ataxic gait; can scarcely walk; paresthesia, but no objective sensory changes except possibly some diminution in senses of touch and position; legs very ataxic; Babinski positive on left; doubtful dorsal flexion on right, clonus of both ankles and increased knee jerks. Six lower teeth removed and cultures showed presence of hemolytic streptococcus; injected into rabbit, caused total paralysis of hind legs and incontinence of urine and feces fourteen days later.

FACIAL DIPLEGIA IN MULTIPLE NEURITIS

By Hugh T. Patrick, M.D.

Infrequency of facial diplegia in multiple neuritis. Etiology. General clinical picture and variations. Relations of double facial paralysis in multiple neuritis to rheumatic facial diplegia and to ordinary facial palsy *ex frigore*. Report of a case. (*See this Journal*, vol. 44, p. 322.)

Dr. Sidney I. Schwab thought the most interesting feature of Dr. Patrick's case, the history of the multiple neuritis with facial diplegia, was the involvement of the bladder and rectum. He had had two cases, in one of which death occurred, the other was with recovery; in both the bladder and rectal reflexes were lost. That brings up two points of particular interest: one is the diagnostic side of these cases, that is the differentiation from so-called myelitic processes and, second, the mechanism of the process itself. It seems almost a clinical proof of the work of Rows, who showed by experimental work that infectious material could be carried through the sheath of the sciatic nerve in certain instances to the spinal cord. Dr. Schwab thought the method by which the spinal cord is finally attacked is through the sheath of some of the peripheral nerves attacked. He believed it is impossible anatomically to produce bladder and rectal insufficiency purely through the peripheral mechanism. It is too complicated and there are too many collateral branches, and it must be concluded that in these cases there is some definite process which reaches the cord along the nerves which happen to be attacked. It would seem we would have to face a toxemia rather than ordinary processes. The other feature Dr. Schwab mentioned is the curious emotional state of these patients. The neuritis itself may be somewhat insignificant, but in the midst of this neuritic complication the sudden or even gradual paralysis causes in these patients, at least in the two he had observed, a most profound emotional disturbance. The last patient he had become very emotional and very depressed, slightly maniacal, and with the disappearance of the facial diplegia these symptoms disappeared. The question arises whether that was not due to the disturbance in the mechanism of expression rather than to anything else. Dr. Schwab had seen it in unilateral facial palsy where the patient becomes suddenly aware of the palsy.

Dr. William G. Spiller said that on account of Dr. Patrick's statement regarding the infrequency of bilateral facial palsy in alcoholic multiple neuritis he desired to place a case on record. A man, aged fifty-seven years, in his service at the Philadelphia General Hospital in 1914, who had used alcohol excessively and had been repeatedly in the drunk ward, had the history of paralysis of the limbs developing as in Landry's paralysis, *i. e.*, the lower limbs were affected before the upper. He was said to have had pain at the onset of the palsy. When seen by Dr. Spiller the lower limbs were almost completely paralyzed but not much wasted. He was able to draw either lower limb up slightly at the hip by the muscles of the trunk. The patellar and Achilles reflexes were lost on each side. The lower limbs were very flaccid. There was little if any pain in the lower limbs in squeezing the muscles and no spontaneous pain. Tactile and pain sensations were normal

everywhere. He had involuntary evacuation of bladder and rectum, possibly on account of his mental condition, and this involuntary evacuation had been of short duration. The thoracic and abdominal muscles were moved freely in breathing. The upper limbs were very weak but not so weak as the lower. The biceps and triceps reflexes were lost. The soft palate, tongue and masseter muscles were moved normally. The lower distribution of the facial nerve was completely paralyzed on each side, and the upper distribution of this nerve was almost completely paralyzed. He could not pucker the lips nor blow out a lighted match. He could close the eyelids only to about half the normal degree. He could not wrinkle the forehead. His voice was peculiar, probably as a result of the facial palsy. He fabricated greatly, said that he had been out of the hospital the day previously, etc. The left pupil was larger than the right. Both irides reacted sluggishly to light, but the Wassermann reaction of the blood was negative.

The man gradually recovered and after some months was dismissed from the hospital.

Dr. Spiller referred to a case of tetanus in a boy he had seen in the Philadelphia General Hospital. Several weeks after the onset of the tetanus when the boy was almost well he suddenly developed unilateral facial palsy. As this paralysis developed as the tetanus was almost cured and without any increase in the symptoms, it was difficult to attribute it to the tetanus, although facial palsy is known to occur in some cases of tetanus. Careful inquiry revealed the fact that the boy had slept in a draught from an open window. The tetanus may have predisposed the nerve to palsy.

Dr. Archibald Church said there was admitted to the Neurological Service at Michael Reese Hospital last November a young man who presented the condition Dr. Patrick has outlined, a rather mild multiple neuritis with diplegia. He made a searching investigation for the cause of the disease. The spinal fluid, blood and everything else which was open to laboratory investigation were entirely negative. The causation therefore was obscure, but this boy's occupation which was a fairly wholesome one, that of a bundle boy in a large retail establishment, gave a little clue. He was excessively fond of candy, to which he had access and ate one to two pounds a day. After the onset of symptoms which were largely sensory, at first, a severe diarrhea developed, attended by numerous very offensive greenish stools. A careful search of the intestinal tract after his admission to the hospital revealed nothing abnormal. His pain was extraordinary, so that he required opiates to control it. He would scream with pain unless this measure was taken. The amount of wasting of the muscles was insignificant. There was slight, practically only a quantitative electrical change. The reflexes were all abolished. Motion was so impaired that he could not rub his face with his hands and the legs were quite inert. He recovered rapidly and was discharged in ten weeks able to walk and feeling quite comfortable.

Another case Dr. Church saw many years ago which followed acute intestinal disturbance. He developed the ordinary type of neuritis, but extremely severe in degree. In this case the muscles of the chest were involved, so that respiration became entirely diaphragmatic and the action of the heart was involved. He could not swallow. Both sides of the face were involved. The man recovered after a long time, but with contractures in the fingers and toes which persisted for many years. In both these cases the presence of intestinal features was of interest in this connection.

Dr. I. Strauss called attention to the possible origin of these cases and that of Bell's palsy. It has always seemed to him that the explanation of exposure to a draught in a susceptible person was very insufficient to explain that condition and that possibly there may be a susceptibility of that nerve in

that individual to a toxin which might be bacterial in its nature. Now, of course, all the members of this Association are familiar with the fact that in Chicago almost every disease has come to be regarded as a streptococcus. He merely mentioned the thyroid, gallbladder, the appendix, the joints, arthritis deformans, lastly cysts of the ovary and now, finally, the nerve has come and the spinal cord. The article read by title by Dr. Williams is a case in point and recently before the Society for Clinical Investigation, Rosenow, who has done meritorious work in this field, reported a case of this type. He thought the individual had a multiple neuritis and out of the tooth cavity he obtained the streptococcus. He injected it intravenously into the animals of four different species. In these animals he obtained lesions of the nerves which he has described as neuritis. In a large percentage of the animals the organism showed selectivity for these nerves. Now Rosenow has shown, whether conclusively or not Dr. Strauss did not believe we are as yet in a position to speak, that the streptococcus has apparently a selective affinity for different organs of the body and his latest work has demonstrated that it is possible for this organism to have a selective affinity for the nerves. It may be an interesting piece of research upon the part of some of us to see whether in these cases of Bell's palsy, so-called, even in the cases reported by Dr. Patrick, there may not be at the basis some streptococcus which lies hidden either in a tooth cavity or the tonsils, or elsewhere which may have a selective affinity for such a nerve.

Dr. Charles L. Dana said that the type of cases described by Dr. Patrick was quite rare. In his service in the alcoholic wards of Bellevue for twenty-five years there never occurred a case of facial diplegia. He had had, however, one case which falls definitely into the group that Dr. Patrick has correctly described. This was a case of a young man, a tennis player, very active, athletic and a moderate drinker, who went down to North Carolina and while there, exposed to a good deal of cold and wet, developed a moderate degree of multiple neuritis and very complete facial diplegia. His multiple neuritis was of the type that we used to call pseudo-tubes, *i. e.*, it was of a sensory type, and the patient had more ataxia than he had motor weakness. He gradually but slowly recovered from his neuritis and almost completely from the diplegia. Dr. Schwab has spoken of the mental state. This patient, ten years afterwards, developed melancholia and shot himself. Dr. Dana thought the suggestion Dr. Patrick made that these diplegias are infectious and toxic in character is in harmony with what has been believed in regard to the pathology of facial paralysis, and he did not see that the fact that a neuritis is unilateral need exclude infection. We get infectious herpes. It seems along that line we shall discover the cause of the disease.

Dr. Smith Ely Jelliffe asked what is susceptibility. Dr. Schwab treads very lightly upon some aspects of the emotional side of the problem. The very interesting series of alliances that Dr. Patrick has pointed out could be enlarged. There are other groups of cases which should be brought into comparison with those which he has mentioned. These are the cases that one sees on the psychotic side. These include the melancholias, the hysterical diplegias, the hysterical monoplegias, katatonic facial blockings, etc. If one will take a comprehensive view of the entire organism the question will have to be answered in a much wider sense than either toxemia or heredity. Toxemia can not explain a hysterical facial palsy. What lies behind the individual susceptibility in these cases? It certainly is to be recognized that a toxic factor may be one of the links in the chain, but what about the constitution of the nervous system itself? What about the variations in the makeup of the nervous system? What about those conditions to which

Adler has called specific attention, that is, organic inferiorities with their compensatory psychological equivalents.

Dr. John H. W. Rhein asked how many other cranial nerves were implicated in the case cited by Dr. Patrick. In a case of profound multiple neuritis that he saw recently, there was almost complete paralysis of the arms and legs. In addition to this there was paralysis and atrophy of the tongue, paralysis of the muscles of the pharynx, the masseter muscles, and bilateral facial paralysis and paralysis of both external recti muscles. The man made a remarkable recovery at the end of five months with only slight tongue atrophy and small hand muscle atrophy on the right side.

Dr. S. Leopold wished to place on record a similar case in which the etiology was clear. It showed the typical facial diplegic and multiple neuritic phenomena in which the origin was given as quinsy. Quinsy is associated with streptococci. The etiology might be fairly closely associated.

Dr. Hugh T. Patrick did not care to discuss all the questions brought up because he was not equal to it. He did not see that we must presume involvement of the spinal cord to explain incontinence. We know that in these toxic cases, as indicated by others here, the distribution may be very peculiar. Dr. Rhein's case is practically a multiple neuritis of the cranial nerves. There are cases even in this group in which contrary to the rule of the distal end of the extremities being more involved, the proximal muscles are more involved. He had seen a case in which multiple neuritis was limited to the pelvo-femoral group of muscles. He did not see why in a bad case peripheral involvement of the supply of bladder and rectum should not be a cause of incontinence. He had had one such case in which perfect recovery ensued. If due to bad involvement of the cord that is not very likely. However, there is no reason why this infectious agent should not involve the cord as it does in some of the Landry cases. There was no emotional disturbance in his own case except what was natural to the patient. She was temperamentally cranky. In regard to tetanus, the case spoken of by Dr. Spiller, at any rate it occurred post infection to the rabic virus. Whether Dr. Spiller's case was caused by actual exposure to cold or whether it might have been due to tetanus is open to question. The element of pain Dr. Patrick did not mention. That varies exceedingly. Some cases were absolutely painless, some very painful. He was very much obliged to Dr. Strauss for bringing up the matter of one-sided infection because this question is considered in the paper as written. It is hard to be explained by local infection of the facial nerve and also by a general infection by the streptococcus. These questions we will have to leave to men like Rosenow.

OBSTETRICAL PARALYSIS

By John Jenks Thomas, M.D., and James Warren Sever, M.D.

A study of nearly five hundred cases of obstetrical paralysis; analysis of 160 more recent cases. Pathology, etiology, treatment, including a new operation for the correction of the deformity following this type of paralysis. (*See this Journal*, vol. 44, p. 289.)

Dr. Charles K. Mills had seen many cases of obstetrical paralysis and had also published something with regard to traumatic brachial palsies. One point seems to have been omitted, or else he did not listen with sufficient attention. These obstetrical palsies are due in a considerable percentage of the cases not to the direct lesion of the brachial plexus or of the shoulder, although these may be present, but to avulsion of the nerve roots of the