stances in which, in syphilitic patients, there exists some form of medullary sclerosis, the most energetic and the most violent treatment will not favorably modify the march of the disease. However, the fact of syphilis coexisting with tabes dorsalis, always gives a hope and a chance for your patient, which you ought to improve.

There are neuropathies which sometimes present ataxic symptoms; these false ataxies are often completely cured by hydrotherapy, or a course of treatment at the thermal springs.

The first condition of treatment of sclerosis of the posterior columns, is to avoid all circumstances which tend to provoke congestion of the cord, a congestion which almost always precedes the hyperplasia of the neurolgia. You should then prohibit, in the case of these patients, prolonged muscular fatigue (a fatigue which is the more easily induced the more voluntary force the patient is obliged to expend in his movements); sexual intercourse ought also to be forbidden. You should also have recourse to energetic revulsion, and in particular to punctiform cauterizations along the vertebral column, and this especially when signs of irritation or congestion of the cord appear. You can also use in the periods of remission nitrate of silver or phosphorus, following, in the case of phosphorus, the rules which I have laid down. You will almost always obtain with this phosphorus medication an amelioration, more perceptible to the patient than to yourselves; he will tell you that he feels stronger, and that he has a better command of his limbs, at the same time that an examination does not disclose any real progress. I have never observed genital excitation following the administration of phosphorus, and I very much doubt the aphrodisiac properties which have been ascribed to this medicament.

The lightning pains are among the most formidable complications of the disease; here our "sheet-anchor" is hypodermic morphia, despite the fact that ataxic patients are very likely to become morphomaniacs (a great many at the present day are such). Sometimes extremes of temperature diminish notably

at the end of a certain time, which is variable, destruction of the cartilages and bony tissue takes place.

The reflex excitability of the cord is sometimes considerable in the ataxic patient, and the contact of a foreign body, especially of a cold object, on the skin produces reflex movements more or less extensive.

Forced flexion of the foot on the leg, produces the phenomenon of reflex trepidation, so also does percussion over the tendo-achillis or the patella tendon, the latter giving the characteristic knee jerk. The "patellar reflex" is, however, often wanting.

The trophic troubles observed in the course of the second period, consist in cutaneous eruptions, in emaciation of the limbs, atrophy of the muscles, and a rarefaction of the bony tissues which results often in fractures.

Intellectual troubles are somewhat rare, at the same time there may be delirium more or less temporary, sometimes dementia.

The motor incoördination makes progress; by and by the patient can no longer walk and is obliged to keep his bed.

Every movement becomes impossible, and the paralysis invades progressively the upper limbs, sometimes even contracture of the affected members is noted (third period).

Purulent cystitis, or incontinence of urine, or of fecal matters soon results in the formation of eschars on the sacrum, or in other regions and the patient succumbs.

this pain; I have lately had under my care a French engineer, who, when constructing railroads in Russia, could succeed in calming the *douleurs fulgurantes* of his disease only by surrounding his feet and legs with ice. In some patients heat has the same effect. In these cases Chapman's rubber bags for pounded ice or hot water will be useful. Nerve-stretching has been proposed for these *douleurs fulgurantes*.[1] This elongation of the sciatic nerve, practiced for the first time in Germany by Langenbuch, in 1879, in France, by Débove and Gillette, in 1880, and since repeated by Esmarch, Erlenmeyer, Sury-Bienz, Hirschfelder, Rzehaczek, has not given any definite results, and though causing a temporary amelioration, it is often accompanied by trophic disturbances of great intensity. Balneotherapy and thermal treatment have an important *rôle* in the therapeutics of ataxia. For my part, I have abandoned hydrotherapy in the treatment of medullary sclerosis. I believe that cold douches can only augment or provoke spinal congestions, and I am happy to see that most of my colleagues of the Society of Hydrology are of the same opinion.[2]

If hydrotherapy has rather inconveniences than advantages in the treatment of ataxia, it is not so with thermal treatment. Three stations claim preëminence, Neris, Balaruc, and Lamalou; the thermal establishments of the latter seem to be most efficacious in chronic myelitis.

Finally, to these balneotherapic means we must add electricity. Onimus and Rosenthal have insisted on the curative action of continuous descending currents in the affections of the spinal cord. It is a means which you ought always to employ, without expecting always to obtain much amelioration.

On the other hand, electricity has a preponderating *rôle* in the atrophic paralysis of infants, which, as the recent researches of Roger and Damaschino and of Laborde have shown, is to be attributed to central myelitis affecting the anterior horns, and destroying the large multipolar cells of that region.[3] These

[1] The first case of nerve stretching for locomotor ataxia was practiced in 1879, by Langenbuch; this operation was performed for the relief of the lightning pains, affecting chiefly the left sciatic nerve; this nerve was stretched on the 13th of September; the 25th of September the right sciatic was stretched and the two crural; the pain disappeared completely, but afterward, in consequence of new pains appearing in the arms, elongation of the median nerve was attempted. The patient, unfortunately, died from chloroform during the operation, January 6, 1880.

Since then the operation has been performed by various surgeons—Esmarch, Erlenmeyer, etc. In December, 1880, elongation of the sciatic for the *douleurs fulgurantes* of ataxia was performed by Gillette under the direction of Debove. Gillette twice performed nerve-stretching for ataxia in 1881, and other continental surgeons have practiced in numerous cases of ataxia this elongation of nerves and with variable results.

[2] Langenbuch, Berliner klin. Wochensch., No. 48, 1881. Esmarch, Deutsche med. Wochensch., 1880, No. 19. Erlenmeyer, Centrablatt für nerv. und psych., No. 21, 1881. Débove. Gaz. des Hôp., 11 Dec. 1880 Gillette, Union Med., No. 17, Jan. 1881. Sury-Bienz, Deutsch. med. Zeitung, 1881. Du traitement de l'ataxie locomotrice (Soc. d. hydrologie Med. Sèance du 4 November, 1878.

[3] Infantile atrophic paralysis comes on generally between the ages of one to three years. Cases are exceptional after ten years of age. The mode of invasion is sudden, being announced by an intense fever, with or without convulsions. From day to day you observe paralysis develop sometimes general, affecting the upper and lower extremities, sometimes

alterations have as a consequence the atrophy or destruction of important groups of muscles. Our only means of combating this disease are galvanism, gymnastics, and hydrotherapy, in a word, all these agencies which can excite nutrition in atrophied parts. These means have real efficacy only at the onset of the disease; unhappily the first periods often pass unnoticed, and it is not till the later periods of the malady that our help is demanded, and then little can be done.

The same treatment is applicable to a disease which resembles the atrophic paralysis of early childhood. I refer to progressive muscular atrophy, which in accordance with the pathological studies of the French school of medicine, and I refer particularly to the labors of Hayem, Vulpian, Troisiêr, Charcot, and Gombault, ought to be attributed, like the atrophic paralysis of early childhood, to chronic parenchymatous myelitis, localized in the anterior horns of gray sub-

three members; sometimes localized in a leg or an arm; rarely it affects the two lower limbs only. This paralysis is at first complete, absolute, with flaccidity of the limbs. It often takes on the paraplegic form, but is not accompanied with sensory disturbances or functional troubles on the part of the bladder and rectum.

Very rapidly, on the seventh or eighth day, Faradic contractility is lessened or abolished in a certain number of muscles, while galvanic contractility persists longer. The reflex movements are not always abolished from the first. From the second to the sixth month after the invasion, the paralysis abandons certain muscles, to localize itself in others; muscles of the leg such as the peronei muscles and the anterior tibial; notably the deltoid in the upper extremity. Then appear various special troubles: the atrophy, which according to Duchenne may already be very apparent at the end of one month, affects the muscles in which Faradic contractility is absent; the bony system suffers an arrest of development and the bones of the affected side may be shorter than the corresponding bones of the healthy side.

The side affected presents a remarkable lowering of temperature. The partial atrophies from which the patient suffers are followed by deformity of the limbs and determine vicious attitudes; and it is in this way that club foot often takes its origin especially *varus equinus*, *talipes valgus*, and sometimes a deformity to which Duchenne has called attention, a sort of *hollow-footed talipes*, in which the forepart of the foot is hollowed out by exaggeration of the plantar concavity.

In the upper limbs the most frequent deformity, sometimes only one observed, is flattening of the shoulder. You readily see the head of the humerus under the atrophied deltoid. In certain cases the arm hangs inert along the body, it is wasted, is much shorter than the other arm, the fingers are flexed on the hand which is itself flexed on the fore-arm.

The prognosis, as far as life is concerned, is favorable; from the point of view of function it is bad. The atrophy in fact is incurable and entails with it persistent infirmities.

The anatomical lesions of atrophic paralysis of infancy are primitive and secondary, the latter consisting in trophic troubles of muscles, bones, and blood vessels. The primitive lesions consist in myelitis of the anterior horns of the cord, starting, according to Charcot, in the ganglionic cells. According to Roger and Damaschino the point of departure is in the blood vessels and neuroglia. In two autopsies of infants that succumbed to the disease, the one two months, the other six months after the commencement of the disease, they noted lesions localized in one of the anterior gray horns, consisting in inflammatory red softening with vascular injection and the production of granular bodies.

According to Prof. Charcot, the nerve-cells are the first seat and origin of the irritative process, and there is produced an anterior acute parenchymatous, tephro-myelitis.

stance.[1] In these cases constant currents applied methodically may have a happy influence.

Such, gentlemen, are the principal considerations which I have to offer *à propos* of affections of the spinal cord; you will find them quite incomplete, but

[1] Progressive muscular atrophy, so-called by Aran and Duchenne de Boulogne, has ordinarily a slow and insidious beginning. Without known cause, after a fatigue, a chill, the patients experience weakness in a limb. Then fibrillary contractions, independent of the will, occur spontaneously, are more or less frequent, and consist in slight starts or shocks of the muscular fibres. To these contractions, succeeds soon a loss of strength which attracts the attention of the patient. Then in the enfeebled parts is observed atrophy of the muscles, diminution in the volume of the part; the muscular prominences disappear, and little by little with the progress of the disease all the soft parts seem to waste away and only the naked bones are left. As long as there are muscular fibres in a healthy state, electrical contractility remains, and it does not disappear till nothing is left but connective tissue. Duchenne has remarked that along with the atrophied muscles there is lowered temperature and cutaneous anæsthesia.

The disease, sometimes hereditary, more frequent in men than in women, seems to affect more particularly persons of adult age, and in full vigor; it generally commences in the upper extremities, especially the right arm, and more frequently selects the right thenar or hypothenar eminences and interosseous muscles.

The abductor brevis pollicis is taken first, then the other muscles of the thenar eminence; the atrophy gaining rapidly, the muscular prominence disappears and the movements of the muscles becoming impossible, the antagonism exerted against the extensors of the thumb is destroyed, the first metacarpal bone is drawn backwards and outwards and the hand takes the aspect of a monkey's hand. When the interosseous muscles are taken, the spaces between the metacarpal bones sink in, the skin shrinks, and the hand seems deprived of flesh.

The fore-arm is taken in its turn, and according as the atrophy affects the flexors, the extensors or the supinators, the corresponding muscular prominences are effaced. The muscles of the arm, those of the shoulder, are stricken in their turn and disappear. The shoulder, is flattened, the head of the humerus and the acromion process are plainly visible; when the fasciculi of the deltoid are invaded, the movements of the arm become impossible.

In certain cases by the side of all the atrophied muscles, a single muscular bundle remains.

Thus, in an observation of Duchenne, all the muscles of the hand and of the arm were completely atrophied with the exception of the supinator-longus which was intact. It is easy to understand the various deformities which result from these atrophies and from the vicious attitudes which the contractions of antagonist muscles, remaining intact, produce.

The muscles of the trunk are next invaded, the atrophy gains the superior part of the trapezius, the pectorals, the serratus magnus and the respiratory muscles, even to the diaphragm and intercostals. These alterations entail important troubles in respiration, while atrophy of the muscles of the abdominal walls impedes the intestinal functions, micturition and defecation. The inferior extremities are ordinarily the last to be smitten, and those most often affected are the flexors of the foot on the leg, and the hip on the pelvis.

In infants Duchenne has remarked that the atrophy instead of beginning by the upper extremities, begins by certain muscles of the face and the first affected is the orbicularis oris.

The march of the disease is slow (from ten to twelve years); it may stop after having destroyed one or two muscles, but more often after a brief respite, the disease progresses anew. The patients succumb, either in consequence of affections of the respiratory passages, by exhaustion, or by reason of asphyxia caused by the penetration of food substances into the air passages (atrophy of the muscles of deglutition.)

from a therapeutic standpoint they are necessarily so, by reason of their very incurability. These diseases of the spinal medulla, in fact, constitute the most gloomy chapter in therapeutics.

To explain the phenomena of progressive muscular atrophy, three theories have been put forth :

1st. That of Aran and Duchenne, adopted by Friedreich, which places the primordial seat of the malady in the muscles themselves which undergo degeneration.

2d. The theory of Schneevogt which has been adopted by Jaccoud, which places the starting point of the disease in an alteration of the great sympathetic.

3d. The theory of Hayem, of Charcot, of Vulpian, the theory to-day considered the most probable, which attaches this affection to the group of central myelites.

PART SECOND.

TREATMENT OF GENERAL DISEASES.