DISCUSSION.

DR. BEEM—This is the room that has been constructed by Dr. Jaques for the purpose of demonstrating the method of disinfection as performed by the Chicago Health Department (showing model). The rubber material over the outside is probably somewhat pervious to the gas, and part of it escapes during the time of the disinfection, but cultures have been placed in the room to determine the force of the disinfection. They are placed at various points. The disinfection simply consists in stretching a clothes-line across the room at any suitable place, usually as near the center of the room as possible; then on that clothes-line suspending a sheet by its edge. For every 1000 cubic feet, we will say one bed sheet, and on that sheet we sprinkle 150 c.c. of the formaldehyde solution, or five ounces, by means of a proper sprinkler. I show you the sprinkler we are using at the present time. It simply consists of an atomizer, and the tube is arranged so that not too much formaldehyde gets out at a time. Then all the clothes in the room are hung out on the clothes-line, beds are opened up, and books opened and stood on their edges, so that the formaldehyde comes in contact with every part of the material. Then the rooms are allowed to remain closed for five hours. At the end of that time we find that all the germs in the room have been practically killed. This has been demonstrated several times to the entire satisfaction of the bacteriologist of the department, as well as myself. (Dr. Beem here showed tubes of cultures of diphtheria and typhoid.)

In the six hours which we generally take for practical house disinfection, the growth is very materially delayed. Even after seventy-two hours of incubation there is very slight growth. We have also tried the germ of scarlet fever, which has recently been discovered, and we find that it has been destroyed in about the same length of time. The test-tubes which have been exposed show the penetrating power of the gas. It is claimed by a great many that the gas does not penetrate into small crevices. (He then indicated on diphtheria tubes, how far the gas had penetrated, under different conditions. He found that diphtheria is very easily killed.)

The tubes shown were from actual disinfections of houses, so the work was not done for any experimental purposes especially, but the disinfectors were given the culture-tubes to expose in the rooms of houses they disinfected. The cryptococcus was found harder to destroy than any of the other germs referred to, and the penetration was not as far as in the other tubes. (He then illustrated, by the tubes, the comparative difference of the killing power of the formaldehyde gas in staphylococcus and cryptococcus, the latter showing the greater resisting power.) These were exposed seven hours. Continuing, he said that there is no method at the present time which he considers equal to this one in formaldehyde disinfection, as practiced by the Chicago Health Department. The simplicity of the method presents itself at once.

A MEMBER—What about the bedding, the bed-clothes, and the mattress?

DR. BEEM—The bed-clothes are suspended over doors; just take them and hang them over the backs of chairs, in order to expose them as freely as possible.

A MEMBER—And it destroys all growths?

DR. BEEM—It destroys all growths.

A MEMBER—What about the mattress?

DR. BEEM—The mattress we leave on the bed, just on the springs, just as it is. We suspend the sheet over the mattress as near as possible. At the present time there are a great many generators on the market for the formation of formaldehyde, which is a gas absorbed by water. The water will hold about 39.67 of the gas, or, in other words, make a 40 per cent. solution. That should be the purity of the commercial article. In the generators the water boils off very quickly, leaving a concentrated solution. This concentrated solution forms a white substance in the bottom of the generator, called paraform. That paraform forms trioxid methyoln, which is then passed into the room in that form, a substance having very little disinfecting qualities but a great deal of stink, as they say. To obviate this the operators at the present time use methyl alcohol; they also add neutral salts; common table salt, or calcium chlorid is more often used.

POLIOMYELITIS ANTERIOR ACUTA
ADULTORUM.*

BY HERMAN H. HOPPE, M.D.

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Poliomyelitis anterior acuta adultorum is a disease which, although it has been described many years ago, at first by M. Meyers, and later by Gumboldt in 1873, and by Shultz in 1878, is of extreme rarity, and on account of the difficulty of differential diagnosis is of sufficient interest to place two well-marked cases on record.

Rand was able to collect only thirty-six cases in the adult, and in a period of ten years since I have been interested in nervous diseases, I have had an opportunity of observing but two. On account of the newer views of the pathology of poliomyelitis, and especially on account of the difficult differential diagnosis and the unusual prominence and the sharp definition of newer diseases, such as peripheral neuritis, many cases of which were formerly grouped under poliomyelitis, it is of especial interest to place on record two cases which are beyond dispute.

In 1897, when Bruns and Windschild wrote an article on this form of poliomyelitis, for the "Twentieth Century Practice," they said: "We must admit, however, that there exists in the adult a disease which is wholly analogous to infantile spinal paralysis. This poliomyelitis adultorum is, however, in itself, and especially in comparison with infantile paralysis, a very rare disease."

CASE 1.—Edward B., aged 28, unmarried, has previously been perfectly well. His mother died of tuberculosis, and he has one sister who is deformed as a result of caries of the spinal column; a step-brother, child of the second wife, shows marked imbecility closely resembling cretinism. The patient himself denies having ever had any venereal diseases, especially syphilis.

On April 1, 1895, while rapidly walking from his residence to the railway station, a distance of probably a thousand feet, the patient noticed, as he stepped on the platform of the station, that his left leg was growing weak, and the moment he stepped on the rear platform of the car he fell to the floor and had to be assisted to a chair. He had no pain nor any other abnormal sensation. Arriving at the city station he found that he could not use his left leg, except with great difficulty. Calling a cab he rode about town, attending to his business, but when he returned home at night he had to be carried from the train on account of the involvement of his right leg. On the next day he was without fever, but was unable to leave his bed.

Dr. F. W. Langdon, who was with him the day after the onset of the paralysis, states that both arms were affected, but the patient was still able to make some movement with his hands and fingers; both legs were completely paralyzed. He reports that the sensation of both arms and legs and all its qualities were perfectly normal.

I saw the patient the next day, i. e., forty-eight hours after the onset of the paralysis, when there was present a complete motor paralysis of all four extremities, together with the muscles of the neck, so the patient was unable to raise his head from the pillow or move it from side to side without the assistance of the nurse.

Examination.—Intelligence seemed normal; the pu-

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pils equal in size, and they reacted both to light and accommodation. The papillæ were normal, and there was no defect in the movements of all external of the muscles of the eyeballs. The muscles of the face were normal. The tongue protruded in the median line; the soft palate was normal. Sensation of the scalp and face was normal on both sides. There was no loss of the sense of smell or taste, but complete absence of all voluntary movements of the muscles of the neck; the patient was unable to turn his head from side to side or raise it from the pillow. The muscular movements of both the right and left arm were reduced to a slight movement of the fingers; he was unable to raise the arm from the bed or to flex the arm on the forearm; and could not use the pectoral muscles nor shrug the shoulders nor lift them.

There was no tenderness of the nerve trunks; no hyperesthesia thereof, nor any diminution or loss of sensation in any of its qualities. The lower extremities were absolutely immovable and there was the same complete absence of tenderness on pressure over the nerve trunks or of any disturbance of sensation. The appetite was good and there was no disturbance of the action of any of the sphincters, nor loss of sexual desire or the power of erection.

The electric examination, one week later, showed a complete loss of all faradic irritability, both direct and indirect. The galvanic reaction through the nerve trunks was absent in all of them, with the sole exception of the right ulnar nerve. The direct response of the muscles was weak and slow. In a word, there was a complete reaction of degeneration in all the muscles of the body, including the neck.

The return of muscular power began to take place after the first week. The patient was soon able to move his legs, arms and head, but the movements were characterized by great weakness, and it was only in the third week that he was able to use the muscles of his right thigh at all. After this there was a steady progress, the patient gradually gaining control of most of his muscles. After three months he was able to walk, with the assistance of a nurse. There was great wasting of the muscles, and the patient's weight was reduced to 106 pounds.

Treatment.—Ergotin was used for the first three days, two weeks after that small doses of iodid and bichlorid of mercury for three weeks; after the third week massage and general faradization. Massage and electric treatment were kept up for two years. After this period there still remained some general weakness, especially marked in the quadriceps femoris.

Examination, June 1, 1899.—Four years after the attack there is present a normal electric reaction of all the muscles of the body as well as their nerve trunks, with the exception of the left ulnar nerve, which does not respond normally to the galvanic current, although the muscles supplied by the ulnar nerve are prompt in their reaction. The volume of muscular strength has never returned to its normal condition. All of the muscles both of the upper and lower extremities are weaker than they were before the attack, but the patient walks without an effort, on the level, but has some difficulty in climbing steps. The muscles have not returned to their normal size. This is especially true of the legs. The patient thinks, however, that he has fully recovered.

CASE 2.—John R., aged 48 years, a native of Ireland, and keeper of a lunch stand, was admitted Jan. 8, 1899, to the neurologic service of the city hospital. He was

discharged May 2, 1899, improved. The family history showed that his father and mother, also two brothers were dead, the causes unknown. One sister was living and well. He had the ordinary diseases of childhood, malaria, piles for the past eighteen years, but no drug habits, and no syphilis. He had been perfectly well up to within one month, when he was attacked with diarrhea which weakened him very much, and during the course of this he lost seven pounds in weight. One week before admitted to the hospital, he noticed a weakness in the left leg, on rising in the morning, and this increased during the day. The right leg became involved on the following day and on the third he was confined to his bed, unable to walk; at the same time his arms became paralyzed, and at the end of a week, on admission to the hospital, he was perfectly helpless. He said that he had a chill and a high fever, but that disappeared on the third day. He had no pain at any time, no hyperesthesia or abnormal sensation of any kind, no headache, no dizziness, and the sphincters were normal.

Examination.—His mental condition was normal, memory good, and the pupils reacted to both light and accommodation. All regions supplied by the cranial nerve were normal. There was a slight nasal intonation of the voice, but no difficulty in swallowing and no paralysis of the soft palate. He was unable to move the head from side to side or raise it from the pillow. There was no voluntary action of any of the muscles of the neck, but absolute loss of all muscular power of the muscles of the upper and lower extremities. He was unable to move either arm or either leg, and there remained only a slight movement of the fingers. There was no hyperesthesia of the skin, no tenderness over the nerve trunks, no loss of sensation, no abnormal subjective sensation. There was an absence of all reflexes excepting those of the skin, pectoral muscles and all of the back, which were also paralyzed. The patient was able to flex the fingers and flex and extend the toes. Sexual functions were normal, and there was no involvement of the sphincters of the bladder or rectum. No evidence of syphilis could be found in any part of the body. Sensation in all its qualities, heat, cold, pain, and touch remained normal.

The electric examination showed an almost complete reaction of degeneration in all muscles of the body. (For details, see chart.) The urine showed a small quantity of albumin.

Treatment.—This consisted of 1-3 gr. of bichlorid of mercury every second day, and 1-60 gr. of sulphate of strychnin every four hours, together with a hot bath every afternoon at 4 o'clock. The patient began to improve almost at once. In twenty-four hours he was able to move the arms, flex the forearm on the arm, and after forty-eight hours could raise the arm from the bed, and move the legs. The improvement continued, and at the end of the first week he was able to raise the head from the pillow, and on the tenth day could walk with the assistance of the nurse. In three weeks he was able to walk alone, with ease, and could use all the muscles of the body, but the movements were characterized by great weakness. The electric irritability began to return after six or eight weeks.

Examination, May 1, 1899.—The results with the faradic current, under Dr. Hoppe, showed both pectorals negative; the median nerve reaction was present but very small: the musculospiral absent on both. The median nerve responded better on the left than the right. Muscular response was also better on the left. The faradic response returned to all the muscles of the

HEAD and UPPER LIMB.		Right.		Left.		TRUNK and LOWER LIMB.		Right.		Left.	
		F	G	F	G			F	G	F	G
		Qnt.	Qlt.	Qnt.	Qlt.			Qnt.	Qlt.	Qnt.	Qlt.
Facial	Orbicularis palpebrarum ..		+		+	Intercostals ..	Intercostals ..		-		
	Lower facial muscles ..		+		+		Rectus Abdominis ..	0	-		0
Spinal Accessory ..	Palatal muscles ..						External oblique ..	0	-		0
	Laryngeal ..					Branches of Lumbar Nerves ..	Erector Spinae ..		+		-
Do. with II & III Cerv.	Sterno-mastoid ..	0	-	0	-		Quadratus Lumborum ..		+		
	Trapezius ..	0				Genito-crural ..	Cremaster ..		+		
Hypoglossal ..	Lingual muscles ..		+		+		Sartorius ..	0	-		0
Do. with II & III Cerv.	Sterno-thyroid ..	+	-	+	-		Pectineus ..				
	Sterno-hyoid ..	+	-	+	-	Anterior Crural ..	Rectus femoris ..	-	+		-
II Cerv. ..	Complexus ..						Vastus externus ..	0	+		0
	Splenius ..						Vastus internus ..	0	+		0
III Cerv. ..	Lev. anguli scapulae ..	+		+			Crureus ..	0	+		0
V Cerv. ..	Rhomboids ..	0		0		Obturator ..	Gracilis ..		+		
Post Thoracic ..	Serratus magnus ..	0		0			Adductor longus ..	0	+		0
Supra scapular ..	Supra-spinatus ..	0		0			Adductor brevis ..	0	+		0
	Infra-spinatus ..	0		0			Adductor magnus (with sciatic) ..	0	+		0
Ext. ant. thoracic ..	Pectoralis Major { Upp. part ..	0	-	0	-	Small Sciatic ..	Gluteus Maximus ..	-	+		0
Int. ant. thoracic ..	„ Minor ..	0	-	0	-		Sup. gluteal ..		+		0
							Tens. vag. femoris ..		-		
Musculo cutaneous ..	Coraco-brachialis ..	0	-	0	-		Biceps femoris ..	0			0
	Biceps ..	0	-	0	-	Great Sciatic ..	Semitendinosus ..	0			0
	Brachialis anticus ..	0	-	0	-		Semimembranosus ..	0			0
Subscapular ..	Subscapularis ..		-		-		Adductor magnus (with obturator) ..	0			0
	Teres Major ..		-		-	Int. popliteal ..	Gastrocnemius ..	0	-		0
	Latiss. Dorsi ..	0	-	0	-		Soleus ..	0	-		0
Circumflex ..	Deltoid ..	0	-	0	-		Tibialis posterior ..	0	-		0
	Teres Minor ..	0	-	0	-		Flex. comm. digit. ..	0	-		0
Musculo spiral ..	Triceps ..	0	+	0	+		Flex. long. hallucis ..	0	-		0
	Ext. Carp. Rad. Long. ..	0	+	0	+	Plantars ..	Flex. brev. hallucis ..	0	-		
	Supinator Long. ..	0	+	0	+		Flex. brev. digit. ..	0	-		
							Abductor hallucis ..		-		
Post interosseous ..	Supinator Brevis ..	0	+	0	+		Adductor hallucis ..		-		
	Ext. Carp. Rad. Brev. ..	0	+	0	+		Ext. brevis digit. ..		-		
	Ext. Carp. Uln. ..	0	+	0	+		Interossei ..		-		
	Ext. Comm. Digit. ..	0	+	0	+	Ext. popliteal ..	Tibialis anticus ..	0	-		0
	Ext. ossis metac. poll. ..	0	+	0	+		Ext. prop. hallucis ..		-		
	Ext. primi. intern. ..	0	+	0	+		Ext. digit. longus ..		-		
	Ext. secund. ..	0	+	0	+		Peroneus longus ..		+		
	Ext. indicis ..	0	+	0	+		Peroneus brevis ..		-		
	Ext. minimi digiti ..	0	+	0	+						
Median ..	Pronator radii teres ..	0	-	0	-						
	Palmaris longus ..	0	-	0	-						
	Opponens pollicis ..	0	-	0	-						
	Abductor pollicis ..	0	-	0	-						
Median & Ulnar (jointly) ..	Flexor longus pollicis ..	0	-	0	-						
	Flexor carpi radialis ..	0	-	0	-						
	Flexor sublim. digit. ..	0	-	0	-						
	Flexor brevis pollicis ..	0	-	0	-						
Ulnar ..	Flexor carpi ulnaris ..	0	-	0	-						
	Adductor pollicis ..	0	-	0	-						
	Muscles of little finger ..	0	-	0	-						
	Interossei ..	0	-	0	-						

upper extremities. The anterior nerve reaction was absent on the left. The anterior crural muscles were very slightly active. The anterior crural nerve reaction on the right was absent, but the anterior crural muscular response present, though diminished. Reaction of the left tibial was present, also the right, but absent in the left and right post-tibial; the muscular response also. The reaction was absent from the muscles of the calves. Atrophy of the muscles was still present, very few having returned to normal, the sternocleidomastoid being an exception. The response was more marked on the ulnar surface on the right than on the left, also in the small muscles of the right hand. There was well marked atrophy of the anterior surfaces of both thighs, especially in the quadriceps femoris.

In calling the attention of this Section to these cases of acute poliomyelitis anterior adultorum, I am convinced of the fact that I am not presenting anything new. I feel, however, that two such sharply-defined and well-marked cases as the above should be placed on record, especially as it is more than probable that some

of the earlier cases were confounded with peripheral neuritis or disseminated acute myelitis.

These cases are also interesting on account of the extent of the paralysis, all four extremities together with the muscles of the neck being involved. In the tables of cases collected by Duchenne and Seelegmiller, in children, one extremity alone was affected in more than 50 per cent. of cases, whereas the four extremities were affected in only 2 per cent.

Pathological Anatomy.—A change has taken place in our ideas of the pathologic anatomy of acute anterior poliomyelitis. Our first knowledge was based on the researches of Cornil and Lockhart Clark. In 1870 Charcot and Joffroy were the first to make known more generally to the medical world the relation between the disappearance of the ganglionic cells of the anterior horns and the flaccid atrophic muscular paralysis, and they formulate the law that the disease consists of a primary acute inflammation of these ganglionic cells and not of an acute inflammatory softening of the interstitial tissue or of a hemorrhage, and that all other

changes were secondary to the destruction of the ganglionic cells. It was very easy to take the next step and attribute all the diseases of the motor system, characterized by a flaccid atrophy of the muscles, into three groups and attribute them to an acute, subacute and chronic inflammation of these ganglionic cells.

We know how this erroneous doctrine jumbled together acute anterior poliomyelitis, peripheral neuritis—which was looked on as subacute anterior poliomyelitis—and chronic progressive spinal muscular atrophy, and taught us to look on them as of a common pathologic origin, the clinical difference being due to a difference in the degree of inflammation. This opinion was accepted universally, and still holds sway among the rank and file of the medical profession, although it has been overthrown as long ago as 1883, by the researches of Archambault and Damaschino, who showed that it was not a primary disease of the ganglionic cells but an interstitial myelitis of the anterior horns, vascular in its origin. This has been confirmed by the more recent researches of Schultze, P. Marie, Goldscheider, Siemerling and Redlick.

We to-day know that acute anterior poliomyelitis exists as a disease *sui generis*; that it is a distinct disease, the result of an infection, which has for its pathologic seat of predilection the gray matter of the anterior horns, and that the infectious material is carried to the horns by way of the central spinal artery. It is primarily a disease or an inflammation whose seat is the gray matter of the spinal cord.

An exudation of serum and leucocytes takes place around the small vessels, arteries, and capillaries. The ganglionic cells are affected secondarily, by the chemical action of the pathologic serum or by pressure resulting from the exudation of the inflammatory material. This inflammatory exudation is not limited to the gray matter of the anterior horns, but there is very frequently an inflammation with an exudation of the pia mater and the white matter of the anterior column at the point of entrance of the vessels which are the carriers of all the infectious agent.

If this view is correct, then all the apparent cases of acute anterior poliomyelitis following a previous acute infectious disease, such as measles, scarlet fever, typhoid fever and gonorrhea are not acute anterior poliomyelitis in the strict sense of the word, but a focal or disseminated myelitis. The distinction which I make will be readily understood. The seat of the inflammation is the same, whereas the disease in its etiology is radically different. Just why the central spinal artery should be the seat of predilection for the disease can no more be explained than the same phenomena in other acute infectious diseases.

These views are radically different, at least in the adult, from that expressed by Bruns and Windschild, who state that poliomyelitis adultorum is almost always seen as a complication of a sequel to acute infectious diseases. The two cases above recorded show that it may occur as a primary affection. The agent at work in producing the disease is not known. All the other causes, like over-exertion, heavy colds, exposure and traumatism are, in my estimation, only predisposing causes. This fact is further borne out in Rank's statistics—in twenty-five out of thirty-six cases, no other cause could be attributed than exposure to cold.

Symptomatology.—There is no need to dwell on the clinical symptoms of acute anterior poliomyelitis. They are the same in the adult as in children. The unusual feature about both of these cases was that the entire

body, from the angle of the jaw down—i. e., in the neck, upper and lower extremities—was completely paralyzed; the other feature was the absence of pain, and all other sensory disturbances.

Authors have called attention to the fact that pains may be present in the nature of shooting pains, or of tenderness over the nerve trunks. I believe that such are not pure cases of anterior poliomyelitis, but rather, either focal myelitis or multiple neuritis. Another feature that bears out the newer pathology views is that the ganglionic cells are affected secondarily, which in the primary diseases is borne out by the history of the first case, in which improvement in the muscular tone and power continued four years after the first onset of the disease. This ought to have an important bearing on our methods and manner of treatment; therefore no matter how small the improvement and how slowly it takes place, we are not to despair, but should continue to treat the case for two or three years.

Differential Diagnosis.—The question may arise, on account of the wide distribution of the paralysis, the involvement of the muscles of the neck as well as all four extremities, as to whether or not these two cases have been properly classified. There is no difficulty in ruling out multiple neuritis, on account of the absence of all sensory disturbances and of tenderness of the nerve trunks. Cervical myelitis can be ruled out by the absence of sensory symptoms and absence of involvement of the bladder and rectum. The progressive involvement from the lower extremities to the muscles of the neck would also render this untenable. The same reasons would hold for hematomyelia cervicalis. Landouzy and Dejerine have described what they termed "acute curable atrophic paralysis," in which, without much general disturbance, there is an equally distributed paralysis of all four extremities, rapid in its onset, wasting of muscles and reaction of degeneration. The cranial nerves are intact, there is no sensory disturbance and the sphincters are normal in their action. Recovery is perfect. It is generally held, a view which is clearly proven by the above symptomatology, that the above is only a modification of anterior poliomyelitis, and although both of our cases resemble the above very much, there is no need of quibbling about terms. The greatest difficulty encountered in both cases was the ruling out of Landry's paralysis, and it was not possible to do this with certainty for the first week or ten days. In this latter, of which I have seen but one case which ended fatally, the paralysis begins in the feet and extends upward, so as to involve the four extremities, muscles of the neck and, in fatal cases the medulla oblongata, and causes death by paralysis of the respiratory or cardiac centers; while the motor system is mainly involved, there may be sensory disturbances. The paralysis is of the flaccid variety, the sphincters are intact, even the involvement of the medulla with difficulty of breathing and deglutition does not afford positive evidence in favor of Landry's paralysis, for in acute anterior poliomyelitis the centers in the medulla may likewise be involved—Oppenheim, Goldscheider, etc. If the case does not end fatally within the first ten days or two weeks, the differential diagnosis, according to the original article of Landry's, is easy, for in his paralysis there is neither atrophy of the muscles, nor any reaction of degeneration. The observations of Landry were confirmed by Kussmaul, by Pelgimo, Levi, who reported the case of Cuvier, by Bernhardt and Westphal. The latter laid down the law that three factors are essential for the diagnosis of Landry's par-

alysis: 1. The ascending course of the paralysis, ending fatally. 2. Normal electric reaction of the paralyzed muscles. 3. Absence of all pathologic changes at the post-mortem examination. If we held strictly to these conditions, there would be no difficulty about the differential diagnosis, but the views have changed very much since then. Cases have been described in which the course of the paralysis has been a descending instead of an ascending one. Changes have been found in the electric reaction from a slight diminution in the quantity to a complete reaction of degeneration, although it is true that cases have been observed which existed for a month without showing any atrophy of the muscles, or changes in the electric reaction. Goldscheider vigorously opposes those who hold that cases which show these degeneration changes should not be classed with Landry's disease, but even goes farther and, basing his opinion on published cases and some of his own, states that we can have all degrees of change from a slight diminution to a complete loss of sensation, and in addition we may have atrophy of muscles and loss of function of the sphincters. With this state of affairs, we have a feeling of relief, when Goldscheider states that very many of the numerous cases reported must be looked on as doubtful.

Many pathologic changes have been found in recent cases since researches are made with better methods and with greater care; so that pathologically, Goldscheider, Oppenheim and others divide the cases into two groups: those in which there is a central inflammatory or hemorrhagic disturbance throughout the entire gray matter of the spinal cord and medulla, and cases which should properly be classed as acute peripheral neuritis. From all this we must conclude that, while Landry's disease may and does exist as a clinical entity, i. e., as an acute ascending paralysis; it has no distinct or individual pathologic basis. In anterior poliomyelitis in which there is an involvement of only one or even two extremities, there will be no difficulty in differentiating the condition from Landry's disease, but in cases like the two recorded in this paper, it seems to me that we have but one point to go by, the absence of sensory disturbances, either subjective or objective, in acute anterior poliomyelitis, and its presence in Landry's paralysis. In all other respects the two diseases may be identical.

DISCUSSION.

DR. F. W. LANGDON, Cincinnati, Ohio—It was my privilege to see the first of the two cases reported by Dr. Hoppe. There are one or two points in connection with that first case which have impressed me in thinking over the matter, since a short talk with Dr. Hoppe a few days ago. One is in respect to the young man; I was called to see him when I was just on the eve of a trip abroad, and recognized the condition as an unusually typical one of poliomyelitis. The history impressed me as of considerable interest. The evening before, as I recall it, he had gone to a restaurant, and eaten a large beef-steak, immediately after which he went to a gymnasium and took a cold shower-bath. He then spent the evening with some friends in the city. The onset, twelve hours later, of weakness in the leg, followed rapidly by paralysis of the whole body, was suggestive to me of a possible connection between arrest of digestion by the cold bath and a consequent ptomain poisoning. The next I had was an infant less than 2 years old, who had been given a large piece of bologna sausage by a butcher, and in two days also had a typical poliomyelitis anterior, so that the question of development of a special poison from dietetic products naturally suggested itself to my mind. As regards the distinction between poliomyelitis and Landry's paralysis if we are only going to say that one does and the other does not get well, that is not a very clear pathology.

We can readily understand the fact that an overdose of poison in one case might cause death while the patient might survive a less quantity. As regards Landry's paralysis, always beginning in the lower extremities and ascending, in about as typical a case as I ever saw the arms were first affected.

DR. W. A. JONES, Minneapolis, Minn.—I think I saw a case of poliomyelitis in an adult two or three years ago—a young man of 24, who had been previously well. He was taken during the night with some acute disturbance, the nature of which we were unable to determine. The following day he had a paralysis of one leg, which gradually developed, and a paralysis of the opposite succeeded, but was only affected in a very mild degree. He had absolutely no sensory involvement, and none of the bladder or rectum, but he had what a great many children have, a rapid wasting of the muscles. The condition went on for a year or two before I saw him, and by that time he had the usual contractures and had passed beyond the flaccid stages of poliomyelitis. It was the first case I had ever seen, and interesting on that account.

A. E. STERNE, Indianapolis, Ind.—I think I can add two cases to this, one is a colleague of ours, who is present at the meeting of this Association and whom I intended to have here this afternoon. I saw him, as Dr. Jones mentioned he did in his case, a short time after the acuteness of the illness had passed away. It seems, however, that in these two we have a distinction to make which Dr. Hoppe mentioned, i. e., in almost all of the cases reported—and they are few—an infectious disease had previously been experienced. He makes the distinction in his cases that these, therefore, are not to be classed truly as conditions of acute poliomyelitis anterior. That distinction is not, on the spur of the moment, easy to subscribe to, as I have always considered these typical cases of poliomyelitis. At the time I saw the first case of mine, I had not heard of a single one of adult poliomyelitis, and had supposed it to be unique, and so referred it to the medical society. The legs were not involved, but both arms were totally paralyzed. There was absolutely no evidence of a neuritic character; no pain, no pressure points, none of the significant marks which we usually associate with such disturbances. The man's father had had acute articular rheumatism, and his mother at the same time, about three months before he became ill. He had attended both. A short time before he became paralyzed in the upper extremities, he began to have pains in various joints, but the joints did not develop the typical pains which we are wont to see in acute arthritis. I have always regarded that case as a case of post-arthritis poliomyelitis, and the marks of the condition were typical enough; decrease of electric reaction without absolute reaction for degeneration; faradic irritability was lost, but the reaction for degeneration to the galvanic current had not set in at the time I saw him, nor had it ever made its appearance, because he was immediately put on treatment and has completely recovered. He was treated steadily for four months, twice a day with the galvanic current until the faradic irritability was restored, and after that with massage. He now uses his arm perfectly. At the time I saw him the supra- and infra-scapular, the deltoid and the extensor muscles were completely paralyzed. He now does his operating himself, and while he still has considerable atrophy, notably in the smaller extensor muscles of the hand, he can get along perfectly well. He is 25 or 26 years of age.

The second case occurred almost immediately after the first. It was also in a young man, who looked in about as near a moribund condition as a walking person could. He was brought to me for consultation, by his family physician, and had a severe typical malarial intoxication, giving the typical febrile reaction that we find in Indiana to day. Both of these developed suddenly, just as we find in children. Both of them, moreover, thought they had simply an acute gastritis. The paralysis was sudden and more wide-spread right at the start, although, as I say, the legs were not involved beyond weakness. The second patient did not respond to treatment. The atrophy remained. I concluded, however, that the ill-success with him was due to the fact that he was still suffering from intense malarial intoxication that had in all probability started the spinal disease. He dropped out of my sight, and

was taken to the hospital, and within a reasonable time, I think possibly four months after I first saw him, he died, and the post-mortem, so much as could be made, revealed a typical case of anterior poliomyelitis. It is the only typical case with a post-mortem that has been reported at all in the adult; the complete report of the case still remains in abeyance.

DR. AUGUSTUS A. ESHNER, Philadelphia—An alteration in any portion of the peripheral sensory neuron, or of the peripheral motor neuron, might readily give rise to the symptoms present in these two cases and similar ones. Nor need we conceive that alterations of this sort are necessarily dependent on any one single toxic influence. There are a variety of agencies, some of bacterial origin, as the infectious diseases, some of toxic, as from poison introduced from without, or generated within, the body, as alcohol, mercury, lead, gout, diabetes, uremia, that are capable of affecting the peripheral motor or sensory neurons, and at times it is exceedingly difficult to make the differentiation.

DR. F. SAVARY PEARCE, Philadelphia—Dr. Sterne, in saying that malarial intoxication was a probable cause of the paralysis, reminds me of a case of locomotor ataxia which I reported last year, in which, so far as I could trace the etiology, the tabes was due to acute malarial intoxication. The case I reported was a precocious one, occurring in a young man in whom there was absolutely no syphilitic history. Overwork alone as the probable cause, was also ruled out when it was learned that the symptoms rather suddenly supervened on acute severe malarial fever.

DR. H. H. HOPPE, Cincinnati, Ohio—Many of the cases of anterior poliomyelitis in children are not poliomyelitis in the strict sense of the word, but focal myelitis. We know that epidemics of poliomyelitis have been reported, one in Stockholm, where thirty cases occurred in a single physician's practice. While the anterior horn or the gray matter of the anterior horn is not a very large area, small areas are perfectly capable of setting up in children or adults a focal myelitis that would simulate poliomyelitis, but I did not regard them as true cases of poliomyelitis. I have placed these two on record because of the extensive involvement of both sides from the lumbar to the cervical region.

CASE OF SPINAL ANTERIOR SUBACUTE GENERAL PARALYSIS.

BY C. EUGENE RIGGS, A.M., M.D.
ST. PAUL, MINN.

On Nov. 24, 1897, Miss A. B., aged 27, a student, and single, consulted me. She had always been perfectly well, with the exception of an attack of measles six years before. There was no history of syphilis or tubercular disease in the family, and no neurotic tendency. Six months before I saw her, she first noticed difficulty in walking. For a year previous to that time she had suffered from cramps in the calves of both legs, which, later, involved the thigh, abdominal muscles and both forearms. At the time she first noticed the trouble in walking there was no pain, but the gait gradually became more and more involved until when I saw her it was of a hobbling character. There was marked weakness of the tibial and peroneal groups of muscles in both legs. The limbs were cold and white. The thigh muscles, posterior aspect, were paretic, especially on the left side. The psoas iliacus and abdominal muscles were also paretic. The patient could not lift herself into the upright position when lying flat on her back. Flexion and extension of the muscles of both legs were much impaired. The flexion of the left foot on the ankle was very slight, and there was some diminution of the same motion in the right foot. The muscles of the legs were greatly atrophied. The organic reflexes were unimpaired. There was static ataxia. Sensibility was normal. There were no sub-

jective sensations. The knee-jerk was normal, there was no ankle-clonus, and the superficial reflexes were normal. The motility of the arm was impaired and the deltoids perceptibly weakened. The dynamometer showed the right hand twenty; left hand, fifteen. Flexion and extension of the arms were impaired. Sensibility of arms was normal, and there were no subjective sensations in them. The wrists and triceps reflexes were normal. The pupils reacted normally to light and accommodation, the ocular muscles were normal, there was no vasomotor disturbance, the mental condition was unimpaired, and no disease of the kidneys was present.

The development of the disease in both extremities simultaneously, together with the extensive loss of power and the fact that the knee-jerk was preserved when there was such extensive involvement of the muscles of the lower extremities, created in my mind a doubt as to whether the trouble might not be peripheral rather than central. It seemed possible the case might be one of those described by Gowers as a purely motor neuritis, and therefore painless. The course of the disease, however, dissolved this doubt. The patient went into the hospital for four months; shortly after she left, atrophy of the left thenar and hypothenar eminences began. The lumbricales and the interossei also became involved. There was also weakness of muscles of the forearm and arm, the muscles showing a quantitative electric change while the muscle and nerve formula on the opposite arm was normal. (There was also atrophy of the muscles of the scapulo-humeral group.) All muscles showed quantitative electric change.

Along with the atrophy of the muscles of the hand there developed muscular twitchings which extended all over the body, with the exception of the anterior aspect of the chest, and were most severe in the atrophied muscles. This tremor seemed to comprehend in its range both fibrillary tremors as well as movements of the whole muscular mass. I have never seen the same kind of tremor save in one indisputable case of progressive muscular atrophy of the subacute type. I was quite apprehensive lest these tremors might mark the beginning of progressive muscular atrophy with a subacute onset. Charcot says that progressive muscular atrophy may follow infantile spinal paralysis, and by analogy one can see no reason why it might not follow spinal paralysis in the adult.

The disease increased steadily until October, 1898, when its progress seemed to undergo an arrest.

Of course a case of this sort barely serves to suggest the difficulties of diagnosis that may arise between poliomyelitis and polyneuritis. The closest students of the subject still hold widely varying opinions as to the relations of the two diseases. Erb considers all cases of amyotrophic paralysis as depending on an alteration of the ganglionic cells of the anterior horns of the cord; the multiple neuritis observed in some cases he considers as much a trophic trouble as the concomitant muscular atrophy. Sometimes the primordial lesion of the gray substance is dynamic only, and then histologic examination does not show any modification of structure save in the nerves themselves.

For Strumpel, polyneuritis is a distinct morbid entity with an individuality all its own, which one may set over against poliomyelitis as one contrasts diseases which differ by their nature, cause, and the seat of the lesion, having nothing in common save a great analogy of symptoms.

Gowers inclines to Strumpel's point of view and be-