If I do not deceive myself, we may conclude from this sketch that, when there is question of complete section or of excision of the nerves, the recent observations harmonize on all essential points with the old researches. On the other hand, the results obtained by MM. Erb and Ziemssen, from the employment of contusion and ligature of the nerves of animals, are comparable with the phenomena which occur in man in consequence of irritative lesions of mixed or purely motor nerves.

Now, if this be so, the dissidences which we pointed out at the beginning of this study are smoothed away, and consequently we have reason to acknowledge, in speaking of muscular affections, *the fundamental distinction between the effects of the absence of action, and those of morbid action of the nervous system* which we have already put prominently forward in discussing cutaneous and articular affections.[1]

Trophic disorders consecutive on lesions of the Spinal Cord.

Irritative lesions of the nervous centres, like those of the nerves, have the power of producing remote trophic disorders in different parts of the body. In the exposition of these consecutive alterations which we are going to offer you, we shall again find the whole series of morbid affections (with the exception of some slight differences) which we have remarked taking place after nerve-lesion. The knowledge of their history, which we have already gained, will singularly facilitate the task that remains to be accomplished.

We may say, in a general manner, gentlemen, that the *skin*, the *muscles*, the *joints*, the *bones*, and the *viscera*, may become the seats of various trophic disorders, consequent on lesions of the spinal cord and of the brain.

of farado-muscular excitability. Galvanic excitability disappeared in the second half of the 3d month, to reappear towards the 7th or 8th month" (*loc. cit.*, pp. 592 et 593).

[1] Recent experiments made by M. Vulpian ('Archives de Physiologie,' t. iv, 1871–1872, pp. 757, 758), confirming on almost every point those of MM. Erb and Ziemssen, establish that the effects of section of peripheral nerves upon the physiological properties and structure of the muscles do not essentially differ from those caused by the application of various irritative agencies, to the same nerves, such as local contusion, ligature, and cautery. On the other hand, the histological observations of MM. Neumann ('Archiv f. Heilkunde,' Leipzig, 1868), Ranvier ('Comptes Rendus de l'Académie des Sciences,' 30th December, 1872), Eichhorst (Virchow's 'Archiv,' 1874, 12th December), have placed beyond doubt that in the peripheral extremities of the divided nerve alterations are constantly produced, *e. g.*, multiplication of the cells of the inter-annular segment, which betray the presence of an irritative process. The opposition between the effects of nerve-section and of nerve-irritation cannot, after this, be any longer maintained in the strict terms in which it was set out in this lecture (*Author's note to the 2d Edition*).

Let us take the affections of the *muscles*, in the first place, since the investigation we have just concluded has led us towards that question. The considerations we are about to set forth concerning these affections relate only to lesions of the spinal cord and medulla oblongata; for it is at least very doubtful whether lesions of the brain proper have ever the effect of directly producing alterations of the muscular tissue, and this, as we shall see in due time and place, is a fact of the highest importance.

Muscular lesions consecutive on affections of the spinal cord.—Of irritative spinal lesions, there are some which determine the very rapid production of all the kinds of functional and organic muscular alterations which we have been studying, as consequences of nerve-lesions. There are others, on the contrary, where the electrical contractility and the trophic condition of the muscles are preserved in perfect integrity, during a comparatively considerable lapse of time, for months and occasionally even for years. The muscle, in the latter case, only becomes slowly altered, under the influence of the functional inertia to which the limbs, stricken with motor paralysis, are subjected. Hence we find it possible to separate the irritative spinal disorders into two very distinct groups, which we shall pass successively in review.

A. In the *first group* we place those of the irritative lesions of the cord which, as a rule, do not directly modify the nutrition of the muscles. They have one character in common:—all tend to limit themselves to the white fasciculi of the cord, and if the gray matter be, at times, invaded, the region of the anterior cornua is respected, or at least the great multipolar nerve-cells which occupy that region are spared. Such are the different forms of *fasciculated sclerosis*, whether it be protopathic or consecutive on a circumscribed lesion (*en foyer*) of the brain or spinal cord, whether it occupy the posterior fasciculi only, or the lateral fasciculi only, or both simultaneously; so long as the express condition stated be fulfilled, that is to say, so long as the integrity of the great nerve-cells be preserved, the lesions in question may attain their highest degree of development, may, for instance, invade the white fasciculi in their whole width and their whole height, without direct deterioration to the nutrition of the muscles animated by nerves issuing from the injured portions of the cord.[1]

The scene would necessarily change, if the irritative process, exceeding the limits usually assigned it, happened to extend from the white fasciculi to the anterior cornua of the gray matter; then, in consequence of the participation of the motor-cells, a more or less rapid and thorough atrophy of the muscles might supervene.

[1] Charcot et Joffroy, "Deux cas d'atrophie musculaire progressive avec lesion de la substance grise et des faisceaux antéro-latéraux de la moelle épinière," in 'Archives de Physiologie, t. ii, p. 635.

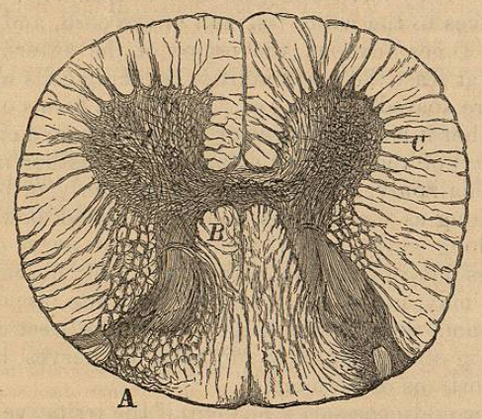
It is in this way, as I have pointed out elsewhere,[1] that the symptoms of general spinal paralysis or of progressive amyotrophy are sometimes superadded to the classic symptoms of posterior or lateral sclerosis, etc. We have quite recently seen many cases of this kind where we had opportunities of observing necroscopically, in the most distinct manner, the alteration of the nerve-cells to which, according to my view, the trophic muscular lesion should be attributed.[2]

[1] Charcot et Joffroy, *loc. cit.*, p. 354.

[2] See, amongst other cases, that recently published by one of my students, M. Pierret,* 'Sur les altérations de la substance grise de la moelle epinière dans l'ataxie locomotrice considérées dans leurs rapports avec l'atrophie musculaire qui complique quelquefois cette affection, (in 'Archives de Physiologie,' etc., t. iii, p. 599). In this instance, phlegmasic action had extended from the posterior columns to the right anterior cornu of gray matter, following the course of the internal radicular fibres of the corresponding side, the consecutive muscular atrophy was exactly limited to the members of the right side (*vide* fig. 1).

The following is a succinct account of a case which shows well the mechanism by which consecutive unilateral fasciculated sclerosis may, by extending to the gray matter, determine the production of muscular atrophy:—

A woman, aged about 70 years, had been stricken with left hemiplegia, consecutive on the formation of a blood-clot in the right cerebral hemisphere. The members of the paralyzed side, which had at an early period been contractured, commenced to diminish in bulk, not quite two months after the attack. The muscular wasting affected all parts of the paralyzed members, in an uniform manner; it was accompanied by a very marked decrease of electrical contractility



* Fig. 1.—The figure is illustrative of M. Pierrett's case (a summary of which is appended); it represents a transverse section of the spinal cord taken from the lumbar enlargement. *A*. Posterior roots. *B*. Internal radicular fasciculi traversing the area of the posterior columns. The sclerosis is seen limited, in the posterior columns, to the course of these fasciculi On the right, the phlegmasic process has extended, along the course of the radicular fasciculi to the right anterior cornu, *C*. This cornu has evidently suffered diminution, in every diameter, moreover the external group of motor cells has completely disappeared, and we find in its place a dense, opaque, apparently fibroid tissue, containing numerous disseminated myelocytes.

The same rule holds good for *disseminated sclerosis* (*sclérose en plaques*),[1] and for the *diffused scleroses*. The same may be said of *primary partial myelitic affections* or of those determined by tumour-pressure, Pott's disease of the vertebræ, etc. These different diseases have no direct influence over the nutrition of the muscles so long as they do not involve the system of motor nerve-cells.

We can scarcely conceive of an exception, save in those very rare cases, where the lesion, though circumscribed to the white fasciculi, occupies that portion of the cord traversed by bundles of nerve tubes, from which issue the posterior roots. If these bundles should be at all involved in the alterations, the equivalent of a lesion affecting the peripheral nerves would necessarily be produced.[2]

B. The *second group* will include those affections of the spinal cord, the almost inevitable consequence of which it is to determine more or less grave disorders in the nutrition of the muscles. This group may be separated into two subdivisions:—

1st. The first includes those acute or subacute lesions, whether *diffuse* or *circumscribed* (*en foyer*), which involve a great length of both the white and the gray substances, but which generally predominate in the latter. They are commonly followed by great

and made rapid progress. At the time when the atrophy became evident, the skin of the affected members, on all points subjected to the slightest pressure, presented bullæ, which soon gave place to eschars. At the autopsy, we observed, on examining hardened sections of the brain, that the descending fasciculated sclerosis of the left lateral column had been propagated to the anterior cornu of the gray matter of the corresponding side, and had there caused atrophy of a certain number of the motor-cells.

[1] In the case of a woman, suffering from multilocular cerebro-spinal sclerosis, whom we treated some years ago, one of the sclerosed patches had invaded, near the mid-cervical region, almost the whole of the gray matter of the cord, for a certain height, and the anterior cornua in particular. At this level the nerve-cells mostly presented grave atrophic lesions, and a good number of them had vanished, without leaving any trace. The woman's hands presented the deformation known as a *griffe*, *i. e.*, they were claw-like. The muscles of the thenar and hypothenar regions, as well as the interossei were atrophied; the forearms also showed great atrophy, limited to certain groups of muscles.

[2] In reference to partial myelitic affections, whether protopathic, or determined by the vicinity of a tumour, the following remark will not be out of place: They are most commonly found at a point of the dorsal region of the spinal cord, which they occupy for but a very small extent, in height. It would follow from this arrangement that if, whether primarily or in consequence of concentric extension of the morbid process, the anterior cornua of the gray matter became involved, the muscular lesions which result therefrom would be confined to very circumscribed regions of the thorax or even of the abdomen, and might not betray themselves during life, by any perceptible symptom. At all times, the nutrition of the muscles of the extremities, if there be no complication, remains perfectly intact, when the partial myelitis occupies the position we have just mentioned. It would be quite otherwise in any case where a focus of myelitis, even though very much circumscribed, should occupy certain parts of the cervical or lumbar enlargements. The muscular lesions supervening, in consequence of the invasion of the anterior cornua, would then have their seat in the extremities and would betray their presence by functional disorders, and by alterations of form, in those parts, which could not long escape attention.

modifications of electrical contractility, and by a rapidly developed atrophy of muscular fibre.

I will refer to *acute central myelitis*, in the first place. When it has been somewhat generalized, and occupies, for instance, a considerable portion of the dorso-lumbar swelling, the early diminution of electrical contractility in the lower extremities is a symptom which probably is never completely absent. Herr Mannkopf has seen, in such cases, the electrical contractility remarkably altered seven days after the appearance of the first symptoms.[1] When the patients are not too quickly carried off, you may follow the development of the correlated phenomena—the atrophy of the muscular masses soon shows itself, and the histological lesions of the primitive (ultimate) fasciculi become promptly perceptible.

According to MM. Mannkopf[2] and Engelken,[3] these lesions are chiefly remarkable on account of the proliferation of the nuclei of the sarcolemma. They bear, in fact, the stamp of the irritative process. Here, also, fatty degeneration of the primitive fasciculi is an exceptional incident. As to the nerves which supply the affected muscles, they were found by Herr Mannkopf, after repeated examinations, to be sometimes healthy, sometimes affected by comparatively slight alterations nowise proportionate in intensity to the severity of the muscular lesions.[4]

Spinal apoplexy (*hæmatomyelia*) should be mentioned in the second place. This is an affection which, considered from the standpoints of pathogeny and pathological anatomy, differs essentially from common intra-cerebral hemorrhage; for, in hæmatomyelia, the effusion usually takes place in the midst of tissues which have already suffered modification from inflammatory action. The blood is chiefly effused in the gray matter, which it often invades throughout the major part of its length. When this happens, diminution or even abolition of electrical contractility, supervening early in the muscles of the paralyzed members, is a symptom which seems constant. It was observed fourteen days after the development of the first accident, by Levier;[5] on the very day of the attack, by Colin(?); on the ninth day, in a case recorded by Duriau.[6] Spinal apoplexy is an affection which, in general, is rapidly fatal; it has as yet furnished no opportunity of observing the histological lesion of the primitive fasciculi and the atrophy of the muscular masses, which would doubtless not fail to follow if life were prolonged.

It is probable, gentlemen, that *fractures and luxations of the vertebral column*, by producing an irritation of the cord which from partial tends to become general, may have the effect of determining, as Dr. Duchenne (de Boulogne[1]) has remarked, a prompt diminution of electrical contractility in the muscles of the paralyzed member.

[1] Mannkopf, 'Amtlicher Bericht über die Versammlung Deutscher Naturforscher und Aerzte zu Hannover,' p. 251. Hannover, 1866.
[2] Mannkopf, *loc. cit.*
[3] Engelken, 'Beitrag zur Patholog. der acuten Myelitis.' Zurich, 1867.
[4] *Vide supra*, p. 36.
[5] Levier 'Beitrag zur Pathologie der Rückenmarksapoplexie.' Inaugural-dissertation. Bern, 1864.
[6] Duriau, 'Union Médicale,' t. i. 1859, p. 308.

2d. The affections which compose the second category are the product of more delicate lesions. These are, in fact, limited, in a seemingly systematic manner, to the gray matter of the anterior cornua, the entire extent of which they rarely invade; we see them often very exactly localized in the circumscribed oval space occupied by a group or cluster of motor cells (Fig. 2).

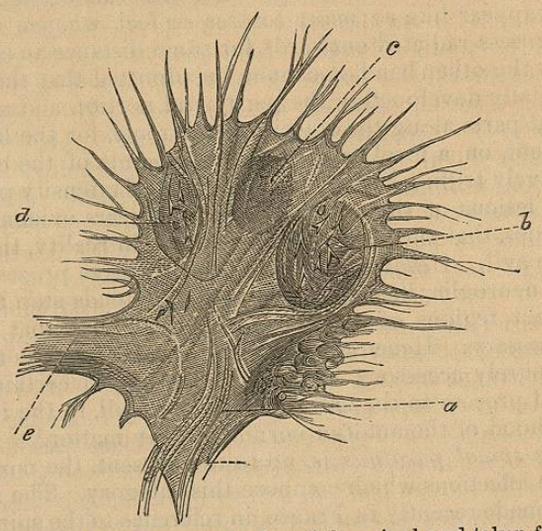


Fig. 2.—Fragment of a transverse section of the spinal cord taken from the lumbar region, in a case of infantile spinal paralysis occupying the right inferior extremity. The right anterior cornu of gray matter is represented. The lesions affect exclusively the antero-external group of nerve-cells: *a*, *cervix cornu posterioris*; *b*, postero-external group of nerve-cells; *c*, antero-external group. The cells of the latter group have completely disappeared, whereas those of groups *b* and *d* are perfectly distinct; *d*, internal group; *e*, the commissure.

The neuroglia, in the affected parts, becomes usually opaque, denser, strewn with numerous myelocytes and consequently exhibits the signs of inflammatory action. At the same time, the nerve-cells present different degrees and different modes of atrophic degeneration. But what are the elements first affected? Everything leads us to believe these to be the nerve cells. It would,

[1] Duchenne (de Boulogne), Observation, p. 246, *loc. cit.*, fracture of the vertebral column, about the middle of the dorsal region. The spinal cord softened for several inches, in the dorso-lumbar region. Enfeeblement of the electrical contractility from the sixth day after the accident.

indeed, be difficult to comprehend how the alteration could show itself strictly localized in the vicinity of the cells if its starting point were in the neuroglia. There are cases besides, in which the atrophy of a certain number, or even of a whole group, of nerve-cells is the only alteration which can be discerned, on histological examination. The connective web preserves its transparency, in those points, and all the characters of normal structure, with but few exceptions. There are other not less significant cases, also, where lesions of the neuroglia appear much more developed about the central parts of a cluster of nerve-cells, than in the peripheral portion, and also much more manifest in the immediate neighbourhood of the cells themselves, than in the intervals between them, so that they appear like so many centres or foci, whence the inflammatory process radiated outwards for some distance in every direction. On the other hand, it cannot be admitted that the irritation was originally developed in the peripheral portion and ascended to the central parts along the anterior nerve-roots, for the latter generally present, on a level with the altered points of the cords, only comparatively trifling lesions and not at all of intensity proportionate to the lesions of the gray matter. It appears evident, from all that precedes, that the motor nerve-cells are, in reality, the primary seat of the evil. Usually, it is true, the irritative process next invades the neuroglia, its second stage; and extends step by step to the different regions of the anterior cornua; but that is by no means necessary. Hence we must, *a fortiori*, regard as a consecutive and purely accessory fact, the extension, in certain cases, of the morbid process to the antero-lateral fasciculi, in the immediate neighbourhood of the anterior cornua of gray matter.[1]

Infantile spinal paralysis is, up to the present, the most perfect type of the affections which compose this category. The numerous researches made recently in France, in reference to the spinal lesions on which they depend, concur to indicate, as an essential fact, the profound alteration of a large number of motor-cells, in those regions of the cord whence the nerves emanate which supply the paralyzed muscles.[2] In the vicinity of the atrophied cells, the connective network almost always offers manifest traces of an inflammatory process. Judging from the general aspect of the phenomena, we are induced to admit, as a highly probable hypothesis, that, in infantile spinal paralysis, a superacute irritative action suddenly seizes on a large number of nerve-cells and makes them promptly lose their motor functions. Some cells, which have been but slightly attacked, will recover their functions some day, and this phase corresponds to the amelioration of symptoms which always supervenes at a certain period of the disease. Others, however, have been more severely involved, and the irritation of which they were the seat is transmitted along the nerves to the paralyzed muscles which, in consequence, suffer trophic lesions of a more or less serious character.[1] However it be, it is known that diminution or even loss of faradaic contractility may be observed, in certain muscles, barely five or six days after the abrupt invasion of the first symptoms. The emaciation of the muscular mass makes rapid progress, besides, and soon becomes evident. The alterations which, on histological examination, are found in the affected muscles are these: firstly, simple atrophy of the primitive fasciculi with the transverse striæ preserved; and, secondly, the marks of a more or less active proliferation of sarcolemma-nuclei on some isolated fasciculi. The accumulation of fat, sometimes seen in old cases, seems to be a purely adventitious phenomenon.[2]

[1] The views set forth above relative to the rôle of the so-called motor nerve-cells in the pathogeny of progressive muscular atrophy, infantile paralysis, acute central myelitis, and of all the amyotrophies arising from spinal causes, in general, were stated in a lecture which I delivered at La Salpêtrière in June, 1868. *Cf.* Hayem. 'Archives de Physiologie,' 1869, p. 263. Charcot et Joffroy, *id.*, p. 756. Duchenne (de Boulogne) and Joffroy, *id.*, 1870. These views have been utilized in the recent work of Dr. Hammond, 'A Treatise on Diseases of the Nervous System,' Sect. iv. 'Diseases of Nerve-cells,' p. 683, New York, 1871.

[2] On atrophy of the motor nerve-cells in infantile paralysis, consult Prevost, in 'Comptes Rendus de la Société de Biologie,' 1866, p. 215. Charcot et Joffroy, "Cas de Paralysie Infantile Spinale, avec lesions des cornes antérieures de la substance grise de la moelle épinière," in 'Archives de Physiologie,' p. 135, 1870, pls. v et vi. Parrot et Joffroy, *id.*, p. 309. Vulpian, *id.*, p. 316. H. Roger et Damaschino, "Recherches Anatomiques sur la Paralysie Spinale de l'Enfance," in 'Gazette Médicale,' Nos. 41, 43, et suiv., 1870 (fig. 2).

Progressive muscular atrophy offers for our study the irritative atrophy of the motor cells in its chronic form.[3] Here we are not concerned with a superacute irritative process suddenly invading the nerve-cells, and laying hold on a large number of them. These are now affected successively, one by one, in a progressive manner; a considerable number of them are spared, even in the most seriously compromised regions, until nearly the last period of the disease. The development of the muscular lesions corresponds to this mode of evolution of the spinal lesions. Thus, it is rare that the trophic disorders affect simultaneously all the primitive fasciculi of a muscle; hence it follows that the latter may respond, in a more or less imperfect manner to the command of the Will, and still enter into contraction under the influence of electrical excitations, even when its volume has been very markedly diminished.[4]

[1] Charcot et Joffroy, *loc. cit.* [2] *Ibid.*, *loc. cit.*; Vulpian, *loc. cit.*

[3] On atrophy of the motor-cells in progressive muscular atrophy, see Luys, 'Société de Biologie,' 1860. Duménil (de Rouen), "Atrophie musculaire graisseuse progressive, historie critique," Rouen, 1867. "Nouveaux faits relatifs à la pathogenie de l'atrophie musculaire progressive," in 'Gazette hebdom.,' Paris, 1867. Lockhart Clarke, "On a Case of Muscular Atrophy," 'British and Foreign Medico-Chirurgical Review,' July, 1872. "A Case of Muscular Atrophy," Beale's 'Archives,' t. iv, 1867. "On a case of Muscular Atrophy," in 'Medico-Chir. Transactions,' t. iv, 1867. O. Schüppel, "Ueber Hydromilus," in 'Archiv der Heilkunde,' Leipzig, 1865, p. 289. Hayem, in 'Archives de Physiologie,' 1869, p. 263, pl. 7. Charcot et Joffroy, in 'Archives de Physiologie,' 1869, p. 355.

[4] Charcot, "Leçons faites à la Salpêtrière in 1870." See also Hallopeau, in 'Archives de Médecine,' Septembre, 1871, pp. 277, 305.

There exist, indeed, at least two very distinct forms of progressive amyotrophy correlated to an irritative lesion of motor cells. One of them, which is *protopathic*, arises exclusively from the lesion in question, and this form, primarily developed because of an original or acquired predisposition, tends, as of necessity, to become generalized. In the other form, to which we called your attention a moment ago, the nerve-cell is only secondarily affected, consecutively on a lesion of the white fasciculi, for instance, and as it were accidentally. Progressive amyotrophy, in the latter case, may perhaps be called *symptomatic*, it has less tendency to become generalized, and its prognosis is certainly less gloomy.[1]

As regards *adult spinal paralysis*, and *general spinal paralysis* (Duchenne de Boulogne), pathological anatomy has not yet given any definite decision. But to judge from the symptoms, it is at least very probable that these affections also depend on a lesion of the motor nerve-cells of the anterior cornua. Adult spinal paralysis resembles that of childhood by the almost sudden invasion of motor paralysis, by the tendency which it shows to retrogade at a given moment, by the diminution or abolition of faradaic contractility showing itself precociously in a certain number of paralyzed muscles, and, finally, by the rapid atrophy which these same muscles constantly exhibit to a more or less marked extent. A slower evolution, often occurring in a subacute or chronic manner, a tendency to become generalized, especially evident in the first stages, frequent pauses, followed by invasion of hitherto untouched parts, distinguish, on the contrary, general spinal paralysis, and make it resemble progressive muscular atrophy, with which it is sometimes, very erroneously, confounded in clinical practice. The former, however, is clearly separated from the latter by the following characters: the muscles of an entire extremity or of portion of a limb, are struck, *en masse*, in an almost uniform manner, with paralysis or atrophy; they present, at a period but little remote from the commencement of the disease, very marked modifications of electrical contractility; usually, in conclusion, a period of recovery supervenes, during which the atrophied muscles regain, at least partially, their volume and their functions.[2]

Muscular lesions consecutive on affections of the bulbus rachidicus.—This is a subject which has been, as yet, but little explored. However some facts, which have accumulated until they now form a respectable number, gleaned from the history of labio-glosso-laryngeal paralysis and disseminated sclerosis (*en plaques*) tend to establish that, in the case of the bulbus as well as in that of the spinal cord, irritative lesions of the white fasciculi have no direct influence on the nutrition of muscles. Those, on the contrary, which affect the motor cell clusters distributed over the floor of the fourth ventricle, or the fasciculi of nerve-tubes emanating from these aggregations may, as I have demonstrated, determine a more or less marked atrophy of the muscular fibres of the tongue, pharynx, larynx, and orbicularis oris.[1]

[1] On the two forms of progressive amyotrophy of spinal origin, see Charcot et Joffroy in 'Archives de Physiologie,' 1869, pp. 756, 757. Duchenne (de Boulogne) et Joffroy in 'Archives de Physiologie,' 1870, p. 499.

[2] Duchenne (de Boulogne), 'De l'electrisation localisée,' 3d edition.

The summarized account which has been laid before you will suffice, I hope, to place in prominence the remarkable rôle which, according to the most recent researches, lesions of the anterior nerve-cells play in the production of trophic muscular disorders consecutive on alterations of the spinal cord. This rôle does not seem doubtful in the pathogeny of infantile paralysis and of the different forms of amyotrophy of spinal origin. Its influence is, certainly, less distinctly demonstrated, though still highly probable, as regards hæmatomyelia, acute central myelitis, and, in a word, all the irritative affections of the spinal cord in which the gray axis is found to be involved. On the other hand, the absence of all participation on the part of the white fasciculi, and of the posterior cornua, in the development of the muscular affections in question is a fact which henceforth rests on abundant evidence.

This acknowledged, gentlemen, we have cause to inquire why lesion of the motor nerve-cells induces that of the muscular fibres, whilst even the gravest irritative alterations of the white fasciculi have no direct influence on the nutrition of the muscles.

With respect to the first point, one cannot fail to imagine more or less plausible hypotheses which, however, are evidently premature. The teachings of experimental physiology cannot be called to our assistance here; its methods of procedure, inferior to those of disease in that respect, are not sufficiently delicate to allow the nerve-cells to be attacked in an isolated manner. We must, therefore, confine ourselves, at present, to registering the facts as they are offered us in clinical practice, illustrated by pathological anatomy, and to point out that the motor nerve-cells, comparable in that respect to the peripheral nerves, possess the power, when they have become the seat of irritation of modifying, by remote action, the vitality and structure of the muscles.

As regards the second point, if what we have said concerning the effects of nerve-irritation be referred to, it may seem contradictory, at first sight, that the nutrition of the muscles should not be affected when the white fasciculi of the cord are occupied by inflammation. To show that the contradiction is only apparent it will suffice, however, to remind you that, in spite of the analogy of com-

[1] Compare Charcot, "Note sur un cas de paralysie glosso-layngée, suivi d'autopsie," in 'Archives de Physiologie,' 1869, pp. 356, 636, pl. xiii. Obs. de Catherine Aubel. Duchenne (de Boulogne) et Joffroy, "De l'atrophie aïgue et chronique des cellules nerveuses de la moelle et du bulbe rachidien," 'Archives de Physiologie,' 1870, p. 499.

position, the white columns are not at all comparable to the nerves. Experiments in fact, reveal, in the latter, properties which are not to be found in the former, and *vice versâ*. Anatomy also shows that the nerve-tubes which constitute the nerves are but to a very small extent the direct continuation of those which, by their union, form the white substance of the cord. These fasciculi appear to be almost entirely composed of fibres which, arising either in the encephalon or in the cord itself, establish, after the manner of commissures, communications between the spinal cord and the brain, or between different points of the gray spinal axis. It was to be anticipated, from this, that, in many respects, the white fasciculi of the cord would, under the influence of irritative lesions, behave differently from the peripheral nerves.

When I formed the idea of laying before you, gentlemen, the principal facts relating to the nutritive disorders which make their appearance consecutively on affections of the nervous system, I hoped that my task might be brought fairly to an end, in the course of two lectures. But, according as I advance in this exposition, the importance and extent of the question display themselves in all their distinctness. Notwithstanding the details which I have already given, I am far from having exhausted the subject, and I dare to hope that you will not have cause to regret the time that yet remains to be dedicated to its study.

LECTURE III.

DISORDERS OF NUTRITION CONSECUTIVE ON LESIONS OF TH SPINAL CORD AND BRAIN.

SUMMARY.—Cutaneous affections in sclerosis of the posterior columns: papular or lichenoid eruptions, urticaria, zona, pustular eruptions; their relations with the fulgurant pains; the former appear to arise from the same organic cause as the latter.

Eschars of rapid development (acute bed-sores) in diseases of the brain and spinal cord. Mode of evolution of this skin-affection: erythema, bullæ, mortification of the derma, accidents consecutive on the formation of eschars: *a*, putrid infection, purulent infection, gangrenous emboli; *b*, simple purulent ascending meningitis, ichorous ascending meningitis. Acute bed-sore in apoplexy symptomatic of circumscribed cerebral lesions. It appears principally in the gluteal region of paralyzed extremities; its importance in prognosis. Acute bed-sore in diseases of the spinal cord; it generally occupies the sacral region.

Arthropathies depending on a lesion of the brain or spinal cord. *A.* Acute or subacute forms; they appear in cases of traumatic lesion of the spinal cord; in myelitis occasioned by compression (tumours, Pott's disease), in primary myelitis, in recent hemiplegia, connected with cerebral softening. These arthropathies occupy the joints of paralyzed limbs. *B.* Chronic forms; they seem to depend, like amyotrophies of spinal origin, on a lesion of the anterior cornua of the gray axis; observed in posterior sclerosis (locomotor ataxia) and in certain cases of progressive muscular atrophy.

GENTLEMEN: In treating of the nutritive disorders determined by lesions of the peripheral nerves, I gave you to expect that these consecutive affections would, for the most part, be represented in cases of lesions of the spinal axis. It is true, we shall not always find here a servile imitation; indeed, as a general rule, the trophic disorders of cerebral or spinal origin, as we shall often have occasion to note, bear with them the distinctive stamp of their cause. But there are circumstances in which the resemblance between affections of central origin and those which depend on a lesion of the peripheral nerves is so striking that discrimination may be a most difficult task. We will cite, as examples of this class, certain cutaneous eruptions which sometimes supervene in the course of ataxia.

I.

The *cutaneous affections*, to which we have just alluded, may be classified as follows: *a*, *papular* or *lichenoid eruptions*; *b*, *urticaria*; *c*, *zona*; *d*, *pustular eruptions*, analogous to ecthyma.