very restricted application, for acute bed-sore arising from lesion of the nervous centres may appear in many cases, after hemi-lateral lesions of the spinal cord for example,[1] on parts of the body where the vaso-motor innervation is not visibly affected and apart from every symptom indicative of great depression of the organism.

We have now to inquire whether the irritation of the vaso-motor nerves can account for the phenomena which are not explained by the paralysis of the same nerves. Let us first take experimental irritation. Partial ischæmia, of a more or less intense character, is the most prominent result of this irritation: it may be carried so far that not even a drop of blood flows on pricking the skin.[2] The parts, in which vascular spasm thus impedes the circulation, grow pale and cold; their vital activity decreases; the excitability of the muscles and of the nerves falls below the normal standard.[3] It is natural to think that grave nutritive lesions, tending to necrobiosis or to sphacelus, should necessarily result from the prolongation of such a state. But it is important to observe that this is commonly a question of a temporary phenomenon, lasting at longest for a few hours only. For by the very fact of the prolongation of the irritation the action of the nerve seems to exhaust itself, and hyperæmia, generally, soon follows on anæmia.[4] However, by reproducing, at short intervals, the irritation of the vaso-motor nerves, it is possible to cause the ischæmic state to predominate for a certain time. Still I do not believe that any trophic lesion would be ever experimentally produced, by this method. Herr O. Weber, who, by means of an ingenious apparatus, says he has kept up, for nearly a week, irritation of the cervical sympathetic nerve, of a permanent character, so to speak, and marked by a thermal decrease of 2° C., has not seen the slightest trace of nutritive trouble supervening in the corresponding side of the face.[5] Cases connected with human pathology yield the same testimony. Thus it is not rare to find, in certain cases of *angioneuroses*, amongst hysterical patients for example, a very intense and very persistent partial ischæmia; yet trophic troubles never show themselves, under such circumstances.[6] As to the instances of spontaneous gangrene, which have been attributed to vascular spasm, they would not have, to judge from my own observations, the signification assigned them; for, in all cases of this kind which I have happened to meet with, I have found the calibre of the vessel

[1] See *ante*, p. 72.
[2] Brown-Séquard, 'Course of Lectures,' &c., p. 147, Philadelphia.
[3] Brown-Séquard, *loc. cit.*, p. 142.
[4] Waller, 'Proc. Royal Society, London,' vol. ii, 1860–72, p. 89 et seq.
[5] O. Weber, 'Centralblatt,' No. 10, 1864, p. 147.
[6] Liégeois, 'Société de Biologie,' 1859, p. 274. Charcot, in 'Mouvement Médical,' Nos. 25, 26, 1re série; No. 1, nouvelle série, 1872.

occluded by an alteration of the arterial walls, or obstructed by a thrombus.[1]

From the foregoing observations you perceive that it is neither to a paralytic nor to an irritative affection of the vaso-motor nerves, *properly so called*, that we should attribute the trophic disorders which supervene in consequence of lesions of the nervous system.

Physiological experiments, in these latter years, have revealed the existence of centrifugal nervous filaments, the irritation of which has the effect of determining dilatation of the bloodvessels, and consequently hyperæmia of the region to which these nerves are distributed.

Whilst irritation of the common vaso-motor nerves produces ischæmia, irritation of the *dilator nerves* determines, on the contrary, a more or less intense hyperæmia. The chorda tympani may be considered, at the present moment, as the prototype of dilator nerves. But nerves endowed with similar properties exist in the face,[2] in the penis,[3] and in the abdomen.[4] There are probably others in existence also in many parts of the body.

We are far from possessing a certain knowledge of the mode of action of these nerves. According to the hypothesis adopted by M. Claude Bernard, this is how we should explain the remarkable afflux of arterial blood which takes place in the submaxillary gland, under the influence of the chorda tympani. In the opinion of that eminent physiologist, the irritation of this nerve is transmitted to the little ganglionic masses which are distributed in great numbers on the intra-glandular extremities of the nerve. These would, in their turn, react by a sort of *nervous interference*[5] on the nerve-filaments of the great sympathetic or vaso-constrictor, and paralyze their action. Thus the chorda tympani, and the same doubtless should be said of all the other dilator nerves, would play the part of a check nerve in relation to the vaso-motors. Hence, as you see, the result of the action of the dilator nerves would, according to this theory, be simply vaso-motor paralysis.[6] Now, if it be true

[1] See the Thesis of M. Benni, 'Recherches sur quelques points de la gangrène spontanée,' Paris, 1867. Obs. v, xi, xvii.
[2] Claude Bernard, 'Revue Scientifique,' t. ii, 2 serie, 1872. Schiff, 'Digestion,' t. i, p. 252.
[3] Erector nerves of Eckhardt, 'Beitrage zur Anat. und Phys.,' t. ii. Löven, 'Bericht der Sachs. Ges.,' 1866.
[4] Claude Bernard, *loc cit.*
[5] Claude Bernard, *loc. cit.*, p. 1201.
[6] For a clinical illustration in the human subject of the physiological theory, see "Note sur la Paralysie vaso-motrice généralisée des membres superieurs," par le Dr. Sigerson (Publications du 'Progrès Médical'), 1874, Adrien Delahaye, Paris; or Translation by Dr. Barnard Ellis, New York.

The following are some of the principal features of this case, to which Dr. Duchenne (de Boulogne) invited the writer's attention, and which, at the request of that eminent physician, was made the subject of a detailed study, from pathological and physiological stand-points. The extract is taken from Dr. Barnard Ellis's translation:—

"*History.*—The patient C—, aged 50, a copper-trimmer by trade, is a man of

that vaso-motor paralysis, even when carried very far, as happens for instance in cases of complete section of the vaso-motor nerves,

robust constitution, and florid complexion, who has hitherto enjoyed excellent health. He has had neither cough, nor colic, nor any of the symptoms usually assigned to copper-poisoning, whether the heart, the respiratory, or the digestive organs be considered. The hands, which are in an abnormal condition, present no lesion except the cicatrix of an old whitlow on the left forefinger. He came to be treated for impotence, and that, at first, was all he complained of; but other phenomena were soon discovered, some of which were traced back several years. By careful questioning the following facts were elicited. In 1872 he was aware of a weakness in the arms and legs, but most especially in the knees. This sensation, however, neither became localized nor remained constant; it seemed to flit through all his members. In 1873, he noticed that it predominated in the left knee. This uneasy sensation, which gave no pain, and was transient, seemed to ascend along the leg from the calf to the thigh; and the proof that it was not merely a subjective sensation lies in the fact that the weakness of the leg increased so much at times that he was obliged to sit down. He usually recovered, however, in a few minutes, and was able to go about his work as before. No aggravation of his symptoms occurred when he walked out; on the contrary, the exercise did him good, and after a brisk walk of half an hour he felt a marked sensation of pleasurable ease.

This disorder, as we see, was intermittent, showing itself after intervals of comparative health. In January last (1874), however, he was attacked, in a more enduring manner, in both upper and lower extremities,—the feeling of debility being greatest in the left arm and right leg. So much was he enfeebled that, whereas when formerly attacked he could lift a weight of about two or three pounds, he became at this time unable even to keep his forearm flexed on his arm. He preserved the power of flexion, but not the power of maintaining it, as, in a few seconds, the forearm would fall of its own weight. At this stage, the muscular force of the hands, tested by the dynamometer of Dr. Duchenne (de Boulogne), was equivalent, on an average, to 43 kilogrammes, or 94.6 lbs.

The colour of his hands had become a deep red, and this florid flush extended up the forearms, gradually diminishing in intensity. Let us add that, notwithstanding the vascular disturbance, there was nothing that could be referred to the existence of scleroderma, to which there was some superficial resemblance. The patient complained of great heat in the hands and forearms, and this increase of temperature was plainly perceptible to all who touched them; and it is a very remarkable fact that their sensibility was so greatly augmented, that everything he touched—instruments, wood, or paper—appeared to him as cold as ice. He was troubled with formication in the forearms, which increased to a painful degree when he rubbed his hands together as when washing them. Heat aggravated and cold diminished the pain,—facts of which he had become aware on using cold and warm water.

As to the inferior extremities, they presented different phenomena. There was, indeed, debility, as has been remarked, but the symptoms of the disease seem to have decussated. Whilst the left arm was the weaker, the right leg was the more feeble. Instead of the hyperæsthesia which we observed in the hands, there was a notable loss of sensibility in the right foot, so that he did not feel the ground when walking. This foot seemed to him asleep or benumbed. There was, at times, slight formication in the right leg, but very little in the left. Nor was there a hypothermal condition here, as in the upper extremities. Although the temperature of the soles of his feet seemed normal to himself, during the daytime, it had been remarked that, when he was lying down and during the night, they were ice-cold to the touch.

In the lumbar region, he had experienced an intense itchiness, as if he had been beaten with nettles. This unpleasant sensation was not constant, and had only appeared five or six times in all, and then only in the morning and at night, when he was dressing or undressing and exposed to the cold air—phenomena occasionally present in the case of persons suffering from urticaria. There were no wheals perceptible. It is highly interesting to note that when this urtication made its appearance in the loins, the formication disappeared from the upper extremities.

As there was reason to suspect the existence of ocular troubles, we interrogated his memory, and found that he had observed something like a mist before his eyes, especially at night. This disorder had, in fact, reached such a point in January, 1874, that he had given up attempting to read. On the left eyeball, a harmless pterygium was remarked. Applying the ophthalmoscope, Professor Panas found that the fundus of the right eye was normal, whilst there was a very marked excavation of the papilla of the left eye, the fundus of which was slightly congested.

Let us note, in conclusion, that during the continuance of his ailment he complained of great thirst, and of unusual drowsiness after meals."

This patient recovered under treatment by faradization, as related in the treatise already mentioned (S.).

is not a cause of trophic disorders, the same rule must plainly hold good as regards the paralysis produced under the influence of the dilator nerves. But, gentlemen, as you will see further on, the mode of action of the dilator nerves may be considered from an altogether different point of view.

I would remind you of the fundamental experiments of Ludwig, relative to the influence of certain nerves on the secretion of the submaxillary gland.[1] Notwithstanding the criticisms which have assailed the conclusions drawn from his experiments by this celebrated physiologist, these conclusions do not appear to have been shaken. I have to request your permission to enter into some details in reference to this subject; they are absolutely necessary for the object we have in view.

When you irritate the peripheral end of the nerve proceeding to the submaxillary gland—a nerve supplied as we now know from the chorda tympani—the following phenomena are observed. A very abundant secretion of saliva is produced, the quantity may be so large that, in a short space of time, the volume of saliva secreted shall greatly exceed the volume of the gland itself. This fact demonstrates at the outset that we have not to deal here with a simple phenomenon of excretion, or expulsion of previously secreted saliva.

According to the views of Stilling and of Henle, which prevailed at the time Ludwig published his first investigations, one might be tempted to explain the phenomena in question by admitting that the irritated glandular nerve acts upon the veins of the gland, causing them to contract. The augmentation of the tension of the blood, consequent on the venous contraction, would, by this hypothesis, be the cause of the augmentation of the salivary secretion. But Ludwig has shown that ligature of the veins, without concomitant irritation of the glandular nerve, does not increase the

[1] Ludwig, 'Mitth. der Zurich Naturforsch.,' 1851. 'Zeitschr. für rat. Med.,' n. f. Bd. i, p. 255. 'Wiener Med. Wochenschr.,' 1860, x, No. 28, p. 483. See also the works published by Ludwig in co-operation with Becher, Rahn, and Gianuzzi.

secretion of saliva. That second hypothesis should, therefore, also be eliminated.

But perhaps the irritation of the glandular nerve which, as you are aware, has the effect of inducing dilatation of the arteries, may determine the secretion, simply because it momentarily augments the afflux of arterial blood into the gland? This argument is rendered invalid by an experiment made by Ludwig, which shows that, during irritation, the manometric pressure in Wharton's duct is superior to the pressure of the blood in the arterial conduits. Besides, the hypersecretion of saliva from irritation of the chorda tympani is still exhibited, after ligature of the arteries supplying the gland—in the case of an animal killed by bleeding—or even in the case of a head separated from the body. Let us also add this most remarkable fact, namely, the saliva and the venous blood which flow forth from the submaxillary gland, whilst the glandular nerve is being stimulated, present, as MM. Ludwig and Spiess[1] have shown, a higher temperature than the arterial blood which passes into the gland.[2]

Judging from the general bearing of these results, it appears evident that the influence of the nervous system on the submaxillary secretion cannot be explained by the simple phenomena of vascular dilatation and constriction. We are induced to recognize in the glandular nerve a two-fold property, since, in addition to its influence over the vessels, the dilatation of which it determines, it also exerts an immediate action on those parts of the gland which accomplish the chemical act of secretion, or, in other words, upon the secreting cells. This influence of the nerve upon secretion seems, indeed, to be the fundamental fact, for it shows itself, in consequence of excitation, even when the effects of the concomitant dilatation are annihilated. As, on the other hand, it does not appear possible, experimentally, to suppress separately the secretor action, leaving the dilator action alone persisting,[3] it is legitimate to suppose that the latter depends on the former as a more or less direct consequence.

We had, therefore, reason to inquire what might be the link of connection between the excitation of the secretor elements determined by stimulation of the nerve, and the hyperæmia which

[1] Ludwig und Spiess, 'Sitzungsber.,' d. v., Ak. Math. Cl., 1857, Bd. xxv, p. 584.
[2] In reference to this, see a Lecture of M. Vulpian, 'Revue des Cours Scientifiques,' 3d année, 1865-1866, p. 741.
[3] By recent experiments, however, M. Heidenhain seems to have been able to demonstrate that, in the chorda tympani, different nerve-fibrils are devoted to secretion and to circulation in the submaxillary gland. He states that in dogs, placed under the influence of woorari, after injection into the jugular vein of a dose of atropine sufficient to completely paralyze the cardiac filaments of the pneumogastric, the stimulation of the chorda tympani no longer determined the slightest secretion. Nevertheless, there was an acceleration of the venous current which did not notably differ from the acceleration determined by irritation of the chorda, before poisoning. 'Archives de Physiologie,' 4 Juillet, 1872.

follows that excitation. Several physiologists have thought that we have here to deal with an *attraction* which the secretor elements of the gland should exert upon the blood. "So that to the force hitherto known as assisting the return of the circulating blood to the heart and which is termed *vis a tergo*, we should add a new attractive force in correlation with the intimate nutrition of the elements, a force named by many authors *vis a fronte*."[1] Is this a purely theoretical conception, unsupported by experiments, and merely destined to cloak our ignorance? By no means. The works of H. Weber, Schuler, Lister, etc.,[2] contain numerous experimental facts calculated to render evident the *attraction* which the tissues can exercise, under certain conditions, over the circulating blood. I will cite two facts of this sort as examples, in which the phenomena may be studied apart from any intervention of the nervous system. I borrow them from a lecture on the Theory of Secretions, delivered in the Museum of Natural History, by Professor Vulpian.[3]

If you cut all the nerves of a frog's limb and then determine an excitation by placing a small drop of nitric acid on the skin of the web of its foot, a more or less intense hyperæmia will be produced in this point, at the end of a certain period. The second fact is conclusive. An egg on the fourth day of incubation presents a very distinct vascularity of the umbilical membrane. At that period, there cannot be the slightest question of nervous influence. Now, if you place a small drop of nicotine on any point of this vascular area, there ensues around this point so great a congestion that almost all the blood flows thither. In truth, this hyperæmia, this stasis by irritation of the tissues, displays itself, at first glance, with I know not what semblance of a metaphysical conception. But an effort has long since been made to give an interpretation of this phenomenon on physico-chemical grounds. Thus, in 1844, Dr. Draper[4] remarked that where a capillary tube contains two liquids, of different natures, if one of them have a greater chemical affinity for the parietes of the tube than the other, motion ensues, and the liquid which has the greater affinity pushes the other before it. The arterial blood having a greater affinity for the tissues than the venous blood, saturated with the products of disintegration, it should follow that the venous blood would be driven back. According to this hypothesis, it would suffice to quicken the chemical process of nutrition, in order to increase the intensity of motion (or afflux), and herein the action of the nerves may intervene. The phenomena of stasis are capable of being

[1] Vulpian, 'Revue des Cours Scientifiques,' t. iii, p. 744.
[2] See O. Weber, 'Handbuch der Chirurgerie,' t. i, p. 111.
[3] Vulpian, *loc. cit.*, p. 743.
[4] Draper, "A Treatise on the Forces which Produce," &c., New York, 1844. Savory, 'British and Foreign Review,' t. xvi, 1855, p. 19.

explained in an analogous manner, by an appeal to the laws of osmosis (blood-stasis, by diffusion).[1]

However it be, whatever may be the explanation of the phenomena, you perceive that the attraction which the tissues, under the influence of certain agents, exercise upon the blood is a fact experimentally established, wholly apart from any action of the nervous system. Now, in order to apply this datum to the case of the submaxillary gland, it suffices to admit that the glandular nerve, when subjected to excitation, induces a modification of the intimate nutrition of the secretor cells—and then, in consequence of this change, vascular dilatation would take place.

Anatomy seems, besides, to throw a new light upon the question by showing that the terminations of the glandular nerves penetrate into the secretor cells.[2] Herr Heidenhain has even endeavoured to demonstrate that a gland, of which the nerves have been subjected to a somewhat prolonged irritation, presents a histological constitution differing in some respects from that of a gland in a state of repose. The old cells, termed mucous cells, appear in fact, after the irritation, to be replaced by young cells of recent formation.[3] If the views of Herr Heidenhain be confirmed, we should attribute to the nerve a direct influence, so to speak, upon the development of gland-cells.[4]

The hypothesis which has just been formulated in relation to secretor nerves, might apparently be extended to other nerves in which experimental physiology has discerned the property of determining the dilatation of vessels under the influence of stimuli. These nerves would act primarily on the inter-vascular elements and quicken therein the movements of composition and decomposition. Vascular dilatation would follow, as a consecutive phenomenon. In support of this view, one may here also invoke the teachings of anatomy which, in these latter days, has, it is stated, succeeded in following, at least in the frog, the nerve-endings even into the nucleoli of the corpuscles of the cornea, and of the conjunctival cells of the nictitating membrane.[5]

[1] O. Weber, *loc. cit.*

[2] E. F. W. Plüger, "Das Nervengewebe der Speicheldrüse," in Stricker's Handbuch, t. i, p. 313.

[3] Heidenhain, "Studien der Physiologischen Instituts," 3e Breslau, 1868, and Stricker's Handbuch, *loc. cit.*, p. 330.

[4] According to M. Ranvier ('Traduction de Frey,' p. 437), and M. Ewald ('Jahresber.,' t. i, 1870–1871, p. 55), the results obtained by Herr Heidenhain ought to be interpreted as follows: Under the influence of the irritation of the gland-nerves, the cells called mucous cells simply lose the mucus they contain and resume the appearence of parietal gland-cells. There would consequently be no formation of new cells here, as Heidenhain asserts.

[5] See Kühne, in 'Gaz. Hebdom.,' t. ix, No. 15, 1862. Lipmann, "Endigung der Nerven im eigentlichen Gewebe und im hinteren Epithel der Hornhaut des Frosches," in Virchow's 'Archiv,' 38e Bd., p. 118, 1869. Eberth, in 'Archiv für Mikros. Anat.,' Bd. iii.

This interpretation was long since proposed by M. Brown-Séquard,[1] and Professor Schiff seems to countenance it when he acknowledges that "active dilatation appears to be alien to the proper coats of the vessels and to take place through the medium of the inter-vascular tissues."[2]

The incursion which we have made into the domain of physiology was undertaken with the intention of collecting, as we went, evidences which we can now profitably apply. It is requisite, indeed, to fix your attention for a while upon the *trophic nerve theory*, as it is called, which, in default of other hypotheses whose insufficiency was admitted, has been sometimes recurred to in order to explain the production of nutritive lesions developed by an influence of the nervous system. Now, by this theory, at least as it has been formulated by Herr Samuel, the supposititious nerves would be, as it were, constructed after the model of the secretor nerves in this respect that, like them, they would exercise, in the normal state, a direct influence over the nutrition of the parts where it is supposed their ultimate terminations are distributed. Their physiological role would be, not to operate directly, but to quicken, throughout the tissues, those exchanges which constitute elementary assimilation and disassimilation, just as the function of the secretor nerves is to set at work in the gland-cell an inherent property, indefeasibly connected with the phenomena of intimate nutrition. The autonomy of the anatomical elements in accomplishing the nutritive act is therefore not at all overlooked; it is only proposed to consider the trophic nerves as forming, in their totality, a perfecting appliance peculiar to superior organisms.

So much for the physiological aspect of the theory: Now, as regards its application to the interpretation of pathological phenomena, it is easy to conceive that a frequent result of morbid irritation set up in nerves, endowed with such properties, would be to carry disturbance into the intimate nutrition of the innervated parts, and to provoke therein, occasion serving, the consecutive development of the inflammatory process. Suppression of the action of these nerves would, on the contrary, have no other effect than that of lessening the intensity of the nutritive movement, and *circumscribed atrophy* is mentioned as an example of the trophic disorders which may thus supervene.

These are the general features of the theory; as to the details, it was to be anticipated that an hypothesis created by the need of explaining as yet but little known phenomena, insufficiently examined at the period of its publication, was destined to become speedily antiquated. That, in fact, is what has happened. It cannot be admitted to-day, that all the trophic nerves have their central

[1] Brown-Séquard, 'Researches on Epilepsy,' p. 70. 'Central Nervous System,' pp. 148, 172, 174.

[2] Schiff, 'Leçons sur la Digestion,' t. i, p. 256.

origin in the posterior spinal ganglia, or in the analogous ganglia of the cranial nerves; for the cases are numerous, as you have seen, where a lesion, situated in the central portion of the spinal cord, or even in the encephalon, provokes the manifestation of trophic derangements in the peripheral parts. Henceforth, also, we must take count of facts, unknown when Samuel's book appeared, which place beyond all doubt the influence of lesions of the anterior nerve cells on the development of different kinds of myopathies.

I have never shared in the disdain with which the theory, that has just been briefly described, was almost universally met. It has ever seemed to me that, in spite of its imperfections, it was worthy of being recommended to the attention of physicians because it explains better, in my opinion, the phenomena which they are called on to observe, in practice, than all the other hypotheses previously invoked. I am very far, however, from wishing to ignore the objections alleged against it. In the first place, the existence of trophic nerves is not, certainly, demonstrated anatomically; it must be admitted, moreover, that most of the experiments made on animals by Herr Samuel, with the object of revealing their existence, have not been felicitous. Some, when repeated by other observers, have not hitherto reproduced the stated results; others have had to be given up, as tainted with numerous causes of error.[1] But all the arguments directed against this theory have not so much value as these. If, for instance, we were bound to condemn the hypothesis of trophic nerves, by the mere fact that it is useless in physiology, I would point out that the utility of the secretor nerves was only recognized, as an afterthought. We should, in like manner, be necessarily compelled to recognize the utility of trophic nerves, if experiments should at any time declare in their favour. Again, it is difficult to believe that the part played by the secretor nerves is absolutely special, and wholly unexampled in the organism. With these nerves we can already compare the dilator nerves, if it be true that they act according to the mechanism already indicated. We should place also beside them, following the recent observations of Herr Goltz, the nerves of absorption which, according to this physiologist, act upon the endothelial cells of the blood-vessels in the same way as the nerves of secretion act upon the glandular epithelium. On the whole, we do not see that any reason exists to decree, *a priori*, that the trophic nerves shall not, some day, be called to a place in this group.[2]

However this be, before adopting a theory which cannot subsist without calling out a whole system of nerves whose existence is as yet problematical, it is necessary to make sure, by every means,

[1] See Tobias, 'Virchow's Archiv,' bd. xxiv, p. 579, and O. Weber in 'Centralblatt,' 1864, p. 145.

[2] Goltz in 'Pflüger's Archiv,' t. i, v, p. 53, and 'Journal of Anatomy and Physiology,' 2d series, May, 1872, p. 480.

that it is really impossible to explain the phenomena, the interpretation of which is required, by appealing to the properties of the different nerves already known. We must take care not to infringe the axiom of Logic, *Haud multiplicanda entia absque necessitate.* Now, the vaso-motor theory being eliminated, there yet undoubtedly remains much to be done from this point of view.

There is one opinion, amongst others, which has not received attention, so far as I know, and which perhaps deserves to be taken into consideration. The numerous and decisive experiments which have been recently made on the connections formed by uniting ends of divided nerves possessing different functions, such as the hypoglossal and lingual nerves for example,[1] have shown that excitations, produced on any point of a sensitive or motor nerve-fibre, are propagated at once and simultaneously in centripetal and centrifugal directions. From this, it is allowable to suppose that pathological irritations, developed on a sensitive nerve, either at its central origin or on some point of its course, reverberating in a centrifugal direction to the ultimate extremities of the nerve filaments, *i. e.*, to the papillæ of the derm, or the substance of the rete mucosum,[2] could there provoke inflammatory action, in certain cases. In this way we could comprehend, for instance, the somewhat frequent development of pemphigoid or bullar eruptions, and of zona, in consequence of lesions affecting the posterior fasciculi of the cord, or the sensitive spinal roots. With respect to the motor nerves, I do not see any serious reason to prevent us from admitting that pathological irritations, affecting the nerve-cells of the anterior cornua, would sometimes be transmitted to the muscular fasciculi, by means of the nerve filaments which, in the physiological state, transmit voluntary excitations. A certain number, at least, of the trophic disorders, consecutive on lesions of the nervous system, would perhaps find their explanation in this hypothesis, without its being necessary to have recourse to the trophic nerve theory.

We have arrived, gentlemen, at the conclusion of this pathogenic discussion, and, as I allowed you to perceive from the beginning, the question in dispute still awaits its solution. I shall not regret, however, the course of explanation we have followed, if, by placing before your eyes the documentary evidences of the case, I have succeeded in inspiring you with the desire of entering more deeply into an investigation which concerns, to such a supreme extent, the pathology of the whole nervous system.

[1] Vulpian, 'Physiologie du Système Nerveux,' p. 290.

[2] See Langerhans, 'Virchow's Archiv,' Bd. 44, and A. Biesadecki, Stricker's Handbuch, p. 595.