mary or secondary irritative lesion of the nerve-cells of the anterior cornua of the spinal gray matter appears, in the majority of cases, to be the starting-point of the amyotrophy which, in clinical practice, is usually designated by the name of progressive muscular atrophy.

For to-day, gentlemen, I shall stop here in this investigation, which I expect to bring to a conclusion at our next conference.

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## *LECTURE IV.*

### NUTRITIVE DISORDERS CONSECUTIVE ON LESIONS OF THE BRAIN AND SPINAL CORD (*Conclusion*). AFFECTIONS OF THE VISCERA. THEORETICAL OBSERVATIONS.

SUMMARY.—Visceral hyperæmia and ecchymoses consecutive on experimental lesions of different portions of the encephalon, and on intra-encephalic hemorrhage. Experiments of Schiff and Brown-Séquard: personal observations. These lesions seem to depend on vaso-motor paralysis: they should form a separate category. Opinion of Schroeder van der Kolk, relative to the relations alleged to exist between certain lesions of the encephalon and different forms of pneumonia, and pulmonary tuberculization. Hemorrhage of the supra-renal capsules in myelitis. Nephritis and cystitis consecutive on irritative spinal affections of sudden invasion, whether traumatic or spontaneous. Rapid alteration of the urine under these circumstances; often remarked contemporaneously with the development of eschars in the sacral region; its connection with lesions of the urinary passages which are due to direct influence of the nervous system.

Theory of the production of nutritive disorders consecutive on lesions of the nervous system. Insufficiency of our present knowledge, with respect to this question. Paralysis of the vaso-motor nerves; consecutive hyperæmia; trophic disorders not produced. Exceptions to the rule. Irritation of the vaso-motor nerves: the consequent ischæmia seems to have no marked influence on local nutrition. Dilator and secretor nerves: researches of Ludwig and Claude Bernard; analogies between these two orders of nerves. Theoretical application of trophic nerves. Samuel's hypothesis. Exposition. Criticisms. Conclusion.

GENTLEMEN: The reverberation of lesions of the nervous system is not felt only in the peripheral parts, in the skin, bones, and muscles, the viscera themselves may also be influenced by these lesions. It is known that certain alterations of the encephalon



especially those which affect the optic thalami, the corpus striata, and particularly the different parts of the isthmus, whether caused experimentally, or spontaneously produced, are occasionally followed by the manifestation of certain visceral lesions.

Thus in some experiments made by Professor Schiff[1] and by Brown-Séquard[2] there frequently supervened in the lungs, stomach, or kidneys, either simple hyperæmia or real ecchymoses, consequent on traumatic irritation of the optic thalami, corpora striata, pons Varolii, and bulbus rachidicus, etc. Again, nothing is more common, as I have shown, than to find in man, in cases of apoplexy symptomatic of cerebral softening, but especially in cases of intra-encephalic hemorrhage in foci, patches of congestion and real ecchymoses on the pleuræ, the endocardium, and the mucous membrane of the stomach.[3]

What is the reason of these singular alterations? Professor Schiff does not hesitate to look on them as being simply the effects of the paralysis of the vaso-motor nerves.

I am very much inclined, for my part, to believe that the pathogenic process is here more complex. Nevertheless, the direct influence, so to speak, of neuro-paralytic hyperæmia on the development of ecchymoses, in apoplectic patients, seems well established by the following case which I communicated to the *Société de Biologie*, in 1868.

A female in La Salpêtrière was struck with apoplexy, followed by hemiplegia of the left side, and succumbed a few days after. The paralyzed members had presented a comparatively considerable increase of temperature. At the autopsy, we discovered in the right hemisphere a recent hemorrhagic focus, occupying the corpus striatum. The epicranial aponeurosis presented on the left, or hemiplegic side, a wine-red colour, and, here and there, spots of ecchymosis.

The abnormal colour and the ecchymoses stopped suddenly at the median line. The right half of the epicranium had preserved its customary pallor: no traces of ecchymosis were to be found. Spots of ecchymosis were observed in the substance of the pleuræ, of the endocardium, and of the mucous membrane of the stomach.[4]

However it be, the visceral lesions in question differ by important characteristics from the affections which form the principal object of our studies. Those are congestions and ecchymoses, as we have said; the symptoms of inflammation are never superadded without the intervention of some accessory cause, a thing

[1] Schiff, 'Gazette Hebdomadaire,' t. i, p. 428. 'Lezioni di Fiziologia sperimentale sul systema nervoso encefalico,' pp. 287, 297, 373. Firenze, 1866. 'Leçons sur la Physiologie de la Digestion,' t. ii, p. 433. Florence, 1867.

[2] 'Société de Biologie,' 1870.

[3] 'Comptes Rendus de la Société de Biologie,' 19 Juin, 1869. Paris, 1870.

[4] Ibid., année 1868. Paris, 1869, p. 213.

altogether unnecessary, as you are aware, in cases of common trophic lesions. We have consequently grounds for placing in a separate category, at least temporarily, the congestions and ecchymoses which make their appearance consecutively on lesions of different parts of the encephalon.

Again, some authors, Schroeder van der Kolk amongst others, consider that the different forms of pneumonia, and even of pulmonary tuberculization, which frequently supervene in the course of certain encephalic affections, depend, in such circumstances, on the influence of lesions of the brain and medulla oblongata upon the lungs. But it must be acknowledged that the cases upon which the alleged connection rests are not yet sufficiently convincing.[1]

*Spinal lesions*, as well as lesions of the encephalon, may be followed by the production of visceral ecchymoses. It will suffice for me to remind you that if the lumbar cord be wounded with a puncturing instrument, in a guinea-pig, effusion of blood into the supra-renal capsules occasionally follows.[2] It seems right to recall this experiment of Brown Séquard, because human pathology supplies us with analogous facts. Quite recently my friend Dr. Bouchard has told me of a case of acute myelitis, observed in Professor Béhier's wards, and promptly terminating in death. At the autopsy, besides the lesions of partial myelitis, recent hemorrhagic foci were discerned in the substance of the supra-renal capsules.

But, I repeat, congestive and ecchymotic lesions appear to form a separate order. On the other hand, the affections of the kidneys and of the bladder, to which I wish now to call your attention, are, by the general bearing of their characteristics, allied to the group of trophic lesions, properly so called.

You are aware that *nephritis* and *cystitis* are very common com-

[1] Schroeder van der Kolk. "Atrophy of the brain," Sydenham Society, 1861. The author dwells on the fact that, according to the statistics published in his Treatise on the Spinal Cord, all the epileptic patients whose tongues were bitten, succumbed in consequence of phthisis, pneumonia, or marasmus. He adds that, according to Durand-Fardel, patients attacked by brain-softening almost always die of a pulmonary affection, and he quotes Engel's statistics which support this view ('Prager Vierteljahrschr.,' vii Jahrg., Bd. iii). He refers to the experiment, now of old date, in which Schiff believed he saw, in the rabbit, tubercles (?) developed in the upper lobe of the lung after the section of the ganglion of the pneumogastric nerve ('Wunderlich's Archiv,' 6 Jahr., 8 heft, pp. 769 et seq.), and finally points out that, among the observations collected by Brown-Séquard in his "Recherches sur la Physiologie de la protubérance annulaire" ('Journal de la Physiologie,' t. i), there are a certain number where phthisis and pneumonia occasioned death. Cruveilhier, Andral, and Piorry had long since noted the predominant part which, according to them, acute pneumonia plays in the issue of apoplexies determined by cerebral softening or hemorrhage.

According to the observations which I have collected at La Salpêtrière, lobular or lobar inflammations of the lungs would be less frequent, under the circumstances, than these physicians seem to believe.

[2] Brown Séquard, "Influence d'une partie de la moëlle épinière sur les capsules surrénales," 'Comptes Rendus de la Société de Biologie,' 1851, t. iii, p. 146.



plications of irritative spinal affections, of sudden invasion, whether they be of traumatic origin, or spontaneously developed.

It has been long recognized that, after fracture of the vertebral column with consecutive lesion of the spinal cord, the urine frequently undergoes rapid alteration. Dupuytren pointed out, as you may recollect, that in such circumstances the catheter left in the bladder to guard against retention of urine, became rapidly coated with a calcareous incrustation.[1] But it was Brodie especially, who called attention to the characters presented by the urine in the case of persons stricken with traumatic paraplegia.[2] On the eighth, on the third, and on the second day, he has observed the urine become alkaline, and exhale a fetid ammoniacal odour, at the moment of emission. Soon afterwards, it contained blood-clots, muco-pus, deposits of ammoniaco-magnesian phosphates. It would, in fact, be easy to gather from authors a very great number of cases in which the urine-changes, noticed by Brodie, have occurred in the first days following on paraplegia, determined by fracture of the vertebral column.[3] At the autopsy, in such cases, more or less advanced lesions of purulent nephro cystitis are found.[4]

But traumatic lesions of this kind are, in general, little suited to illustrate clearly the relations which exist between inflammation of the urinary passages, and alterations of the spinal cord. For it can always be supposed, if strictly considered, that a fall or concussion violent enough to produce fracture of the spine, may have determined the vesico-renal lesions by the same shock.

It is otherwise when we have to deal with an affection, spontaneously developed in the spinal cord, or with a wound determined in this organ by the blow of some sharp weapon. Now, even in cases of this kind, it is common to find, a short time after the invasion of the paralytic phenomena, a more or less marked modification in the constitution of the urine, connected with nephro-vesical alterations, not unfrequently of a serious character. I shall confine myself to mentioning, by way of example, the following facts.

In one case, previously described, of hemiparaplegia caused by a knife stab, the urine became alkaline on the third day, and soon after muco purulent. Death occurred on the thirteenth day.

At the autopsy, very evident inflammatory lesions were found in the kidneys, ureters, and bladder.[5] In an analogous case,

[1] Ollivier (d'Angers), *loc. cit.*, t. i, p. 372.

[2] Brodie, 'Medico-Chirurg. Transactions,' *loc. cit.*

[3] See Stanley, 1st case. Urine strongly ammoniacal on the fifth day; 2d case, ammoniacal urine on the fourth day. 'London Medico Chirurg. Trans.' t. xviii, p. 1. Jeffreys: urine ammoniacal and sanguineous, the seventh day (Ollivier, d'Angers, *loc. cit.*, t. i, p. 322).

[4] Molendrinski, "Bruch des Zweiten Lendenwirbels," Langenbeck's 'Archiv,' xi Bd., 1869, p. 859.

[5] Case of W. Müller, see *ante*, 'Third Lecture,' p. 70.

reported by M. Brown-Séquard, on the authority of Dr. Maunder,[1] the urine was likewise found to be alkaline, a very short time after the accident. Cases of this kind are very interesting inasmuch as they show that a unilateral and very circumscribed lesion of the cord suffices to determine a more or less grave and generalized affection of the urinary passages.

Alike in spontaneous acute myelitis, of sudden invasion, and in hæmatomyelia, is the appearance of ammoniacal, sanguineous, and muco-purulent urine a fact of frequent occurrence, soon after the manifestation of paralytic symptoms. Thus the urine was already greatly altered on the fifth day, in the case of acute myelitis, which we have quoted from Dr. Duckworth;[2] on the sixth day, in that given by M. Joffroy.[3] It was ammoniacal the fourth day, in Dr. Gull's patient;[4] sanguineous the third, and purulent the ninth, in a case recorded by Herr Mannkopf.[5]

In the case of hæmatomyelia, recorded by M. Duriau,[6] the urine was ammoniacal and contained blood clots the fourth day; it presented the same character the sixth day and became gradually purulent in a case reported by Ollivier (d'Angers) on the authority of Monod.[7] In this instance, there was hemiparaplegia, consecutive on the presence of a hemorrhagic focus occupying a lateral half of the spinal cord. You will find, in the work of M. Rayer, the description of lesions, frequently grave, affecting the kidneys, the renal pelves, and the bladder, to which these changes in the urine should be attributed.[8]

Many of the observations, just cited, contain an item of information the importance of which cannot escape your notice. It is mentioned that the urine which until then was normal became, as I have said, ammoniacal, sanguineous, or muco-purulent, at the very time when eschars were being developed on the sacral region, and when the electrical contractility was beginning to grow feeble in the paralyzed muscles.[9]

[1] 'Journal de Physiologie,' t. vi, p. 152, 1863.
[2] See *ante*, 'Third Lecture,' p. 72. [3] *Idem*, p. 72. [4] *Idem*, p. 72.
[5] 'Berliner Klin. Wochenschrift,' t. i, No. 1, 1864.
[6] 'Third Lecture,' p. 72.
[7] Ollivier (d'Angers), *loc. cit.*, t. ii, p. 177.
[8] Rayer, 'Traité des maladies des reins,' t. i, p. 530 et seq. "According to my observations," Rayer writes, "in the diseases of the spinal cord, when the urine contained in the bladder is alkaline, it is so, not because of a decomposition difficult to be explained without atmospheric contact, and in *a short space of time*, but rather by a vice of renal secretion which should be attributed, in most cases, to *an inflammatory irritation of these organs.*"
As regards the description of alterations in the urinary passages, consecutive on acute affections of the spinal cord, consult, Engelken, *loc. cit.*, p. 12. Mannkopf, 'Bericht über die Versammlung zu Hannover,' p. 259; and 'Berlin. Klin. Woch.,' t. i. Compare, Rosenstein, 'Nierenkrankheiten,' 2 Ed., p. 287. Berlin, 1870.
[9] Ollivier (d'Angers) had already remarked that, in traumatic paraplegia, when the urine alters at an early period the eschars are found to form rapidly in the sacral region. *Loc. cit.*, t. ii, p. 37.



How are we to understand so rapid a development of the inflammatory lesions of the urinary passages after acute affections, spontaneous or traumatic, of the spinal cord? Manifestly, the paralytic retention of the urine cannot here be pleaded, at least not as the sole, nor even as the predominant, pathogenic element. Neither is it possible to attach great weight to the opinion[1] which would attribute the urine-changes, in such circumstances, to the introduction of unclean catheters, carrying vibriones. In point of fact, the introduction of vibriones into the bladder could only be a chance occurrence, whilst the appearance of ammoniacal, sanguineous, and purulent urine, in the course of acute myelitis is, like the production of eschars, what may be termed a regular fact.

The notorious insufficiency of the pathogenic conditions just enumerated, renders it at least highly probable that there is a direct action of the nervous system engaged in the production of the affection of the urinary passages which we are considering. The cause of this affection, as of the other trophic lesions which often show themselves at the same time, would therefore be the irritation of certain portions of the spinal centre, and more particularly, no doubt, of the gray substance.

## Theoretical Portion.

Gentlemen: In the foregoing series of studies, we have often had occasion to acknowledge that the development of the trophic disorders, ensuing after lesions of the nervous system, is not in general (contrary to a wide-spread opinion) the consequence of absence of action of different parts of that system. Far from that, these affections would result, in our view, from the irritation set up, under certain conditions, either in the peripheral nerves or in the nervous centres themselves. Thus, we find ourselves possessing a view, which is of primary importance to the pathologist, and you, without further explanation, can readily divine the practical deductions to which it may guide us.

But it must next be ackowledged that this wholly empirical notion marks only the first step taken towards the scientific knowledge of the phenomena, which observation has allowed us to establish. For, if we know the mode of initial alteration and its seat as well, there remains yet to be determined, in the first place, the means by which this lesion reacts upon the peripheral parts.

Evidently, this reaction is produced by means of the nerves, but that also, from a theoretical point of view, is an insufficient datum. It is necessary to endeavour to be more precise, and to seek what is the element, in that physiologically complex totality called a nerve, by which the transmission is operated, and also the mechanism of this transmission.

[1] Traube, 'Munk. Berliner Klin. Wochensch.,' p. 19, 1864.

I approach the question just raised, with an almost absolute certainty of not being able to answer it by rigorous arguments. Perhaps, I should have avoided it, desirous of not wasting your valuable time, if I were not convinced that it behooves us at least to show the inanity of a theory which professes to resolve it, and which to-day enjoys an almost uncontested sway.

You are not unaware, gentlemen, of the considerable influence which has been attributed to the vaso-motor nerves in the explanation of pathological phenomena. I am far from wishing to ignore the fact that a goodly number of these phenomena do, indeed, directly depend either on the dilatation or on the contraction of the smaller vessels, determined by nervous influence. But in so far as the trophic disorders which form the object of our studies are concerned, I hope that it will not be difficult to show, from a brief examination, that the *vaso motor* theory is altogether insufficient.

In order to attain this aim, I am induced to remind you of some of the experimental facts which have unveiled the functions of these centrifugal nerves whose ultimate ramifications go to animate the muscular coat of the smaller vessels. I shall, in the first place, recall the phenomena noticed when these nerves have been paralyzed in consequence of complete section, for instance.

Section of the vaso-motor nerves[1] has the immediate effect of producing a paralytic dilatation of the vessels to which they are distributed. Hence results a state of hyperæmia, termed *neuro-paralytic*, which has been especially well studied in cases of section of the great sympathetic nerve in the cervical region, but which is to be found with almost identical characters after a great number of lesions of the nervous centres or of the peripheral nerves. The consequences of this hyperæmia are, from our point of view, particularly worthy of interest. You know that the part answering to the divided nerve, presents a relative elevation of temperature, which appears solely to result from the afflux of a greater quantity of blood. You know that throughout the whole extent of the hyperæmic territory an exaltation seems also to ensue of all the vital properties of every element and every tissue. At least, the sensitive as well as the motor nerves, and the muscles themselves become more excitable,[2] and the latter preserve, longer than is usual after death has occurred, their proper contractility.[3] Nevertheless, in spite of these new conditions—and this is a point which requires to be set prominently forth—the accomplishment of the intimate acts of nutrition appears to be modified in nothing essential. Thus, in the experiments of M. Ollier,[1] agreeing in that respect with those of M. Claude Bernard, there is not found to supervene, in young animals after section of the great sympathetic in the neck, either an acceleration or an exaggeration in the growth of the parts of the face, even when subjected for months to neuro-paralytic hyperæmia. Nor does it appear that this hyperæmia, however intense or prolonged it may be, has ever the effect, save under exceptional circumstances to be hereafter mentioned, of determining by itself the development of inflammatory action. And if the experimenter intervenes and applies agents capable of provoking inflammation, the morbid process determined by this influence goes through its course in the hyperæmic parts as if under normal conditions; it offers no special characters, except, indeed, that the injured parts tend to heal with greater promptness.

It is true that, in reference to the latter points, M. Schiff professes a very different opinion. He affirms, in fact, that changes of nutrition originate in the hyperæmic parts, in cases of vaso-motor paralysis, under the influence of the slightest local mechanical irritant,[2] and that inflammation here readily takes on a destructive character.[3] But upon this subject he is in direct opposition to MM. Snellen, Virchow,[4] and O. Weber.[5]

In a recent experiment, besides, Herr Sinitzin states that after the extirpation of the superior cervical ganglion on one side, the introduction of a slender piece of glass into the cornea of the same side caused merely a very slight inflammatory reaction, sometimes scarcely noticeable; whilst on the opposite side, in the selfsame animal, its introduction caused, on the contrary, a most active inflammation with purulent infiltration of the cornea, iritis, panophthalmia, etc.[6] M. Claude Bernard, indeed, long since pointed out that ablation of the superior cervical ganglion appears to retard the manifestation of the nutritive disorders occasionally determined in the eye by section of the fifth pair of nerves,[7] and Herr Sinitzin has arrived at the same results in his experiments.

From this you may perceive that, contrary to the opinion of Professor Schiff, neuro-paralytic hyperæmia does not create in the parts it occupies, a peculiar predisposition to the production of trophic derangement. It would even seem that these parts are better able to resist the action of disorganizing causes and that any disorder set up there is more speedily repaired than elsewhere.

In man, so far as this question is concerned, little difference is to

[1] For the physiology and pathology of the vaso-motor nerves, consult, Vulpian, "Leçons sur l'appareil vaso-moteur," recueillies par C. Carville, Paris, 1875 (*Note to the second edition*).

[2] Brown-Séquard, 'Lectures on Physiology and Pathology,' Philadelphia, 1860, p. 1457.

[3] Brown-Séquard, *loc. cit.* Joseph, in 'Centralblatt,' 1871, No. 46.

[1] Ollier, 'Journal de la Physiologie,' t. vi, p. 108.

[2] Schiff, 'Physiologie de la digestion,' p. 235, t. i. 'Lezioni di Fisiologia,' Firenze, 1866, p. 35.

[3] Schiff, 'Digestion,' t. ii, p. 423.

[4] Virchow, 'Cell-pathologie,' 4 ed., p. 158.

[5] O. Weber, 'Centralblatt,' 1864, p. 148.

[6] Sinitzin, 'Centralblatt,' 1871, p. 161.

[7] Claude Bernard, 'Système Nerveux,' t. ii, p. 65, 1865.

be found occurring, from what is observed in animals. At all events, neuro-paralytic hyperæmia has been seen to persist for a long period in parts of the body, as for instance in the face, without any nutritive disorder ever supervening. M. Perroud has collected a certain number of cases of this kind, in a memoir read in 1864, before the Medical Society of Lyons. It suffices indeed, to glance at the numerous works which, of late years, have been published upon *Angioneuroses* to perceive that nutritive disorders are a rather rare accompaniment of neuro-paralytic hyperæmia. A new argument may, perhaps, be added in support of the thesis which we uphold. It is this: The elevation of temperature, tested by means of a thermometer, is, we have said, a phenomenon indissolubly linked with the existence of partial hyperæmias of neuro-paralytic origin. This local hyperthermia should necessarily exist in parts presenting the trophic derangements we have described, if these were really dependent on a neuro-paralytic cause. Now that does not happen, as a general fact. If a marked elevation of temperature has been many times observed in those regions of the body where an eruption of zona, consecutive on neuralgia, or neuritis had developed;[1] on the other hand, it may be said that irritative lesions of the peripheral nerves, in the conditions when they usually determine trophic disorders, appear to be accompanied rather by a lowering of the thermal standard than by its elevation. This lowering has been observed at every period of the nerve-affection; it has been noted near the commencement,[2] still oftener in the advanced stages.[3] When spinal lesions are concerned, it is true that occasionally the members subject to trophic troubles, rapid muscular atrophy, bullar eruptions, or eschars, exhibit a more or less marked elevation of temperature.[4] But at other times, perhaps in the majority of instances, this phenomenon is absent; thus it is absent in partial myelitis,[5] and in infantile paralysis;[6]—the same rule

[1] Horner, quoted by O. Wyss, 'Archiv der Heilkunde,' 1871. See note to p. 563. Charcot, 'Neuralgie du nerf cubital. Eruption du Zona sur le trajet du nerf affecté; examen thermométrique,' Thèse de Mougeot, Paris, 1867, p. 101.

[2] Folet, "Cas de Contusion du plexus brachial, observé par M. Lannelongue," 'Etude sur la température des parties paralysées,' Paris, 1867, p. 7.

[3] Hutchinson, *loc. cit.* Earle, "Medico-Chirurg. Trans.," vol. vii, 1816, p. 173. Yellowly, *id.*, t. iii. W. B. Woodman, in 'Sydenham Society's Transactions.' Translation of Wunderlich, 'On Temperature in Diseases,' p. 152. S. W. Mitchell 'Injuries of Nerves,' Philadelphia, 1872, p. 175. In two cases of nerve-wounds with "glossy skin," the region occupied by the trophic lesion was from one to two degrees warmer than the corresponding region of the healthy limb. But above this point, the thermometer marked one degree lower than on the healthy limb. H. Fischer, "Ueber trophische Störungen nach Nervenverletzungen an den Extremitäten," in 'Berliner Klin. Wochenschr.,' 1871, No. 13. The temperature of the limbs, on which the most varied trophic disorders occur, is, at first, higher than that of the healthy members, afterwards it is relatively lower; but there are many exceptions to this rule.

[4] Levier, "Cas d'Hematomyélie," *loc. cit.*

[5] Mannkoff, *loc. cit.*

[6] Duchenne (de Boulogne), *loc. cit.*, 3d edition, p. 398.



holds good for cases of slow evolution, such as, for instance, progressive muscular atrophy.[1]

You observe, from what precedes, that the trophic disorders connected with irritative lesions of the nervous centres may, in a considerable number of cases at least, occur without that elevation of temperature which should, I repeat, be necessarily present in all cases, if they really originate in hyperæmia, consecutive on paralysis of the vaso-motor nerves.

Hence it follows that neuro-paralytic hyperæmia and the production of trophic derangement are, in ordinary conditions, phenomena independent of each other. But as we suggested, a little ago, there are circumstances in which, contrary to the usual rule, local nutrition may receive a serious blow from the mere fact that the part has been withdrawn from vaso-motor innervation. This happens, as experiments attest, when the whole organism has been subjected to potent debilitating causes. Thus a vigorous animal has long had the great sympathetic nerve divided on one side of the neck; nevertheless, no injury has been experienced in the parts corresponding to the distribution of the divided nerve. But let the animal fall sick, or be deprived of food, then the scene changes immediately, and we see, says M. Claude Bernard, inflammatory phenomena ensue in that side of the face which corresponds with the experimental section. On that side, even without the intervention of any external agent whatever, the conjunctiva and the pituitary membrane rapidly begin to suppurate.[2]

It is legitimate to suppose that the animals in which Professor Schiff saw trophic lesions supervene, consecutively on neuro-paralytic hyperæmia, under the influence of the slightest mechanical irritation, had been suffering from the debilitating conditions noticed by M. Claude Bernard. In man, the same concurrence of circumstances ought necessarily to determine effects analogous to those observed in animals, and we may, indeed, question whether some of our trophic derangements are not really produced in this manner. Such is, perhaps, the case as regards the *acute bed-sore* of apoplectic patients. Here, in fact, the general condition is most unfavourable, and the gluteal eschar occupies precisely that side of the body which on account of the motor paralysis, presents a relative elevation of temperature, evidently connected with vaso-motor hyperæmia.[3]

However it be, this pathogenic interpretation can have but a

[1] Landois und Mosler, in 'Berliner Klinisch. Wochenschr.,' 1868, s. 45. For examples of depressed temperature supervening after spinal injury, see J. Hutchinson: "Temperature, etc., after crushing of the cervical spinal cord," 'Lancet,' pp. 713, 747. 1875. (S.)

[2] Claude Bernard, 'Physiologie du Système Nerveux,' t. ii, p. 535, Paris, 1858. 'Medical Times and Gazette,' p. 79, t. ii. 1861.

[3] See *ante*, Third Lecture, p. 62.