mann could have but a very narrow bearing. The influence of a mere mechanical cause cannot be invoked, at least not as a principal agency, even in cases where the arthropathy occupies the lower extremities. I have, in fact, taken care to point out, supporting my words by oft repeated clinical observations, that the articular affection in question is developed at a comparatively early epoch of the sclerosis of the posterior columns, and at a time when motor incoördination is as yet null, or scarcely manifest.

The clinical characters of our arthropathy are, besides, really special. Its sudden invasion, marked by the general tumefaction of the member; the rapid alterations of the articular surfaces; finally, its appearance at, as it were, a determinate epoch of the spinal disease with which it is connected, constitute so many peculiarities which are, if I err not, found together in no other articular affection.

But here is a more direct argument. Holding as we did that the arthropathy in question is a trophic lesion consecutive on the disease of the spinal cord, we yet could not think of connecting it with any of the common alterations of progressive locomotor ataxia —with sclerosis of the posterior columns, posterior spinal meningitis, or atrophy of the posterior roots of the spinal nerves. On the other hand, a minute examination of many cases had taught us that it was impossible to invoke a lesion of the peripheral nerves. It is in the gray matter of the anterior cornua of the cord that the starting point of this curious complication of the ataxia is to be found according to our belief.[1] It is not very rare to find the spinal gray matter affected in locomotor ataxia; but the lesion is then generally found in the posterior cornua. Now, it was quite different in two cases of locomotor ataxia, complicated with arthropathy, in which a careful examination of the cord has been made; the anterior cornua were, in both cases, remarkably wasted and deformed, and a certain number of the great nerve-cells, those of the external group especially, had decreased in size, or even disappeared altogether without leaving any vestiges. The alteration, besides, showed itself exclusively in the anterior cornu corresponding to the side on which the articular lesion was situated (Fig. 6). It affected the cervical region, in the first case, where the arthropathy occupied the shoulder; it was observed, a little above the lumbar region, in the second case which presented an example of arthropathy of the knee. Above and below these points, the gray matter of the anterior cornua appeared to be exempt from alteration.

It may be asked whether this alteration of one of the anterior cornua of the cord, which microscopical examination reveals, may

[1] See Charcot et Joffroy, "Note sur une lesion de la substance grise de la moëlle épinière, observée dans un cas d'arthropathie liée à l ataxie locomotrice progressive," 'Archives de Physiologie,' t. iii, p. 306, 1870.



not be a result of the functional inertia to which the corresponding member has been condemned on account of the articular lesion. This hypothesis must be rejected because, on the one hand, in both of our cases, the members affected by the arthropathies had preserved to a great degree their freedom of motion; and, on the other hand, the lesion of the gray matter differed essentially here from that which is produced after the amputation of a member, or the section of the nerves supplying it.

From what precedes, I hope to have made it appear at least highly probable that the inflammatory process, first developed in the posterior columns, by gradually extending to certain regions of the anterior cornua of the gray matter was able to occasion the development of the articular affection in our two patients. If the results obtained in these two cases are confirmed by new observa-

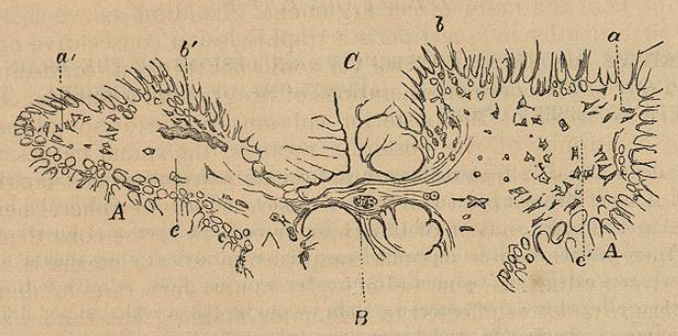


FIG. 6.—*A*, Right anterior cornu. *A'*, Left anterior cornu. *B*, Posterior gray commissure and central canal. *C*, Anterior median fissure. *a a'*, Anterior external cell-group. *b b'*, Anterior internal cell-group. *c'*, Right posterior external cell-group. The corresponding left group (*c*) is almost altogether absent.

tions, we should be naturally led to admit that arthritic affections connected with myelitis, and those observed to follow on cerebral softening are likewise due to the invasion of the same regions of the gray matter of the spinal cord. In cases of brain-softening, the descending sclerosis of one of the lateral columns of the cord might be considered as the starting point of the diffusion of inflammatory work.

MM. Patruban,[1] Remak,[2] and quite recently, Herr Rosenthal,[3] have observed in *progressive muscular atrophy*, arthropathies which by their clinical characters are closely allied with those of ataxic patients. This is nothing surprising, if we remember that a pri-

[1] Patruban, 'Zeitschrift für prakt. Heilkunde,' 1862, No. 1.
[2] Remak, 'Allgemeine med zinische central Zeitung,' March, 1853, 20 st.
[3] Rosenthal, 'Lehrbuch der Nervenkrankheiten,' p. 571. Wien, 1870. See also Benedikt, 'Elektrotherapie,' t. ii, p. 384.

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.