arms are at rest, but when motion is attempted the tremor increases with the extent of the motion.

In the lower limbs the thighs are quite wasted, calf muscles about normal. Measurements equal on both sides. Double Babinski. Knee jerks and ankle jerks both greatly increased. Double ankle and patellar clonus. Spasm in both knees on passive flexion and extension. Marked ataxia in heel to knee test, tremor similar to that described in the arms on attempted motion, none at rest. There is some loss of strength. Sensation normal on right arm and side of face, diminished on left. Somewhat reduced over entire left side of body. Temperature sense also diminished. No vasomotor or trophic changes, skin and keratin tissues normal. Cutaneous reflexes not tested. Electrical reactions not tested. No

contractures anywhere. Some atrophy of the interosseal.

Station is very unsteady, apparently due to both weakness of the muscles and to incoördination, as well as to vertigo, from which the patient suffers only when attempting motion. Cannot stand without assistance. Sways forward and back, never from side to side. Not more pronounced with eyes closed. Gait shows muscular weakness, active spasm, ataxia, and cerebellar staggering. A general tremor of the whole body is produced. Back held rigid, whole body bent forward from the hips. Toes drag, although passively foot can be brought easily beyond a right angle with leg. Physical examination otherwise negative. No stigmata of degeneration. Urinalysis negative for albumin, sugar, and lead. Wassermann on both blood and spinal fluid negative. No excess of lymphocytes or globulin in spinal fluid.

Differential Diagnosis: First should be ruled out *functional disorders*. Many features of *hysteria* are present. In the motor system we have paralysis and paresis, spasm tremors and the history of seizures of various kinds. In the sensory system we have hemianesthesia, contracted visual field, transitory loss of vision and taste. In the psychic sphere we have lack of will power, excitable disposition, easy laughter, and tears. We lack the customary fondness for sympathy often observed in hysterical cases. But there is no need of considering hysteria as the sole trouble present when we have such definite signs of organic disease as are supplied by Babinski's sign, increased tendon reflexes and the slight though definite optic atrophy. These three things of course indicate organic trouble, but it is possible that there is some element of hysteria in this case, too.

Neurasthenia can be ruled out on the same grounds, although that diagnosis was held by his family physician for some time, who considered the tremor, the weakness, the increased reflexes, the mental asthenia, the sensory disturbances, and the easily exhausted vision only exaggerated symptoms of the neurasthenia prevalent in the family. The clonus he considered spurious. The Babinski had not appeared when he made his diagnosis, and he never had an ophthalmoscopic examination done. Our patient had never had the insomnia, the phobias, the headaches which accompanied neurasthenia in the other members of the family. He may indeed be afflicted with neurasthenia, but it is certainly not all of his trouble, as he and his family believe still.

In many ways this case resembles another so-called neurosis, paralysis agitans. There is muscular rigidity, tremor, increased reflexes, face expressionless and mask-like, speech monotonous and deliberate, face held in the median line and mental enfeeblement. But here the resemblances cease. In this patient there

is no tremor while at rest; in paralysis agitans it would be constant and would be reduced by muscular effort. In this case the tremor is extensive, involving the whole arm; in paralysis agitans it is more marked in the distal portions of the limbs. The latter disease seldom begins as early in life as in this case, and for some reason almost always shows the peculiar bent forward position in walking and also in sitting, which is not a prominent feature here. Ankle clonus is not present in paralysis agitans, but it often parallels our case in producing an uncommon amount of disability in proportion to the physical findings, together with a somewhat similar mental condition to that of our patient.

Considering the tremor as the prominent symptom we should also have to rule out *essential* tremor, a disease which produces an identical tremor to the one described. But it is a family disease, and does not carry with it such a train of other symptoms, since it is probably a neurosis without lesions. Alcoholic tremor, which also occurs only on motion, does not need consideration here. Nicotine poisoning would certainly have to be considered in finding a cause for this case of multiple sclerosis, but could not be considered as an entity.

Having sufficient signs of organic lesions, and being obliged to abandon functional conditions, we are led definitely to multiple sclerosis and possible associated functional disturbance, by the irregularly distributed paralysis, with some reflexes increased, others diminished, with Babinski, intention tremor of which the nystagmus is a part, scanning speech and partial optic atrophy. It is easy to throw a collection of symptoms together and call it multiple sclerosis, but before doing so a number of other possibilities in the way of organic disease should be ruled out.

In the first place, disease confined solely to the *peripheral nerves* must be excluded. The quadraplegic distribution suggests *multiple neuritis*, also the intention tremor and the optic nerve trouble. But in multiple neuritis the reflexes are diminished and the limbs are flaccid, and are particularly weak in extension. There is no true clonus, and there are prominent sensory symptoms. Atrophy comes on and vasomotor changes, but not any abnormal reflexes. Contractures are quite likely. Henschen has reported a case of diphtheritic polyneuritis which went on to become multiple sclerosis, as shown by autopsy.

Leaving the peripheral nerves, we come to *cord diseases*. *Chronic poliomyelitis* can be ruled out on the grounds just given, that is, it would give a paralysis of the lower segment type. The wasted interosseal and the flaccid arms in our case suggest that there must be involvement of the anterior horn cells of the cervical region, but there is no evidence of such involvement elsewhere.

Degeneration of the posterior columns. The

patient told me quite frankly that he supposed he had locomotor ataxia, but I had to rule it out because of the increased reflexes and spasticity, and the absence of lightning pains and other tabetic symptoms, as well as the history. Any ataxic condition the public is prone to label syphilis, regardless of the unhappy effect on the patient,—not only the public in this case, but the man's oldest neighbor, a physician.

Primary lateral sclerosis. Such a condition may be possible uncomplicated. While it would account for the condition in the legs, as far as spasm and increased reflexes are concerned, it would not satisfactorily account for the condition in the arms, nor for the sensory disturbance, the eye symptoms, and the general weakness.

Erb's spinal paralysis, of toxic origin,—usually syphilis of the lateral columns as tabes is of the posterior columns. This accounts nicely for the picture in the legs of this patient, but leaves unaccounted for the flaccid arms, the tremor, the speech defect, and the sixth nerve involvement.

Secondary spastic paralysis, following chronic myelitis or thrombosis of spinal vessels, caries of the vertebrae, tumor pressure, fracture or meningo-myelitis. Any of these causes would also produce a spastic paralysis below the level of the lesion. Some of them also produce a flaccid paralysis by involvement of the anterior horn cells at the site of the lesion. In this way we could account for the condition of both the arms and legs in this patient, by supposing the lesion to be at the level of the arm area, but we should still have to seek further for an explanation of the condition in the head, and of the tremor. The possibility of trauma at the time of the fall on the ice is somewhat suggestive, but there were no symptoms other than brief unconsciousness at that time. There was no pain to have been suggestive of myelitis, and what few sensory disturbances he has had were slight and have failed to persist. Furthermore, he had had strabismus before the fall.

Combined System Diseases. If a lesion in no one tract will account for the symptoms in this case, perhaps we can account for them by a lesion in more than one tract. There are two types of combined system diseases to consider. The first is *postero-lateral sclerosis or ataxic paraplegia*. This condition, in some cases, is difficult to differentiate from multiple sclerosis, as it may clinically almost correspond to it. Had my case not presented the nystagmus, the scanning speech, optic atrophy, and intention tremor, in other words, had it been confined to the cord, the remaining condition could not have been differentiated from ataxic paraplegia. In reality it is a limited ataxic paraplegia plus other lesions. The dorsal and lateral tracts are also involved in another disease which presents a somewhat different picture

clinically,—*Friedreich's ataxia*. In order to exclude this hereditary or family disease, I examined the patient's mother, who was said to be awkward. I found her quite ataxic in all motions of arms and legs, speech incoördinate and slovenly within normal limits, attended by slight grimacing. She is an old woman, but goes out every day alone, although she frequently falls down. Also falls down stairs in the house occasionally. In all other respects, normal. She and her son have neither form of hereditary ataxia as it is commonly known, probably the condition in each of them being founded on neuropathic tendency. Friedreich's ataxia shows flaccidity and absent reflexes, rarely has eye symptoms, has an atypical tremor and speech defect, while the hereditary cerebellar form lacks spasticity until late in the disease. Furthermore, no other case resembling ataxia has ever appeared in the family on either side, for several generations certainly, or as far back as their knowledge. Strange to say, our patient's uncle has an invalid wife in much the same condition as this patient. She was not where she could be examined by me, and, so far as any of the family know, no diagnosis has ever been made.

One form of *secondary posterolateral sclerosis* is related to anemia. It is possible that some of these forms which have been considered primary may be in reality secondary. Some cases certainly are definitely secondary. Interstitial changes arise and are later followed by sclerotic foci. They may also arise in pellagra, ergotism, and chronic wasting diseases. They are accompanied, as would be supposed, by neuralgias and sensory disturbances. Unless the cause is suspected and measures taken toward cure, the primary condition usually results in death after a shorter period than our patient has survived. But with anemia in mind, we had a blood count and found it normal. Riggs reports several cases in which anemia and multiple sclerosis were combined, and he thinks both came from the same toxic origin, and believes the nerve symptoms may precede the blood picture by some time.

The other type of combined system disease is *amyotrophic lateral sclerosis*, involving the anterior horns and the lateral columns. It is worth considering in this case, because we have definite signs of pyramidal tract involvement in the legs and of anterior horn involvement in the arms. If the anterior horn cells were involved before the lateral tract we should still have flaccidity, even after lateral tract involvement, because there would be no way by which spasticity could be produced after the anterior horns were gone. But in this case we have definite pyramidal tract symptoms. The sequence in antero lateral sclerosis is finally a complete anterior sclerosis, including the nuclei of the cerebrum, which are analogous to anterior horn cells of the cord. When this takes

place any spasticity which had been present would of necessity disappear, leaving atrophy and flaccidity throughout. After more than four years there is no indication that this is taking place in this patient. There is still practically no atrophy. For this reason, and on account of the less complete loss of power, the slight but definite sensory involvement, the nature of the tremor, which is not fibrillary, and the non-appearance of bulbar symptoms, we can rule out amyotrophic lateral sclerosis.

Syringomyelia was easily ruled out on account of the absence of the peculiar sensory features and of complete atrophy with vasomotor changes in any one set of muscles. If our anterior horn cells are involved at all they are only lightly attacked, perhaps with minute microscopic plaques.

Cord and brain symptoms in the same patient always suggest *cerebrospinal syphilis*. The absence of positive laboratory findings and also the absence of the quite usual headache and third nerve involvement is against it. In the cord it has already been ruled out in the form of disease of the posterior or the lateral tracts. It can be ruled out as meningitis or meningomyelitis, because there is not and has not been enough pain, especially pain of the back and limbs. Other forms of meningitis can be ruled out in the same way.

Concussion of cord and brain is suggested by the history of falls. It is quite possible that hemorrhages which have since been organized might have been the cause of this trouble, but it seems as if there would have been some symptoms between the time of the last fall and the onset of the present illness. My theory is that the falls were the result rather than the cause of the disease, the actual onset of the unsteadiness being much earlier than the patient realizes.

That the conditions might be a *diffuse encephalomyelitis* would certainly have to be considered. This is probably the most difficult condition to rule out. There is very little, however, in the history to suggest a general infective process, except the fact that there are multiple lesions of some sort to be accounted for. Had the attack of malaria appeared before instead of after the initial strabismus, it would have been easy to attribute the whole condition to malaria. If it is infective at all, it is certainly a low grade type, such as is seen in chronic arthritis. Starr says that a diffuse encephalitis is often the starting point for the so-called cases of multiple sclerosis. Some authorities consider the two conditions identical. Most authorities, however, seem to consider sclerosis, occurring in the form of multiple foci, a separate condition from similarly multiple foci of inflammation: in other words, although sclerosis may be secondary, it may also be primary, at least as regards inflammation. That this case is one of the latter type is suggested

by the neuropathic history, the absence of any other signs of chronic infection, the gradual progress from the onset on, the symptoms showing a less absolutely destructive lesion than inflammation would be likely to produce, no pronounced pain or atrophy or paralysis, and the strict adherence to the rather definite and fixed symptomatology commonly found in cases which have afterward been shown post-mortem to be sclerosis. Still, if a disease with multiple foci cannot be attributed to vascular conditions, nor to that most common infection of the nervous system, syphilis, no stone should be left unturned in the effort to unearth another chronic infection, febrile onset or not, typical symptoms or not. Unfortunately, this patient's psychic condition has been just enough altered to make it impossible for any of the number of physicians who have seen him to interest him in any further diagnostic measures.

Brain Lesions. With the exception of the flaccid condition of the arms and the wasted interossei, we could attribute this trouble to conditions entirely within the brain, or rather the cranium. Imagining a lesion in the motor cortex, or the internal capsule on each side, and in the sensory area of the cortex on one side, or the posterior limb of the internal capsule, we can account for the spastic paralysis and the hemianesthesia, not with single, but with multiple lesions. The condition of the second and the sixth nerves and the mental condition would require still more lesions to be included. We should even then have to leave the brain to account for the arms.

Huntington's chorea, a diffuse chronic inflammation of the cerebral cortex, was ruled out by the amount of disability, the sensory and cranial nerve involvement, the condition of the arms, and the different character of the speech defect and tremor.

Paresis, presenting a similar anatomical lesion to the above, did not have to be ruled out after a consideration of the mental defect. The changes in our patient seem to be more those of disposition and concentration, and are not very marked except on occasion. Seiffer, who made a study of mental states in multiple sclerosis, believes there is no qualitative difference between the dementia which occurs in multiple sclerosis and other dementia. Dercum has reported a case which was diagnosed as paresis during life, in which a post-mortem examination showed multiple sclerosis with most of the lesions in the cortex. Usually in paresis the tremor is finer and there is seldom nystagmus, and the symptoms are those of a diffuse rather than multiple lesions.

Brain tumor is not suggested except by vertigo, optic neuritis, and the cerebellar staggering, which in this case point undoubtedly to lesions in the cerebellum, perhaps several large lesions fused into one, but which do not point

to an exclusively cerebellar involvement. The symptoms which might be referred to increased intracranial pressure can better be accounted for by multiple focal lesions.

Finally, we may consider a form of *pseudo-sclerosis*, of which only a few cases have been reported, in which the symptoms are identical in all respects with multiple sclerosis, but which post-mortem shows no lesions. This condition certainly could not be ruled out if such a condition exists. It is difficult to imagine any functional disorder doing exactly this, but when this possibility was mentioned to the patient's wife she expressed an opinion that if it could take place with anyone, it could in her husband, as she has considered him highly neuropathic for many years.

There are probably as many more conditions which might occasionally have to be differentiated from multiple sclerosis, for a disease with multiple lesions naturally takes multiple forms. The three symptoms first mentioned by Charcot are strangely likely to appear in every case, although it has been recognized that they frequently do not go on to completion in this disease as in some others. Their great prevalence may be due to the fact that we do not often make a diagnosis of multiple sclerosis without them. Given them, we have the disease; given the disease, we have them. The diagnosis is made about four times as often in Europe as here, perhaps because they make it on the grounds of multiple lesions, regardless of the presence of the Charcot triad, which, it seems, might easily be absent, even with many other lesions present.

The gait is often the first symptom which is particularly observed. It usually shows some combination of the spastic and the cerebellar. It is rare to find the lower extremities uninvolved. Unilateral cases have been recorded and those which changed from side to side during the disease.

The tremor is usually, as in this case, more marked in the upper extremities. My patient knocks most things he reaches for onto the floor, and can handle only those things which are unbreakable. He drinks through a rubber tube because a glass cannot be put near his mouth without risk of his breaking it by intention tremor of the hands.

The nystagmus is, in this case, of the type it should be in order to correspond with the tremor of the intention type. His nystagmus comes on only when he attempts to use his eyes for a definite purpose. While sitting at

rest, with his eyes not especially in use, there seems to be no nystagmus, but in trying to fix on an object at an angle, without turning the head, the nystagmus becomes quite marked. I believe it is recorded that about 14% of cases show constant nystagmus. It seems reasonable to think of the nystagmus as intention tremor of the eye muscles, but since the eye muscles are virtually in use all the time, a constant nystagmus might very well occur.

Some authorities consider optic atrophy more important than nystagmus, but since a plaque need not necessarily be located so as to cause optic atrophy although many plaques might be demonstrated in the body elsewhere, it seems to me that nystagmus would be more typical, while possibly optic atrophy may be an incident which is equally common. Vision frequently does not correspond to fundus finding, and often suggests hysteria. This is undoubtedly due to the fact that the optic nerve may be involved in neuroglia proliferation while at the same time the axis cylinders may remain intact. Pupils do not seem to be typically involved in this disease, nor do cranial nerves, although the sixth was involved in sixteen out of twenty cases I have seen.

The sensory symptoms are not often as well defined as in this case. Fremd says they are usually at the distal portions of the limbs. They have been throughout distributed only to one-half of the body in this case. As in the case of many of the symptoms of multiple sclerosis, their transient quality and changeability make one think of hysteria.

Vertigo, which was present in this case I am sure, at least part of the time, while he was still ambulatory, although he always denied it, has not troubled him, apparently, while sitting. If present in this disease it is difficult to say whether it is due to involvement of the cerebellum, or the nystagmus, and consequent dancing of objects before the eyes, or to diplopia. He denies diplopia, does not complain of the effect of the nystagmus when seated, and we are thus obliged to consider it as cerebellar. This is confirmed by the fact that his station was poor, apart from lack of good muscle control, even with the eyes closed.

It is rather odd that this disease should so systematically develop a tendency to spasmodic laughter. In this case it was not due by any means to lack of coördination of the muscles

used in laughing. The impulse to laugh seemed uninhibited. Crying has not been a prominent part of his program, and is seldom so to as marked a degree as laughing.

The cerebral features are often made to include the speech defect, but it seems more natural to consider it a motor trouble, spasm of the muscles of articulation. The patient pounced on the end of each sentence in the same way that he pounced on an object when grasping it. Excitement greatly increases this symptom, as it does most of his symptoms.

Charcot, long ago, described a typical facies, —lips hanging down, half open, expression of mild stupor. This exactly describes this patient. Until I saw a picture of him taken ten years before I had supposed it to be more or less his natural expression, increased by illness. But it is plainly a change entirely due to the disease. There is no loss of fat in the face, but considerable elsewhere. Actual atrophy does not appear to have occurred apart from the interossei and possibly the thighs.

In the consideration of the probable pathology in this case, the idea is very strongly suggested that whatever agent is responsible for the condition, its work began in his childhood, before his tendency to lie on the floor began,—a habit which was considered by his family to be undoubtedly due to some discomfort he would never mention, and before the first appearance of strabismus. One would certainly be justified in considering the possibility of a specific infection of some kind which attacked the mother, the mother's sister-in-law, and the patient. Nothing can be recalled of an acute attack, but the history included a short time when the two families were more or less closely associated domestically, during the patient's childhood. The symptoms of nervous diseases came on more rapidly in the aunt, but subsequently were almost matched by the unrelated patient. The mother had meanwhile developed many similar symptoms to a very much less degree. As regards the location of the lesions I would venture to prophesy that autopsy will reveal several plaques in the lateral columns of the lumbar region, others in the anterior horns of the cervical region, one in the posterior limb of the internal capsule, others in the nucleus of the sixth nerve, one behind the optic disk of each side, and many small ones in the frontal region.

Since the patient refuses all medical care, no

treatment is possible. An autogenous vaccine has been urged but refused. Not much is permitted, even in the way of nursing and hygiene. Unless his will power weakens or his sufferings increase, it will probably continue thus until he ultimately succumbs to intercurrent disease,—nephritis or pneumonia, most likely. The apoplectiform and epileptiform seizures, to which he is liable, may cause complete disability at any time, although it is reported that even a complete paraplegia following such an attack may be recovered from. Several remissions may be considered to have occurred in this case, since he got around again twice after having been temporarily bed-ridden. Bulbar accidents may be looked for, in which case death would be directly due to the lesions of the disease, a rather rare conclusion. If the medullary centers were involved more often in this disease it might be better for its unfortunate victims.

APPENDIX.

Five years after the above history was written the patient is still living, now quite helpless. He has been bed-ridden for four years, but can be moved to an invalid chair, where with the aid of supports he sits for a few hours every day. He has no motion of any limb except a useless amount in the right arm. He is unable to see for reading, but can recognize people and objects fairly well. Optic atrophy has progressed to a considerable degree. Other neurological findings have remained about the same.

He has twice had attacks of retention of urine, which lasted several days, requiring catheterization, but both times he came back to his previous condition of continuous involuntary flow from a full bladder. He has had little rectal retention throughout. In spite of poor nursing at various stages, he has not developed trophic sores. Speech is almost incoherent, and the mind seems rather feeble. He has kept in a very cheerful frame of mind, frequently having bursts of uncontrollable laughter, which are perhaps not as merry as they sound. He shows a rather childish irritability, some loss of memory for recent events, considerable loss of the ability to concentrate, great indifference to the cares of the world. The fact that his wife must support the family leaves him quite content, although he would have been horrified at the thought of it a few

years ago. The death of his almost worshipped daughter made very little impression on him. His own comfort, however, matters a great deal.

There has been no treatment of the case, except of symptoms as they appeared, and even that was sometimes refused absolutely. Efforts have been largely directed toward keeping his mind content and his courage good, and also toward keeping him from the restlessness which has been his great trouble. It is fortunate for these chronic invalids that there are so many cults which promise mental healing. He has tried one after another and has gotten happiness from each in turn. His former intelligence would have scorned to be helped by anything save established philosophy, religion and psychology, but in his enfeebled state, these fantastic cults have been invaluable, chiefly because they promised cure, which, of course, nothing more scientific could do. He is unable to acquire in legitimate ways the state of mind which would enable him to cope with his condition. Lately he has become more and more a burden to himself and his family and is less interested in cures. Sometimes he asks for drugs so that he can "go to sleep and forget."

CLINIC ON THE DIAGNOSIS AND TREATMENT OF NEUROSYPHILIS.*

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(Concluded from page 750.)

CASE 12. *Tabes Dorsalis*: Orissa I. is a woman 37 years of age. For the past eight years she has been troubled with acute lancinating pains of the legs, which at times had been so severe as to necessitate hypnotics and prevent her from working. She had been told that she had neuritis, and was given various drugs, which she claimed had had no effect. In addition to her pains, she had some difficulty in walking, especially in the dark. The neurological examination showed unequal pupils, which reacted poorly, absent knee jerks and ankle jerks, and a positive Romberg. The blood and spinal fluid were positive. Everything, of course, was consistent with the diagnosis of *tabes dorsalis*. Under intravenous injections of *arsphenamine*, there was marked improve-

ment of the symptoms. The pains have entirely disappeared, and have been absent for a period of a year. The gait is markedly improved. The spinal fluid, however, remains positive. According to my experience, a few intraspinal injections would probably change the spinal fluid reactions to normal, and these injections are now indicated.

CASE 13. *Improvement following Treatment in the Paralytic Stage of Tabes Dorsalis*: James F. is a man 34 years of age, who was practically bedridden as the result of his *tabes*. He had all the classical symptoms of the disease, including *Argyll-Robertson* pupils and *strabismus*, absent knee and ankle jerks, impotence and such marked *ataxia* that he was unable to walk even with the aid of canes. When seen at the hospital, he had already received quite a number of intravenous injections of *arsphenamine*, but had steadily become more helpless. He was then given intraspinal injections of *Swift-Ellis* serum, reinforced with about one-third of a milligram of *arsphenamine*. He has now had nine such intraspinal injections, and at the same time is able to walk fairly well without the aid of a cane, and with the aid of a cane is able to go about freely. When he first came to the clinic he was carried from a taxicab to the operating table in the arms of an attendant. Today he comes in the street car, and walks into the operating room unaided. This case is particularly interesting, not only from the therapeutic results, but from the fact that when seen he had a negative blood and spinal fluid. It is generally believed that the cases of *tabes* most amenable to treatment are those which show high cell count and evidence of an active inflammatory process. In a general way, this is quite true. However, this is somewhat of an exception to the rule, and brings out the rather interesting point, that starting with the practically negative spinal fluid, a couple of intraspinal injections resulted in the production of a comparatively strongly positive spinal fluid, which remained positive for a number of months and then again approached a negative condition.

I have no hesitation in insisting that much symptomatic improvement may be obtained in the treatment of *tabes*. The earlier the case is treated, the more effective the treatment. However, good results are not infrequently obtained in the late cases. This does not mean that every case of *tabes* will show satisfactory improvement, but that many will. Theoretically, the results are obtained by an improvement of the inflammatory process, the removal of the spinal cord irritation, which then allows the patient to utilize the undestroyed tissues, which, in many cases, are quite sufficient to carry on all necessary functions.

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