A Note On Acute Ascending Paralysis

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PART:

I would like to focus attention on a rather rare and generally unknown illness which merits inclusion among the most remarkable afflictions in pathology.

In the "progressive diffuse" paralyses, the initially restricted area of paralysis gradually enlarges from its origin. This proximal propagation may be stepwise and orderly, as in the extenso-progressve ascending paralysis (ascending or centripetal paralysis), or intermittent and random (extenso-progressve intermittent paralysis). In the former, which is of some importance, the symptoms start in the distal extremities, progress proximally and cephalad, and become more intense with each area affected. The symptoms become generalized, producing a generalized paralysis which is quite distinct from syphilis.

I do not intend to present a description of this progressive diffuse paralysis (well described by Oliver of Angers, and Sandra) which characterizes many illnesses which have already been studied. However, it should be noted that, although almost always slowly evolving, it may also progress with extreme rapidity and be serious or even fatal. I would like to distinguish this latter variety by the name acute ascending paralysis or centripetal paralysis. This acute ascending paralysis shows features of malignant or pernicious illness by its insidious, ever worsening progression by the initial poorly defined symptoms and yet over-

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afebrile and had minimal pain. These symptoms persisted, waxing and waning without ever becoming unbearable.

On March 16, the patient suddenly felt an intense chill and pleuritic pain while working, began to cough up sputum, and became febrile. He was diagnosed as having "pulmonary congestion" and was bled three times, following which emetics and topical agents to induce epidermal swelling [topical applications of cantharide which induced epidermal blebs were felt to be of diagnostic or therapeutic value] were applied to the left posterior thorax and the patient was instructed to restrict his dietary intake. He took no nourishment for eighteen days, following which he was allowed some bouillon. He slowly recovered and returned to work on May 9, still very weak and barely eating. His strength, far from improving, continued to diminish, and on May 15 he stopped working again. Feeling weaker, he returned and was admitted to the hospital on June 1.

From May 11 or 12, he felt formications in the tips of his fingers and toes. However, these sensations were minimal and he paid little attention to them. Although generally weak, he remained in control of all movements. His limbs functioned normally with the exception of the unaccustomed weakness that necessitated undue exertion. On June 1, he walked from Boulogne-sur-Seine to the Hospital Beaujon without difficulty. At that time he complained only of generalized weakness, and from his general appearance his complaints seemed somewhat exaggerated and difficult to believe to M. Gubler (who was suspicious of the story).

On June 13, the patient noted that his knees gave way frequently when he walked. On the following day, these flexions became more frequent, his feet seemed heavy and difficult to raise, as if glued to the ground. Several days before, the formications had involved his feet and had begun to gradually involve his legs, thighs, and arms. The sensations seemed to be moving upwards, as a band around the extremity. As they progressed, the caudal areas were left numb as if by cold.

During the following days, walking became more and more difficult. He could no longer raise his legs, their movements now being slow and associated with dragging of the feet. During morning rounds on June 17, Grellier claimed he could no longer walk or hold himself upright. He stood up, held by two persons, who had to support him to prevent his legs from collapsing. When he tried to walk, his movements were slow and weak, but not abrupt or disorganized. He dragged his feet across the floor, being unable to lift them. When he lay down, he could not raise either of the lower extremities off the mattress and attempts to flex his thighs were painful. He could not turn onto his side as he could not move his legs across each other despite being able to rotate his trunk. Movements of the upper extremity were minimally involved. His grasp was sufficient to allow him to cling to helpers who might have aban-

donned him while walking; but he complained that his fingers were rigid and stiff, as if bound and compressed by twine—"all tied up." This sensation, apparent for several days, was also noted when he wiggled his toes, but the sensation only appeared during spontaneous movements and not with reflex movements. His joints were normal and the paralyzed areas were supple. In the upper extremity only the elevators of the arm were weakened. This deficit, worse on the right, made it impossible for him to raise and maintain his arm in a horizontal position.

There was no fever, no extremity or vertebral pain, headache, contracture or convulsive movements. Reflexes could not be elicited and sensation was only minimally impaired on the soles of the feet. Intellectual functions were intact. He had experienced diminished appetite without gastrointestinal tract difficulties. His general state was such that M. Gubler still feared that he was being deceived.

By June 20, the motor paralysis had increased in the previously involved areas and had affected formerly intact locations. The lower limbs were now almost completely paralyzed and the upper extremities could no longer be used, although their strength was not completely lost. The continually advancing formications were now felt around the thorax and at the base of the neck and Grellier now complained of slight respiratory difficulty—a sort of distressful constriction of the thorax. He spoke of an epigastric "barrier" which obstructed inspiration. On examination, his rib cage expanded as a passive wall, the individual intercostal movements were very limited, the epigastrium descended slightly during inspiration and rose during expiration. This was best seen when the patient lay on his back, but scarcely visible when he was seated. When Grellier made the effort to control it, his epigastrium moved normally during respiration. Slight dyspnœa was present, his speech was halting in quality, and his cough was feeble. He complained that his tongue and jaw felt heavy and less mobile. Movements of eating were more difficult, food seemed harder to chew, and some dysphagia was present. His general condition had not changed from the preceding days.

On June 21, his clinical symptoms were more pronounced than previously. He continually sweated, appeared cachectic, and had a cough that produced a thick mucus. His pulse was rapid (85–90 beats per min.) but was thready and easily obliterated. His temperature was elevated, but was diminished in the distal portions of the limbs. Heart sounds were normal and his veins were not prominent. His appetite was fair, tongue normal (small, oval, and red) and gastrointestinal functions were undisturbed, with stool and urine unremarkable. His motor paralysis had now become generalized, but to varying degrees, and was most pronounced caudally. The distribution of the paralysis may be seen from the following details.

*Lower extremities* could only be minimally moved but not at all against
gravity. Only the triceps surae (crural) contracted appreciably. There were no movements of the toes and feet and no contractions of the leg muscles with maximal effort. If his thigh was elevated, he could maintain his leg in extension for a short while. If the least force was exerted on this extended leg, it gave way, flexed and fell like a dead weight. With his leg supported (to lighten the weight of the limb) he was unable to flex his thigh. Adduction, abduction, extension, and rotation were completely absent. With effort, he could produce small adductor contractions which could be seen and felt by the examiner; but the glutei were silent. In summary, the paralysis was less complete in the anterior and internal thigh muscles than in those innervated by the sciatic nerve.

Upper extremity movements were limited in their range. Arm abduction and elevation were impossible. When his arm was placed at right angles to his shoulder, it fell without any opposition on his part. Contractions of the deltoid could be felt, but these were insufficient. Internal and external rotation of the arm was weak and incomplete. The remainder of the limb, except for distal areas, showed less complete paralysis. Finger abduction consisted only of a few oscillations; adduction and opposition of the thumb was almost nonexistent. His fingers were held semiflexed. Further flexion was slight and he could not grasp or hold objects placed in his hand. Extension of the fingers and wrists was impossible. Lateral and rotatory movements of the hands were restricted. On the left he could flex and extend the forearm—motions which could be impeded by the least resistance. The right forearm motions were weaker and more limited than the left.

Trunk, etc. Sitting was impossible. He fell forward or to the sides when not supported. Voluntary abdominal muscle contractions were feeble. The thoracic walls moved together in inspiration as a result of the isolated action of the cervical muscles. The widths of the intercostal spaces did not change in a physiologic manner. The patient could move his shoulder forward, backward, or upward provided there was no resistance on my part. The trapezius and pectoral muscles contracted well. When resisted minimally these muscles no longer moved. The serratus magnus did not move with any motion or with deep inspiration, and the scapula maintained its normal position. When he was placed in a sitting position, his head fell forward or to the side. He could raise it up again with effort.

The diaphragm was presumably paralyzed, as inspiration, especially if deep, caused the epigastrium to hollow out to raise again with expiration. He could bear down only for a short time, following which he became short of breath and exhausted.

As a result of the paralysis, the thorax filled incompletely and respiration was severely compromised by the defective diaphragmatic movements. Although the sternomastoid and scalene muscles contr-
Although naturally limited, the patient's intelligence had not deteriorated. He appeared more lucid, sharper, more appropriate, perhaps owing to lessening timidity. Moreover, his general condition was not alarming. His expression was calm, and at first sight, his dyspnea was scarcely noticeable. He was apprehensive about his state and at times seemed to have sad presentiments.

Treatment consisted of limb massages with volatile liniments (terebenthine, quinine) and electrical stimulation along with substantial nourishment (cutlets and Bordeaux wine). These approaches were in use for several days.

During the course of the day his symptoms became more aggravated. Towards 4 o'clock, dyspnea was extreme, his speech halting and weak, his face and neck became slightly cyanotic and were covered with a cold sweat. He complained of breathing difficulties and of a constricting sensation in the larynx.

At 5 o'clock, upon the insistence of the ward nurse, he decided to eat, but could not swallow. He then asked to sit up to facilitate respiration and swallowing, but after several moments, he became weak, asked for help, grew pale, and suddenly died, eight full days after the start of the paralysis.

Autopsy was performed on the evening of June 23 (good weather, 17°C) forty hours after death. Postmortal rigidity was marked, but the body was well-preserved, appearing as in life.

The skull and vertebral column were opened with care. The sinuses were engorged with blood as were the cortical and spinal meningeal veins, but there was no evidence of subarachnoid or subpial blood. There was very light serous fluid found, but no deposits or inflammation.

The cerebral convolutions and cerebellar lobes were normal in color and consistency. Some finely arborized plaques, which did not enter the grey matter, were seen on the mesial surface of the left hemisphere. At these points, as on the entire surface of the cerebral mass, the meninges were easily removed without tearing the subjacent tissue.

The most minute examination revealed no abnormality of the brain stem, cerebellum, or brain proper. The white and grey matter appeared normal. Petechiae, congestion, induration, softening or anemic change of the neuropil were not found. There was no trace of recent or old blood in the parenchyma or in the ventricles.

The spinal cord, likewise, was intact in its entirety and in all its elements. The nerve origins were well-formed. Several sections of the cord, taken at different levels, were submitted for microscopic examination by Messrs. Bourguignon, Gubler, Ch. Robin, and myself. These different examinations produced identical conclusions: the grey and white matter were entirely normal.

The muscles were deep red. Several microscopic specimens of the soleus appeared normal in character.

Very solid adhesions were present in the right thoracic cavity. The right lung appeared purplish wine-red and was engorged like a spleen throughout. Its tissue was harder and more friable than normal. However, several sections thrown into water floated. The largely intact left lung was similar to the right. In several places, the tissue was even more strongly discolorated and appeared infiltrated with black pigment. Several calcific areas were seen in both lungs but nothing suggested fresh or softened tuberculous granulomas. The other organs were not examined.

In summary, we have presented a pitiful man of forty-three years of age, weakened by a series of acute illnesses, blood loss, and a prolonged restricted diet. During a slow and incomplete convalescence, he experienced gradually increasing generalized weakness without any sign of paralysis. Formicaclonations were initially limited to the toes and fingers and were not associated with impairment of movement or ambulation.

After a prodomal period of approximately six weeks characterized by these phenomena, the extremity paresthesias crept upwards, leaving numbness and paralysis behind. The latter, which especially affected ambulation, moved rapidly from the feet to the rest of the lower extremities, to the upper extremities, to the trunk, to the respiratory muscles, to the tongue, etc. Movement was most impaired in the limbs, but micturition and defecation remained intact until the end. Muscle tone and irritability as well as nerve excitability were not altered. There were no contractures, convulsions, tremors, fibrillations, or pathologic muscle movements. The patient did not complain of pain in the extremities, vertebrae, or head, and tenderness was not present. Fever was absent and intelligence was unaffected.

Terminally, respiration became more and more difficult, signs of asphyxia appeared and the patient died suddenly, eight days after the onset of paralytic symptoms.

Autopsy did not reveal any lesion of the nervous system. We found only traces of pleurisy and recent pneumonia.

PART II

Thus, there exists a rapidly generalized paralysis which terminates with death in a few days. Autopsy examination provides no clue as to its origin, and its onset offers no hint of the proximity of its fatal outcome. On the contrary, observers have noted the contrast between the outcome of the disease and its benign initial appearance. This initial appearance is so benign that the observer often thinks that the patient has voluntarily exaggerated, or even simulated, his illness, so that even
eight hours before death, there is little concern. The paralysis extends relentlessly from the lower to the upper extremities and from the limbs to the trunk, producing a quiet asphyxia when the respiratory muscles become involved. This insidious and rapid progression to an unexpected and fatal outcome suggests that the illness should be considered a malignant or pernicious disease.

I have observed four cases which were similar to the case described, and have uncovered five more from the literature, bringing the total to ten. I will limit myself to a brief presentation of the principal features of the group, rather than point out the differences in pattern of evolution, muscular irritability, etc., among the cases.

In this type of paralysis, sensation and movement can be equally involved. However, in general, movement is especially affected by gradual diminution of muscle strength and flaccidity of the limbs and loss of reflexes without tremor, contraction, localized or generalized convulsions, or pathologic muscle reflexes. In almost all cases, micturition and defecation remained intact, and there is no evidence of central neurologic symptoms—i.e., no pain along the vertebral column—occurring either spontaneously or as a result of compression, and there is no headache or delirium. Until the very end, intellectual faculties are completely preserved.

As in our patient, paralytic signs can be preceded by a slight generalized weakness, formations, and even fleeting cramps, or the onset can be sudden and unexpected. In each case, the paralysis rapidly advances from the lower to the upper extremities, with a constant tendency to become generalized. The first deficits always appear in the periphery of the limbs, most often in the lower extremities. From there they follow a progressively ascending course and invade the muscular system in an almost constant sequence: first, the muscles of the toes and feet, then the posterior muscles of the thigh and pelvis, and lastly, the anterior and medial muscles of the thigh; second, the muscles of the fingers of the hand and of the proximal arm at the shoulder, and the muscles between the forearm and the upper arm; third, the trunk muscles; fourth, the respiratory muscles, tongue, pharynx, and esophagus. The paralysis is then generalized, but is not as complete as it is in the extremities.

This phase of the illness is more or less rapid, occurring over eight days in M. Gubler's patient, and fifteen days in another case. More often, the process evolves over two or three days and sometimes over several hours.

When the paralysis has reached peak intensity, death by asphyxia is imminent. However, in eight of the ten cases, this fatal outcome was avoided by judicious intervention or a spontaneous arrest in the progression of the disease. Two of the ten cases died during this period.

An illness to which a fifth of the patients succumbs is without doubt a grave process. It should be understood that in spite of the relatively favorable outcome of this illness, the danger is great and the prognosis remains uncertain. The patient remains in evident peril while the paralytic symptoms are rapidly ascending and his life may be endangered when his respiratory muscles are involved. As asphyxia has been the most common cause of death, it is impossible to predict the extent of involvement and effectiveness of therapy in any single case.

The pattern of resolution of the paralysis reverses that of the development of the paralysis. The last invaded upper extremities are the first to recover; the area of resolution progresses from proximal to distal. Some patients may remit rapidly while others may enter a chronic state, from which they slowly improve. One patient who showed well-defined, frequent fluctuations between improvement and worsening over several months died during a seizure. A woman, reported by M. Cavare (of Toulouse), developed steady waves of paralysis—the illness ran its course in several hours.

I believe it is useless to dwell on the diagnostic features of acute ascending paralysis as no other disease state presents a similar constellation of signs.

The paucity of facts makes it impossible to study the etiology of this process, but the circumstances in which it develops can be noted.

In two instances, it developed during convalescence from an acute illness (our case description is one of these) which must play some role in the pathogenesis of the paralysis. The second subject developed the illness over five days while convalescing from a long bout of typhoid fever. The paralysis remitted spontaneously, and less than two weeks later he was entirely well.

In two instances, the acute ascending paralysis occurred against a background of menstrual difficulties. In one case, menses were retarded by a cold; leeches were applied to the vulva, and recovery was complete within seven days. In another case, suppression of menses during a moral crisis was followed by multiple neurologic symptoms and an acute ascending paralysis. The paralysis became generalized and life-threatening within three days. Remarkably, inhalation of chloroform and opium produced a sudden and marked improvement. After several remissions, the illness became chronic, resulting in death during an unexpected paroxysm.

In two additional cases, exposure to cold was associated with an acute and life-threatening illness which in one patient arrested spontaneously and in the other slowly advanced.

Paralysis leading to death in two days occurred in a woman convalescing from childbirth. Treatment consisted of bleeding and purges.

One patient presented during a syphilitic diathesis. The paralytic
signs progressed more slowly and were less alarming than in the other cases, and rapidly disappeared following antisyphilitic treatment.

In the two remaining cases, there was no information as to the cause of the events. In one woman, the paralysis developed as steady paroxysms which responded to quinine sulfate (as I noted above).

As can be seen, the causes or circumstances under which the illness occurs are variable. The influences I have mentioned are remote causes. The direct cause of the disturbances remains to be determined.

The two autopsies performed prior to this writing have only furnished negative anatomic-pathologic data. However, Oliver (of Angers), who had a good knowledge of acute ascending paralysis, believed it was due to vascular congestion of the spinal cord. The symptoms do not suggest this is the case. It is known that the accumulation of a certain amount of blood in the veins of the nervous system, especially following asphyxia as in his patients, does not establish the nature of a disease. Indeed, this illness ought to be placed among the essential paralyses, i.e. those without an apparent lesion of the nervous system. I feel these facts are noteworthy. In this simple note, I would like to avoid considering the mode of production of ascending paralysis in general and of the acute form in particular.

On a syndrome of radiculoneuritis with hyperalbuminosis of the cerebrospinal fluid without a cellular reaction.

Remarks on the clinical characteristics and tracings of the tendon reflexes

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We would like to draw attention to a clinical syndrome observed in two patients, that is characterized by motor difficulties, loss of the deep tendon reflexes with preservation of the cutaneous reflexes, paresthesias with slight impairment of objective sensation, muscle tenderness, slight alterations in nerve conduction and electromyographic patterns, and a remarkable increase in cerebrospinal fluid albumin in the absence of a cellular reaction (albumino-cytologic dissociation). This ostensibly infectious or toxic process appears to simultaneously involve nerve roots, peripheral nerves and muscles. It is distinct from the simple radiculopathies, pure polyneuropathies, and from the polymyositides. Experimental data derived from tracings of the latency and speed of the reflex response and muscular contraction indicate that the entire peripheral neuromuscular motor apparatus is involved. We particularly emphasize the increased cerebrospinal fluid albumin content without

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Cellular reaction, an observation which has not previously been reported.

Case 1

D..., a twenty-five-year-old soldier of the ...th cavalry, entered the Neurological Center of the Sixth Army on August 20, 1916, because of weakness of the upper and lower limbs. On about July 25, he first noted formation in the feet and weakness of the lower extremities, the latter forcing him to halt every 200 to 300 meters. During the following days, the formation spread to his upper extremities and lower face. Muscular weakness appeared in the upper limbs.

These signs and symptoms developed without apparent associated cause. He had had no recent infections (including sore throat and gastrointestinal disturbance) or fatigue. His past medical history was benign; he denied any syphilis or excessive alcohol intake.

When examined on August 25, he exhibited diffusely diminished muscle strength in the upper and lower limbs but without a total paralysis. This marked distal extremity weakness was especially obvious in the flexors and extensors of the toe, ankle, finger, and wrist.

The trunk muscles were weak. He could not rise from a lying position without using his hands for support. He was able to walk a few steps; some instability of upright posture was noted, and he was unable to stand on one foot.

There was no weakness of the facial muscles.

Electrophysiologic studies in the upper extremities showed normal faradic excitability and brisk jerk responses to galvanic excitation of all muscles. Polar inversion was not seen. Slight hypoelectricity of the common extensor of the fingers was detected. Faradic excitability in the lower extremities was slightly diminished, and galvanic excitability was diminished in the trunk of the sciatic and internal popliteal nerves, as well as in the semitendinosus muscle and the extensors of the fingers. Responses were sometimes slightly slowed. Polar inversion was seen in the external gastrocnemius but the degeneration reaction was very incomplete.

The deep tendon reflexes at the knee, ankle, and medial plantar surface, as tested with a percussion hammer, were abolished, as were those of the antibrachial radio- and cubito-pronator and olecranon areas.

The cutaneous plantar reflex induced frank flexion of the toes with distant contraction of the tensor fascia/ata. The cremasteric and cutaneous abdominal reflexes were normal. There were no defensive responses to pinching the sole of the foot or hyperflexion of the toes.

Neuromuscular excitability as elicited by the reflex hammer was preserved.

The patient constantly complained of formations just above the malleoli and just above the wrists. There was no deficit of objective sensation, except possibly for slightly hypesthetic touch, pain, and temperature sensation in the feet and hands. The muscles of the upper and lower limbs were painful to pressure.

The pupils, which were equal, reacted to light and to accommodation. There were sphincter difficulties. No fever, respiratory, or gastrointestinal signs were found, and the pulse was normal.

The urine, examined in the Army Bacteriologic and Chemical Laboratory, contained no sugar, albumin, or indoxyl, the chemical elements being in their normal proportions.

Lumbar puncture showed a clear cerebrospinal fluid under normal pressure; but with increased albumin (2.5 grams of albumin per liter) and without leukocytic reaction (two to four lymphocytes per field). The blood Wassermann reaction was negative. Pharyngeal and nasal mucosal cultures showed no diphtheritic bacilli.

Therapy consisted of absolute bed-rest, liniment massage of the upper and lower extremities, strychnine injections and oral salicylate of soda compounds.

On August 27, the lower extremity formations had diminished. On September 2, the muscular weakness had ameliorated and the foot formations had disappeared, although still present in the hands. The tendon reflexes were still absent. Another lumbar puncture showed, as previously, a marked increase in cerebrospinal fluid albumin without appreciable leukocyte reaction.

On September 19, the motor difficulties had very much improved. The patient was able to walk for an hour and stand on one foot. The paresthesias had completely disappeared in the inferior extremities, but persisted, although attenuated, in the hands. Tendon reflexes and withdrawal responses were still absent but cutaneous reflexes were normal. Neuromuscular excitability in the upper and lower limbs and in the face, elicited by reflex hammer, appeared normal.

The patient continued to improve and was sent to a convalescent center on September 30.

Case 2

D..., a thirty-five-year-old infantry soldier, was admitted to the Neurologic Center of the Sixth Army on September 5, 1916 with lower limb motor difficulties, which appeared under the following circumstances.

On August 28, after a march of fifteen kilometers, he experienced unusual fatigue, headache, and erratic pains in the upper and lower extremities. He lay down, but could not sleep, and shivered part of the
night. The next morning, he walked with great difficulty to report to sick call, and was exempted from duty for the next four days. The weakness began in the lower extremities and subsequently involved the upper extremities. On the fourth day he decided to set out with his comrades at about 5:00 A.M. He dressed for the march, but fell backwards with his knapsack and could not get up again. He was taken to a first-aid station and then evacuated to the Army Neurological Center. These complaints developed without apparent cause: he had had no recent infectious illnesses, no symptoms of gastrointestinal intoxication or other disturbance. He very strongly denied having had syphilis.

On September 5, we found the patient able to make, with difficulty, little movements of flexion and extension of the toes, flexion of the leg on the thigh, and the thigh on the pelvis. The same difficulty was present with regard to movements of the upper extremities, where the difficulty was much more prominent peripherally. He held his head rotated to the left, and experienced difficulty in turning it to the right. He could open and close his mouth, but slowly and incompletely.

Electrophysiologic examination showed slight faradic hyperexcitability of the nerves and muscles. Excitability was slightly enhanced to galvanic stimulation, especially for the nerves of the upper extremity. There was no reaction of degeneration.

The knee jerks were very difficult to elicit because of increased muscular tone, but they seemed to be present. Ankle jerks and medial plantar responses could not be determined because of hyperreflexia and the impossibility of provoking a complete muscular movement. The cutaneous plantar reflexes showed frank flexion of the toes; the cremasteric and abdominal cutaneous reflexes were normal. No withdrawal response was seen, either by pinching the dorsum of the foot or hyperflexing the toes; but the patient perceived the sensations produced by these stimuli.

Neuromuscular excitability (tested with the reflex hammer) was preserved.

The patient complained of formication in the extremities. There was no impairment of objective sensation except possible touch, pain, and temperature hypesthesia in the feet and the hands. The muscles of the calf and forearm were tender on compression. The pupils were equal, reacted to light and to accommodation. The patient voided spontaneously. He felt the need to micturate but was unaware of urination. Fever, Kernig's sign, nausea or vomiting were not present. Urinalysis in the Army Bacteriologic and Chemical Laboratory showed no sugar, albumin, or indoxyl, and the chemical analysis was normal.

It should be noted that a cutaneous eruption had appeared three or four days earlier. This was erythematous and papulomacular in character and was localized principally to the upper part of the thorax and the lower part of the abdomen. In addition to the areas we have mentioned, eruptive lesions were disseminated over the rest of the thorax and the abdomen but not on the upper or lower extremities.

Lumbar puncture showed clear cerebrospinal fluid, not under increased pressure, with increased albumin content (more than 0.85 grams of albumin according to the rachialbuminometer of Sicard) but without significant leukocyte reaction (three to four lymphocytes per field).

The symptoms found on the first exam remitted slightly. On September 20, distal extremity weakness was still present, all tendon reflexes except the left antibrachial were absent, the cutaneous reflexes were preserved, and muscular tenderness to pressure, as well as paresthesias in the extremities with slight hypesthesia persisted. Intermittent little myoclonic jerks were seen in the calf and thigh muscles. Another lumbar puncture revealed the same abnormalities seen on the antecedent examination, a clear fluid under normal pressure, containing markedly increased albumin without leukocytes (three to four lymphocytes per field).

The patient was evacuated to the rear on October 1. . . .

These two cases are quite similar. Each developed a clinical syndrome without apparent cause, characterized by motor difficulties of all the muscles of the upper and lower extremities, but predominantly distally, by loss of deep tendon reflexes with preservation of cutaneous reflexes, by paresthesias with slight loss of objective sensation, by tenderness of the muscle bodies to pressure, and by minimal alterations in the electrophysiologic reactions of the nerves and muscles, by a rather special abnormality of the cerebrospinal fluid characterized by increased cerebrospinal fluid albumin concentration without cellular reaction.

The significant rise in cerebrospinal fluid albumin concentration without cellular reaction seems to be an important peculiarity. This albumino-cytologic dissociation (Sicard and Foix) is seen most often in certain cases of medullary compression, in Pott's disease, and in certain instances of neurosyphilis, but it has not been reported, we believe, in the pure radiculopathies or polyneuropathies. Notably, our second case developed muscle hypertonia in addition to paralysis. With the patient at rest, the muscle consistency was clearly greater than that of a healthy resting individual. The limbs maintained their full range of passive motion, but voluntary movements were limited and made with a certain stiffness and slowness. Tendon reflexes were difficult to elicit because the usual muscle responses were hindered by the continued contractions of their antagonists. In spite of these signs, also frequently found in meningitis, the patient could be seated, keeping his upper extremities almost completely extended. The slight knee flexion accompanying this maneuver could be overcome by insignificant pressure. Kernig's sign was not present in our patient: when the
lower limbs were raised to form an almost right angle with the trunk they flexed as would those of a normal subject. This state of hypertonicity has nothing in common with meningitis, but relates rather to a special state of muscular contractility which appears to depend on a peripheral nerve lesion. We have already stressed the fact that hypertonicity can be encountered in the course of certain peripheral neuritides and of incomplete nerve injuries, and specified on that occasion that contractures frequently observed in the course of certain facial palsies are not an exception among peripheral nerve lesions as had been classically believed.

The syndrome observed in our two patients is due to simultaneous involvement: of the nerve roots, peripheral nerves and muscles. The considerable increase in cerebrospinal fluid albumin indicates meningeal involvement, while the pattern of extremity muscle paralysis and the muscle pain in response to compression indicate the involvement of nerve and muscle. Besides, it seems to us that neurologists become too precise when they try to completely separate the polyneuritides and polymyositis. In a greater number of cases of infectious or toxic polyneuropathy, the intramuscular nerve endings and the muscle fibers themselves can be involved. In reality, the pathologic process is much more a polyneuromyositis than a pure polyneuritis.

In the case of our first patient, experimental research using myographic tracings enabled us to bring out certain new features in the study of reflexes and muscular contractility. Such tracings can give us important data for the interpretation of symptoms and lesions.

In the first case, although the tendon reflexes (by clinical examination) appeared to have been lost throughout the course of the illness the tracing of the distension of the quadriceps femoris and gastrocnemius muscles following percussion of the tendons or muscle bodies displayed interesting features. Thus, from the onset of the illness, the knee jerk showed a contraction following a mechanical blow (Fig. 1). This contraction, notably weaker than that obtained in a normal subject, appeared after a latency of 0.056 seconds, but was not followed by a second contraction of greater amplitude and duration that represents the true "reflex" response in a normal tracing. Scarce 0.152 seconds after the beginning of excitation, we noted a very slight upward deflection of the curve, indicating the vestige of the reflex contraction. The knee jerk was thus almost completely reduced to an idio-muscular contraction up to the end of the illness. During this period, percussion of the quadriceps provoked a good muscular contraction with a latency of 0.051 seconds followed by a second contraction having all the characteristics of a response of reflex origin and appearing 0.150 seconds after the onset of excitation. The muscle, which only weakly and incompletely responded to mechanical excitation of its tendon (transmitted by propagation to the muscular fibers), demonstrated, when it was struck directly, a double

Figure 1. R.R.R. Myogram of the quadriceps femoris during patellar reflex. The signal of Desprez indicates the percutaneous stimulus and indicates the time in fractions of 1,000 second. I.I. The same tracing for direct percussion of the quadriceps femoris, recorded on August 21, 1916. Of note is the almost total absence of "reflex" contraction following patellar percussion in comparison to its clear preservation in response to direct percussion.

Figure 2. A. Myogram of the internal gastrocnemius during the Achilles reflex. M. The same during the medial-plantar reflex, recorded on August 21, 1916. The first evaluation in record A is a mechanical jerk, the second is a "muscular" contraction which is absent during the Achilles reflex but weakly visible during the medial-plantar reflex.

Figure 3. A. Myogram of the external gastrocnemius during the Achilles reflex, recorded September 15, 1916. The reflex shows three characteristic elevations. The "muscular" contraction and especially the "reflex" contraction are weaker than normal.
contraction which was almost normal. The muscle seems to be mechanically hypoxicitable except to sudden blows to the muscle body itself.

The ankle jerk was similarly altered and reduced, leaving only its mechanical response. The latter (Fig. 2), of very low amplitude, appeared after an extremely long latent period of around 0.110 seconds. It was not followed by reflex contraction. In contrast to the knee jerk, these changes partially resolved and already on September 5 (Fig. 3), we detected a higher-amplitude muscular jerk, more brisk, more rapid (0.035 seconds), followed by a second response (recognizable as a reflex) appearing after a latency of 0.140 seconds. The neuromuscular response of the gastrocnemius followed a similar course and gradually resumed a more normal shape.

It is interesting to note that, at the start of the illness, when percussion of the Achilles and gastrocnemius tendons elicited only a muscular response, study of the medial plantar reflex, even at this time, showed a second contraction which was weak, but clearly a reflex, with a latency of 0.144 seconds.

In summary, while the clinical exam permitted only the demonstration that tendon reflexes were lost, detailed analysis of the myographic curves indicated which elements of the reflex were altered, and led us to the following observations: First, the reflex segment of the myographic curve is either absent, or when it persists, extremely reduced in amplitude with a markedly slow inscription. Its considerable latency, almost double the normal, demonstrates the profound and predominant alteration of nerve conduction of the central part of the reflex. Furthermore, the muscular response appears equally modified, diminished in height, and slowed and delayed in appearance, which leads one to think that the muscular element has been equally affected by the toxic process. Finally, comparison of the curves obtained after percussion of the patella and Achilles tendons shows a different evolution for the two reflexes. While the first had been rapidly abolished and had not reappeared by the patient's discharge from hospital, the second, which seemed clinically absent, had begun to approach normal by myography. This emphasizes the fact that the graphic method is a more precise indicator of the state of the tendon reflex than is the reflex hammer.

The pathogenesis of this radiculoneuropathic syndrome cannot be precisely defined. Although an infection or toxic insult should be considered, we have found no supporting evidence for either. Judged by the evolution of the disease in our two patients, the prognosis does not appear to be grave, as the first patient was almost cured and the second on the road to recovery when discharged from the army.