

LECTURE XIII.

HYSTERO-EPILEPSY.

SUMMARY.—Hystero-epilepsy. Meaning of this term. Opinions of authors. Epileptiform hysteria; hysteria with mixed crises. Varieties of hystero-epilepsy; hystero-epilepsy with distinct crises; hystero-epilepsy with combined crises, or *attaques-acces* (seizure fits). Differences and analogies between epilepsy and hystero-epilepsy. Diagnostic signs supplied by examination of central temperature in hystero-epileptic acme, and in epileptic acme. Epileptic acme; its phases. Clinical characters of hysteria, epileptic acme. Gravity of certain exceptional cases of hystero-epilepsy. Case recorded by Wunderlich 247

APPENDIX.

Case of paralysis agitans 261

LECTURES

ON

DISEASES OF THE NERVOUS SYSTEM.

PART FIRST.

DISORDERS OF NUTRITION CONSEQUENT ON LESIONS OF THE BRAIN AND SPINAL CORD.

LECTURE I.

DISORDERS OF NUTRITION CONSEQUENT ON LESIONS OF THE NERVES.

SUMMARY.—Preliminary observations. Object of these lectures: they shall be devoted to those diseases of the nervous system, and of the spinal cord, especially, which are most usually met with in the Salpêtrière Hospital. Nutritive disorders consequent on lesions of the cerebro-spinal axis and of the nerves. These morbid alterations may affect the skin, the connective tissue, the muscles, the articulations, the viscera. Their importance in relation to diagnosis and prognosis. Nutritive derangements consequent on lesions of the peripheral nerves. Slight influence (in the normal state) of the nervous system upon nutritive action. Passive lesions of the nerves and spinal cord do not directly produce disorders of nutrition in the peripheral parts. Demonstrative experiments. Influence of the irritation and inflammation of nerves or of nervous centres on the production of nutritive disturbances. Nutritive disorders consequent on traumatic lesions of nerves, considered specially. They arise not from complete but from imperfect sections or from contusions, etc., of the nerve. Cutaneous eruptions: erythema, *zona traumatica*, pemphigus, "glossy skin." Muscular lesions, atrophy. Articular lesions. Lesions of the osseous system: periostitis, necrosis. Disorders of nutrition consequent on non-traumatic lesions of the nerves; their analogy with those which result from traumatic lesions. Nutritive disorders affecting the eyes in cases of compression of the trifacial by tumour. Inflammation of the spinal nerves, consequent on vertebral cancer, on spinal pachymeningitis, on asphyxia by charcoal fumes, etc. Cutaneous eruptions (*zona pemphigus*, etc.), muscular atrophy, and articular affections, which, in such cases, are developed in consequence of the neuritis. Anæsthetic lepra, leprous perineuritis, lepra mutilans.

GENTLEMEN: Never without emotion, yet never without great gratification, do I inaugurate, each session, the series of lectures

which you have assembled to hear. On such occasions, indeed, I never fail to discover the friendly faces of former students, some of whom have attained professorial rank, and some of whom have already signalized their career by brilliant researches. Their presence affords me a great satisfaction, and I gladly seize the occasion to testify my gratitude.

It seems to me that the unusual number of those who have assembled here to-day is a convincing proof of the correctness of my belief when, five years ago, I ventured to maintain that this vast emporium of human suffering might one day become a seat of theoretical and clinical instruction, of uncontested utility.¹

It is true, gentlemen, that the field of observation before us does not embrace the entire of pathology. But, taken for what it is, who shall complain of its extent, or say that it is not vast? On the one hand, it offers for our study the ailments of the aged, which call for a share of our attention. On the other hand, amongst chronic diseases, it exhibits, under conditions peculiarly favourable to research, and gathered together in numerous array, those diseases of the nervous and of the locomotor systems which are so common, and consequently so interesting to the physician—diseases the pathology of which has begun, within the last twenty years, to emerge from the deep darkness which had previously covered it.

As for myself, gentlemen, I have never doubted that the Hospital of La Salpêtrière was destined to become, both for the diseases of old age and for many chronic disorders, an incomparable centre of instruction. All that was required to realize this idea was that certain modifications should be made in the internal arrangement of this institution, and I am happy to inform you that circumstances, at present, seem wholly favourable to our views.

The authorities have already, without any solicitation, placed under our care wards containing nearly one hundred and fifty beds, where we may study all the forms of epilepsy and of the graver hysterical affections. The Director of the *Assistance Publique* has also formed the project of opening in this hospital a dispensary specially destined for patients suffering from chronic ailments, and a ward to which a certain number of them should be admitted, temporarily, to undergo treatment.

When all these elements of study shall have been classed and organized with a view to scientific investigation and clinical instruction, I have no hesitation in saying that we shall possess at Paris an institution which, of its kind, can scarcely have a rival.² I hope to have soon the happiness of seeing this plan realized in all

¹ This lecture was delivered in May, 1870.

² This project has, unfortunately, not yet been made a reality (September, 1874).

its details. But if unforeseen circumstances should call me elsewhere, it would still yield me a deep gratification to see my successors crowning the edifice whose first foundations only I had been allowed to build.

Gentlemen, your time is valuable, and I do not desire that this preamble should extend too far. It is time to come to the special subject of these lectures. I purpose, then, to devote this session to the study of those diseases of the nervous system, and especially of the spinal cord, which are most usually met with in this hospital. As I feel it would be objectionable to plunge at once into technical details, it seems to me suitable to invite your attention to a question of general interest, and one which we shall encounter at every step in the course of our studies.

I.

Lesions of the cerebro-spinal axis frequently react upon different portions of the body, and produce there, by means of the nerves, various disorders of nutrition. These secondary affections constitute one of the most interesting pathological groups, and I shall therefore devote several sittings to trace out for you the principal features of their history.

The consecutive lesions in question may affect most of the tissues and may occupy the most diverse regions of the body; thus, we may find them in the skin, the connective tissue, the articulations, the bones, and even the viscera. They generally present, at least at the beginning, the characteristics of inflammatory action. Frequently they play in the drama of disease but an accessory part, being simply added on to the usual symptoms, hyperæsthesia, anæsthesia, hyperkinesis, akinesis, motor inco-ordination, etc. But were it only for the interest they have, when considered from the standpoint of pathological physiology, they should not be neglected.

Occasionally, however, these lesions assume an unmistakable importance in the eyes of the clinical observer, either because of the serious ailments which they cause, or because of their value as regards diagnosis or prognosis. Allow me to offer some examples in support of this assertion.

Last year I pointed out to you, and I shall return to this symptom again, that the sacral eschar which is developed in the course of apoplexy from cerebral hemorrhage or from softening of the brain allows us to lay down a prognosis of almost absolute certainty. The sacral eschars, the affections of the kidneys and of the bladder, which are produced with such rapidity in certain acute diseases and in the exacerbations of some chronic diseases of the spinal cord, are often the immediate cause of death.

An arthropathy, arising in the course of locomotor ataxia, may

deprive the patient of all future use of a limb which might otherwise have served him long.

Finally, these consecutive lesions of nutrition sometimes deceive the physician, who may mistake them for the disease itself. Such, for instance, are certain forms of progressive muscular atrophy which were formerly regarded as primary affections of the muscles themselves, and whose origin really lies in certain morbid alterations which have taken place in the gray matter of the spinal cord.

It would, I believe, be superfluous to multiply examples, for these observations should now suffice to indicate the interest which belongs to the study of such lesions of nutrition.

The power of producing, under certain morbid conditions, lesions of nutrition, in the peripheral parts of the body or in the viscera, is not an attribute of the brain and spinal cord alone. These centres share the privilege with the nerves which radiate from them. And it is to be observed that the consecutive affections produced by protopathic lesions developed in the most widely different regions of the nervous system present most remarkable analogies, in spite of some specific differences. Hence when the physician's attention has been called to such affections it is often a question of extreme difficulty to determine what portion of the nervous system was originally affected, and what is the true cause of the trophical lesion.

This consideration has induced me to limit our study to the lesions which are assignable to cerebral or spinal causes alone. These shall be, if you will, our objective point; but it seems useful to draw out, in parallel lines, the history of those trophical troubles which appear in consequence of lesions of the peripheral nerves. Is it not, indeed, one of the greatest advantages of the comparative method that it creates light by contrasts? In order to bound our field of study, we shall, however, only take into consideration those nutritive disorders which appear in the *peripheral domain of the suffering nerve*. The trophical changes which take place in consequence of reflex action, in a region more or less remote, and within the domain of nerves which have undergone no primitive lesions, constitute undoubtedly an interesting subject, but one which deserves to be treated specially.

II.

In hearing me speak, gentlemen, of the nutritive disorders which arise under the influence of lesions of the nervous centres or of the nerves, most of you, I am sure, have been immediately reminded of the corresponding problem which is debated in normal physiology.

There is nothing better established in pathology (as I hope to demon-

strate) *than the existence of trophical troubles consequent on lesions of the nervous centres or of the nerves*. Nevertheless, you are aware that the most advanced physiology teaches that, in the normal state, the nutrition of different parts of the body does not essentially depend upon the influence of the nervous system.

These statements appear contradictory, but the opposition is only in appearance and not in reality. This I shall endeavour to prove, and with that object I have to ask your permission to make a short incursion into the domain of experimental physiology.

You are aware that in order to show that the chemical acts of molecular renovation, which constitute nutrition, are not immediately dependent on the action of the nervous system, many kinds of arguments are adduced:—

1. The most complex acts of nutritive life take place in certain organisms without the intervention of a nervous system. Plants, for instance, and some of the lowest animals, such as certain protozoa, though unprovided with nervous systems, manifest great vital activity. Does not the embryo, it is also asked, perform all the acts of organic life, at a period when it as yet possesses no nervous element whatever?

2. They base another argument on the fact that certain tissues, even in the superior animals, are totally devoid of nerves and vessels. As instances, they refer to the epithelial layer and to cartilage, which if placed under pathological conditions will become seats of cell-proliferation—a plain proof that nutrition can take place there in a very energetic manner.¹

¹ . . . "The whole organic life of animals, *i. e.*, everything which goes on in them without the intervention of any sensation, or other mental act, may go on without the intervention of the nervous system and stands in no relation of dependence to any change in nervous matter; just as the corresponding functions of circulation, nutrition, secretion, absorption, go on in equal perfection in the lowest class of animals where no nerves are detected, and in the whole vegetable kingdom, where there is no plausible reason for supposing that nerves exist; . . . the nervous system lives and grows within an animal as a parasitic plant does in a vegetable."—'British and Foreign Med.-Chir. Review,' vol. iii, 1837, pp. 9, 10; and Carpenter, 'Principles of Human Physiology,' Philadelphia, 1855, p. 59.

The following is a succinct analysis of an essay in which M. Charles Robin has quite recently expounded the prevalent ideas of the present day in reference to the far from prominent rôle which the nervous system plays in the work of nutrition: "Those chemical acts which, in a living organism, constitute molecular renovation, otherwise called nutrition, are not under the direct influence of the nerves. There can be no question here of an influence of nerves over tissues, comparable to that of electricity upon chemical action. There exist no nerves which extend over the extra-vascular anatomical elements, such as the epithelium, like those nerve-tubes which proceed and are applied to the muscular fibrils. The cause of the movement of nutrition lies in the anatomical elements themselves. In plants, where no nervous system is found, we see the tissues suddenly swell, the cells increase and multiply. In the embryo, cells are formed, and increase and multiply before the appearance of any peripheral nerve-element. Nutrition is, therefore, a general property of anatomical elements, be they animal or vegetable. Secretion itself is a property inherent in anatomical elements, as

3. Finally, arguments bearing more directly on the subject are drawn from the arsenal of experimental physiology. You know that, after section of the nerves supplying them, and even when the spinal cord has been destroyed, the peripheral parts of the body, such as the muscles or the bones of a limb, will continue to live and be nourished for a considerable time, almost as efficiently as though they were under normal conditions. In such cases, lesions of nutrition do not make their appearance until a comparatively long period has elapsed. Even then they are almost always purely *passive*, and seem, in reality, due to the state of inaction to which the parts are condemned in consequence of the suppression of nervous influence. This belief is supported by the fact that lesions, displaying similar characteristics, present themselves when limbs are kept in a state of immobility, though the nervous system be not directly implicated. Such passive lesions, which we shall meet with in different paralytic affections, have nothing in common with the special trophical lesions which engage our attention. Generally they can be distinguished from them, objectively, by certain particular signs.

The special lesions are almost always characterized, at some period of their evolution, at least, by evidence of phlegmasic irritation. From the commencement they usually take on the appearance of inflammation; and they may, as we shall see, issue in ulceration, gangrene, and necrosis. There is, besides, one characteristic common to most of them, and that is the great rapidity of their development, after the lesion of nerves or nervous centres which provokes their manifestation. Sometimes they make their appearance with incredible quickness. Thus we frequently see eschars visible on the sacrum, the second or third day after the accident, in certain cases of fracture of the backbone, with compression and irritation of the spinal cord.

It may therefore be laid down as a general rule that there is a striking distinction between *passive lesions*, resulting from functional inaction alone, and *trophic disorders* which follow on certain lesions of the nervous centres. The former are slow of production, and usually manifest no symptom of inflammation; the latter often suddenly break out and generally present, at least at the commencement, more or less notable signs of phlegmasic irritation.

De Blainville and A. Comte have observed. In the lower animals, and in the case of animal grafts, it is evident that the nutrition of tissues is independent of the nervous system." "Disorders of secretion and of absorption, induration, softening, hypertrophies, and other alterations consecutive on nerve-lesions, are a consequence of perturbations of the circulation through the medium of the preceding (vaso-motor) nerves, directly affected by reflex action, and are not a consequence of the action of nerves which should, like electricity, have an influence over the molecular or chemical acts of assimilation and dis-assimilation in a zone of a certain extent beyond their surface."—'Journal de l'Anatomie,' etc., 1867, pp. 276-300.

Allow me, gentlemen, to remind you, briefly, of some of the experiments to which I have just alluded, and which tend to demonstrate that the nerves and spinal cord have no direct immediate influence upon the nutrition of peripheral parts.

1. One of the first of these relates to the section of the ischiatic nerve in mammalia. Schroeder van der Kolk, who was one of the earliest to make the experiment, attributed the disorders of nutrition which followed, with some rapidity in such cases, in the corresponding member, to the abolition of the action of the nervous system in consequence of the section. M. Brown-Séguard repeated the experiment, in 1849, on guinea-pigs and rabbits, and succeeded in showing that the trophical troubles which follow in the course of a few days, and which consist of tumefaction of the extremity of the member, ulceration of the toes, loss of the nails, only make their appearance because the animal is no longer able to preserve the limb, now devoid of movement and sensation in consequence of the section of the ischiatic, from the action of external influences, such as contact with the hard rough ground over which it is dragged. When the animal experimented on was placed under proper conditions, confined in a box, for instance, the bottom of which was covered with a thick layer of bran, there was no modification of nutrition to be remarked in the paralyzed member, except a more or less perceptible atrophy which, however, only made its appearance slowly in the course of time.¹

This atrophy, which follows the section of the ischiatic nerve, evidently results from the functional inaction to which the paralyzed limb is condemned. It affects not only the muscles, but also the bones and the skin, as J. Reid has already remarked. It will not be produced, even when the section has been complete, if, following the example of the physiologist quoted, you take care to pass a galvanic current daily through the muscles of the paralyzed member.

2. The complete section of the trifacial nerve, made within the cranium, presents results perfectly in keeping with those produced by section of the ischiatic. You are aware that the lesions of the eye which are found in animals subjected to this operation, and which were formerly considered by some physiologists as the consequence of the abolition of the nutritive influence of the trifacial, have, since the experiments of Snellen in 1857 and those of Büttner in 1862, been recognized as resulting from the consecutive anæ-

¹ Brown-Séguard, "Sur les altérations pathologiques qui suivent la section du nerf sciatique," 'Comptes-rendus des Séances de la Société de Biologie,' t. i, 1849; and 'Experimental Researches applied to Physiology and Pathology,' New York, 1863, p. 6.

After the section of a mixed nerve the atrophy of the muscles does not generally begin to show itself, in man and mammalia, by a slight emaciation, until the end of about a month. At the close of the second month it is more marked; it is very evident at the end of three months.—Magnin, *Thèse de Paris*, 1866, p. 19.

thesia which exposes the parts deprived of sensation to all kinds of traumatic causes. If the eye be protected after the section of the nerve, either by Snellen's method of tying the still sensitive ear of the same side in front of it, or by Büttner's plan of covering it with a piece of thick leather, the trophical troubles will not make their appearance in the cornea. A certain amount of neuro-paralytic hyperæmia in the iris and conjunctiva is, in short, the only phenomenon observable, after section of the trifacial, when the eye has been properly protected.¹

3. With respect to the spinal cord it seems demonstrated that a complete transverse section, or even its destruction for a certain length, when resulting in no considerable inflammation of the organ, is not immediately followed by troubles of nutrition in the paralyzed members. M. Brown-Séguard has shown that the ulcerations which appear, rather quickly, in the vicinity of the genital organs of mammalia and birds, after complete transverse section of the cord, are not direct consequences of the absence of nervous influx. They are produced by the prolonged pressure, and the contact of fecal matters and decomposed urine, to which these parts are exposed.

The hinder limbs of a young cat, which survived for nearly three months the complete destruction of the lumbar region of the spinal cord, were seen to develop in a normal manner. The functions of organic life seemed to proceed there in due accordance with physiological order; the secretion of claws and hair went on as in a healthy and uninjured animal.²

According to Valentin, when the posterior portion of the spinal cord has been destroyed, in mammalia and frogs, you will find the electrical contractility of the muscles of the hind members persist until death supervenes, that is to say, for several weeks or even several months after the operation.³

To sum up: in those animals whose spinal cords have been completely divided transversely, or extirpated in part, ulcerations and even eschars may form, principally in those regions subject to pressure; but it is always possible to attribute these lesions to the anæsthesia and paralysis, in consequence of which the animal lies constantly in contact with its excrements or unwittingly wounds itself, when dragging about its paralyzed members. As to the atrophy which supervenes, in the long run, in the paralyzed limbs after this operation, it arises solely, as in the case of the section of the ischiatic nerve, from the functional inertia to which they are condemned.

¹ See the experiments of M. Schiff relating to this subject in the thesis of M. Hauser, entitled 'Nouvelles recherches relatives à l'influence du système nerveux sur la nutrition,' Paris, 1858.

² Brown-Séguard, *loc. cit.*, pp. 14, 15, 16.

³ Valentin, 'Versuch einer Physiologischen Pathologie der Nerven,' 2 Abth., p. 43, Leipzig, 1864.

It follows from all these facts, which experimental physiology offers, that the abolition of the action of the nervous system, whether determined by complete section of the peripheral nerves or by destruction of a portion of the spinal cord, produces no other nutritive disturbance in the anatomical elements of the paralyzed members than what would be caused in the same elements by the influence of functional inertia, or prolonged inaction alone.

The discovery of the vaso-motor nerves and of the effects which follow the paralysis of these nerves was not destined to modify this formula, in any essential manner. It is in fact demonstrated at present, that neuro-paralytic hyperæmia, however far it may go, is never of itself alone sufficient to cause an alteration in the nutrition of tissues. Undoubtedly, this hyperæmia, as M. Schiff has pointed out, creates a certain predisposition to inflammatory action, which may supervene either spontaneously (to all appearance, at least) in the diseased animal or in consequence of irritative causes which would be comparatively trifling in a healthy organism. But lesions of nutrition of neuro-paralytic origin are nowise comparable to the trophical troubles which form the special object of our study, —they constitute a class apart. The latter, as we shall frequently have occasion to observe, may develop and accomplish their evolution without being preceded or accompanied by any of the phenomena which betray the paralytic state, or the contrary condition, of the vaso-motor nerves. At present we shall dwell no longer upon this subject, which we shall have an opportunity of referring to hereafter.

III.

If lesions, whose consequence is the abolition or suspension of the action of the nervous system, are impotent to produce in distant parts other nutritive disturbances than those attributable to prolonged inaction, *it is not thus as regards lesions which determine either in the nerves or nervous centres an exaltation of their properties, an irritation, or an inflammation.*

That, gentlemen, is a proposition of capital importance: it controls, in fact, the question which engages our attention. Although long since discovered by M. Brown-Séguard the principle upon which it reposes is still, if I do not mistake, too frequently overlooked both by physiologists and by pathologists.¹ We shall find, in due time and place, that human pathology presents many facts and decisive arguments in support of this proposition. On the other hand, we shall have less frequently to quote the results of experiments on animals. The especial reason of this paucity lies,

¹ "Note sur quelques cas d'affection de la peau, dépendent d'une influence du système nerveux," par J. M. Charcot, suivies de "Remarques sur la mode d'influence du système nerveux sur la nutrition," par le docteur Brown-Séguard, 'Journal de Physiologie,' t. ii, No. 5, p. 108, 1859.

undoubtedly, in the fact that the nervous tissue of animals seems much better able than that of man to resist the influence of the diverse causes of irritation and inflammation. All experimenters are aware that even the most serious traumatic lesions of the spinal cord or of the peripheral nerves do not readily produce, in the case of most animals, a myelitis or a neuritis, having some duration, which could be considered comparable with those developed so quickly in man, after the very slightest lesions.

The experiments which go to show that irritative lesions of the nerve-tissues are capable of determining various trophical troubles in the parts they supply, are, as we have said, few in number. They relate almost exclusively to the fifth pair.

The following is an abridged account of an experiment of Samuel:

In the case of a rabbit, two needles are applied to the Casserian ganglion and an inductive current produced; immediately ensue a more or less marked contraction of the pupil, and at the same time a slight injection of the vessels of the conjunctiva. The lachrymal secretion is greatly increased. The sensibility of the eyelids, conjunctiva, and cornea is augmented. After the operation, the contraction of the pupil persists, though not to the same extent, and the hyperæsthesia is still further increased. Inflammatory action generally sets in at the end of twenty-four hours; it increases in intensity during the second and third days and then gradually diminishes. All the stages of ophthalmia may be observed, from the slightest conjunctivitis to the most intense blennorrhœa. The exaltation of the sensibility still proceeds, and the hyperæsthesia may rise to such a degree that, at the slightest touch of the eye, the animal is seized with general convulsions. On the cornea a general opacity develops, and we find, besides, sometimes little ulcerations, sometimes a solitary oval-shaped ulcer occupying the middle portion of this membrane. In one case a small purulent collection formed in the anterior chamber. Hyperæmia excepted, no pathological alteration of the iris, neither adhesions nor changes of colour, are ever observed.

In every instance, hyperæsthesia of the ophthalmic branches of the fifth pair is specifically remarked. Hence it is plain that we cannot here, as in the experiments of Snellen and Büttner, invoke the aid of anæsthesia in order to explain the trophical troubles supervening in an imperfectly protected eye.¹

After an unsuccessful attempt to divide the trifacial in a rabbit, Meissner observed certain remarkable lesions of nutrition to ensue in the eye, which had preserved its sensibility. The author carefully points out that *these lesions were produced without having been preceded by any sign of neuro-paralytic hyperæmia*. A post-mortem examination revealed that the (internal) median part of the trifacial

¹ Samuel, 'Die Trophischen Nerven,' Leipzig, 1860, p. 61.

alone had been wounded by the instrument (a neurotome).¹ Schiff also cites four cases, in support of Meissner's observation, of partial lesions of the trifacial in the cranium, which were followed by inflammation of the eye, although its sensibility persisted.²

In Samuel's experiment trophical troubles arose in the eye, in consequence of faradaic irritation of the fifth pair. May we not infer that, in the experiments of Meissner and Schiff, the lesions of the eye were caused by phlegmasic irritation developed in the nerve in consequence of the imperfect section? In support of this opinion, I would remind you that incomplete sections, in man, are much more likely than complete sections to give rise to irritative action. This fact has long been familiar to surgeons. We may suppose that it holds good, at least to some extent, in the case of animals as well as of man.³

Let me place, side by side, with these facts several observations recorded in reference to the human organism, to which I shall afterwards recur. They relate also to the trifacial nerve. Like the preceding experiments, they show that irritative lesions of this nerve, spontaneously developed, may also, without being followed by anæsthesia, give rise to very striking nutritive disorders in the eye.

A woman, aged 57, whose case has been noted by Bock,⁴ experienced, for about a year, violent pains in the right side of the face. Though intermittent at first, they became afterwards almost continuous. The sensibility of the face never completely disappeared; slight pressure was, indeed, scarcely felt, but if the pressure was increased, it brought on acute pains. The conjunctiva of the right eye was injected. The cornea, slightly opaque all over, presented a hypertrophic ulceration in its lower part, of about two lines in length. Afterwards, the ulceration increased in depth, and the opacity of the cornea was augmented. Perforation at last ensued,

¹ G. Meissner, "Ueber die nach der Durchschneidung der Trigemini am Auge der Kaninchens Eintretende Ernährungstörung," 'Henle und Pfeufer's Ztsch.' (3), xxix, 96-104. 'Centralblatt,' 1867, p. 265. 'Gazette Hebdomadaire,' 1866, p. 634.

² Schiff, 'Henle's Zeitsch.' (3), xxix, 217-229. 'Centralblatt,' 1867, p. 655. 'Gazette Hebdom.,' 1867, p. 634.

³ This is not the interpretation which Meissner proposed for his experiment. He supposes that the innermost fibrils of the trifacial, which had alone been cut, in the case quoted, have a special action on the nutrition of the eye. He bases his opinion on this that, in three other cases where the trifacial had undergone incomplete section, but where the innermost nerve fibrils had been respected, no trophical troubles in the eye ensued, although this organ which had lost its sensibility was not protected from external agencies. We think that incomplete sections need to be repeated a considerable number of times before it is possible to pronounce a definite judgment on the interpretation proposed by Meissner.

⁴ Bock, 'Ugeskrift for Læger,' 1842, vii, p. 431. Extract in 'Hannover's Jahresbericht,' 'Muller's Archiv,' 1844, p. 47, and Schiff's 'Untersuchungen zur Physiologie des Nervensystems mit Berücksichtigung der Pathologie,' Frankfurt am Main, 1855, pp. 63, 64.