the nates of the member deprived of sensibility but not paralyzed in motion, became the seat of an eschar.[1]

I take the following case from an interesting work by Herr W. Müller:[2] in this instance the arthropathy is not mentioned; on the other hand, we find mention of rapid wasting of the muscles of the paralyzed limb, preceded for several days by a well-marked diminution of faradaic contractility. In all other matters, Müller's observation is in conformity with those of MM. Viguès and Joffroy. The case is that of a woman, aged 21, who received a stab with a knife in the back, at the fourth dorsal vertebra, the weapon, as the autopsy demonstrated afterwards, had completely divided the

[1] On account of the interest connected with it, we shall mention the principal details of this case.

The patient, Martin, aged about 40 years, was stabbed with a poignard, in the night of the 15–16th February, 1871. The weapon entered at the third dorsal vertebra. The direction of the wound is downward, backward, and to the right. Having been brought to hospital immediately after the wound, it was observed that, even then, the left inferior extremity was completely stricken with motor-paralysis, whilst the corresponding member on the other side showed nothing of the kind. February 16th, in the morning, the following note was made: Left lower extremity, complete motor-paralysis. The limb is perfectly flaccid; no trace of contraction, or rigidity, no spasmodic movements, nor subsultus. On the contrary, sensibility appears in the same limb to be exaggerated in all its modes; the least touch of the skin, especially near the foot, causes pain. Pressure has the same effect. A slight pinch or a tickle is followed by very painful sensations. Finally, the contact of a cold surface also produces painful sensations which the patient compares to those producible by a series of prickings. Right lower extremity: all the voluntary movements are perfectly normal, but *per contra*, the sensibility is almost completely destroyed. Complete analgesia; sensitiveness to touch almost null. The contact of a cold body is marked by an obscure dull prickling sensation. The insensibility is not limited, on the right, to the lower limb; it ascends to a level with the nipple. The urine and feces passed involuntarily.

February 24th (eighth day).—The same phenomena are observed; in addition it is noted that the left (motor-paralyzed) limb is warmer than the right. The patient complains of a sensation of constriction or rather of compression at the base of the thorax.

March 5th (seventeenth day).—The patient complains of troubled sight: the left pupil is more contracted than the right, and the vessels of the left eye are more voluminous and more numerous than those of the right eye. The evacuations have again become voluntary, for two days past. The state of the lower extremities is still unchanged.

March 13th (twenty-fifth day).—The right nates, since yesterday, has been the seat of vivid redness, and the epidermis has already fallen off from a part of the erythematous patch.

March 14th.—The derm is denuded to the size of a crown-piece on the right nates, near the sacrum: it is also ecchymosed (*acute bed-sore*). On Feb. 24th, it had been already remarked that some pain was felt when the left knee (motor-paralyzed limb) was moved; to-day, it is noted that this joint is swollen and red, and that it is, besides, the seat of spontaneous pains, exaggerated on movement (spinal arthropathy).

March 24th.—An ulceration, this day covered with granulations, has formed on the right nates, on a level with the ecchymosed patch. The swelling, redness, and pains have almost completely disappeared from the left knee.

[2] W. Müller, "Beiträge zur pathologisch Anatomie und Physiologie des menschlichen Rückenmarkes," Leipzig, 1871. Obs. i.

left lateral half of the spinal cord, two millimetres above the third dorsal pair. On the very day of the accident complete paralysis and hyperæsthesia of the left lower extremity was observed; the opposite limb was anæesthetic, but not paralyzed. On the second day it was found that the muscles of the paralyzed member and those of the lower part of the abdomen gave no reaction under the influence of faradaic stimulation, whilst, in the homologous parts of the opposite side, the electrical contractility remained normal. On the eleventh day an eschar was formed, occupying the sacral region and extending to the right gluteal eminence. On the same day, it was remarked that the paralyzed limb had notably wasted away, measuring about two inches less in circumference than the anæsthetic member. Death occurred on the thirteenth day. On a post-mortem examination, the borders of the spinal wound appeared tumefied, and of a reddish-brown colour; a thin purulent layer covered it. Below the wound the left lateral column, throughout its whole length, offered the anatomical characteristics of descending myelitis.

The simultaneous appearance of different trophic disorders noted in these cases, and in some others of the same kind, seem to indicate a common cause. This cause, to all appearance, is nothing other than the extension to certain regions of the inferior segment of the cord, of the inflammatory action originally set up in the immediate vicinity of the wound.[1]

That being admitted, it would seem legitimate, relying on the facts stated in the preceding lecture, to assign the rapid and general atrophy of the paralyzed muscles, noted in Herr Müller's case, to the invasion of the anterior cornu of the gray substance throughout the whole extent of the cord, whence nerves are given forth to the paralyzed muscles; the invasion in question taking place either progressively by direct downward propagation; or indirectly by the lateral columns. This lesion of the anterior cornu we shall mention, in a moment, to explain the development of the arthropathy described in the observations of Viguès and Joffroy. Now, with respect to the eschars, their appearance on the side opposite the spinal lesion tends to establish that the nerve-fibres (whose alteration, under such circumstances, provokes the mortification of the external tegument) do not follow the same course as those which influence the nutrition of joints and muscles, and that they, on the contrary, decussate in the cord in the same manner as the nerve-fibres subserving the transmission of tactile impressions.

[1] In a work, recently published, I have endeavoured to establish that, after wounds of the spinal cord, irritative lesions such as hypertrophy of the axis-cylinders, proliferation of myelocytes, &c., may be observed at some distance from the spinal wound, above and below it, scarcely twenty-four hours after the accident. Charcot, "Sur la tumefaction des cellules nerveuses, motrices, et des cylindres d'axe des tubes nerveux dans certains cas de myélite," in 'Archives de Physiologie,' No. 1, 1872, p. 95. Obs. i.

Another item of information which we get from cases of hemiparaplegia consecutive on a unilateral lesion of the spinal cord, is this, namely: acute bed-sore may show itself independently of all neuroparalytic hyperæmia, since we observe it forming upon that side of the body where the vasor-motor nerves are not affected.

c. I shall now mention the case where myelitis results, not, as in the preceding instance, from a wound or attrition of the spinal cord, but from indirect traumatic influence, such for example as an effort made in raising a weight. Acute bed-sore may, in cases of this kind, be produced as rapidly as though there had been fracture of the vertebral column, as the following fact recorded by Dr. Gull demonstrates:

A man, aged 25, by trade a labourer in the London Docks, felt, after lifting a load, a sudden pain in his back. He was able to walk to his home, about a mile off. On the morning of the second day after, his lower limbs were completely paralyzed; two days later, or four days after the accident, an eschar had begun to form on the sacral region, and the urine which flowed from the bladder was ammoniacal. The patient succumbed ten days after paralysis had set in. At the post-mortem examination, it was noted, after careful scrutiny, that the bones and ligaments of the vertebral column presented no lesion; in the neighbourhood of the fifth and sixth dorsal vertebræ the spinal cord was transformed throughout its whole breadth into a thick liquid, muco-purulent in appearance and in colour both brown and greenish.[1]

Following the example of traumatic myelites, spontaneous acute myelitis also very frequently determines the precocious formation of sacral eschars, principally when it sets in suddenly, and when the evolution is rapid. In order not to enter on lengthy details, in connection with this matter, I shall confine myself to indicating some examples illustrative of this class of cases. The sore has been noticed on the fifth day in a case reported by Mr. Duckworth,[2] on the sixth day in the case of a patient under the care of M. Woilliez, which M. Joffroy has communicated to me; on the ninth day in an observation of M. Engelken, on the twelfth day in another case related by the same author;[3] finally, in a case of a cervico-dorsal meningo-myelitis, published by MM. Voisin and Cornil, the eschar formed on the sixth day.[4] These examples might be easily increased.

Acute bed-sore frequently accompanies hæmatomyelia (which indeed appears to be, at least in a certain number of cases, only an accident of central myelitis); thus we found it in the case of Duriau,

[1] W. Gull, "Cases of Paraplegia," in 'Guy's Hospital Reports,' 1858, p. 189, Case xxii.
[2] 'The Lancet,' 6 Nov., 1869, p. 638.
[3] *Loc. cit.*, 'Pathologie der acuten Myelitis,' Zurich, 1867.
[4] 'Gazette des Hôpitaux,' 1865, No. 26.

already quoted, where mortification showed itself in the sacral region only four days after the appearance of the first symptoms.[1]

We may also observe rapid mortification of the skin of the sacral region supervening, even in spinal diseases of slow evolution, when a new course of active irritation intervenes on a sudden, or when acute inflammatory action is suddenly superadded to the initial lesion. Not only the exacerbation of partial sclerosed myelitis, but the sudden irruption into the rachidian cavity of pus emanating from an abscess, in the case of a patient suffering from vertebral disease may, as I can attest, determine the rapid formation of eschars. The same result would be likewise produced in case a tumour occupying the central portions of the cord should, by its presence, provoke the development of acute myelitis. Several examples of this kind are on record.[2]

If the evidences which we have collected here do not yet allow us to construct a pathogenic theory of acute bed-sore of spinal origin, they at least suffice, if I mistake not, to exhibit the principal conditions of the phenomenon. Manifestly, we must relegate to a secondary position the influence of pressure; and also that of vaso-motor paralysis which may be completely absent, as we have seen in relation to the hemiparaplegia resulting from the traumatic lesion of a lateral half of the cord. On the whole, the dominant and ever present fact is the active irritation of a more or less extensive region of the spinal cord—mostly showing itself, anatomically, by the characteristics of acute or superacute myelitis, and, clinically, by the assemblage of symptoms which are assignable to this kind of lesion. To explain the production of trophic disorders which issue in sacral mortification, here again it is not to absence of nerve-action that we should appeal, but to irritation of the spinal cord. This conclusion is in conformity with the experimental results which show that, in animals, the development of gangrenous ulcerations over the sacrum do not supervene on ordinary sections of the cord, but only in cases where inflammation has been set up in the neighbourhood of the spinal lesion.

It is scarcely probable that all the constituent parts of the cord are indiscriminately apt, under the influence of irritation, to provoke the development of acute bed-sore. The great frequency of this accident in cases of hæmatomyelia, and of acute central myelitis, where the lesion occupies chiefly the central regions of the spinal cord, seems to designate the gray substance as playing a predominant part in this respect. And this power is no doubt shared in by the posterior white fasciculi, for we know that the irritation of certain parts of these fasciculi has the effect of determining the

[1] 'Union Médicale,' t. i, 1858. p. 308.
[2] Amongst others see MacDowel's "Case of Paraplegia," in 'Dublin Quarterly Journal,' 1862.

production not only of different cutaneous eruptions, but also, though rarely indeed, that of dermal necrosis.[1]

On the other hand, it is perfectly established that all portions of the gray matter should not be indifferently accused; some of them, in fact may, as we have already suggested, undergo the gravest lesions, without acute bed-sore ever supervening. Such are the anterior cornua, whose lesions, *per contra*, have, as you are aware, a most decided influence on the nutrition of muscles and, as we shall soon see, on that of joints also.

Hence it is that sacral eschar is often absent in infantile spinal paralysis, and in adult spinal paralysis—diseases which are characterized anatomically by acute inflammatory lesions, systematically limited to the area of the anterior cornua; whereas, those other diseases, which affect the skin, depend upon irritative lesions occupying, either the central and posterior portions of the gray matter, or the posterior white fasciculi. From this particular point of view there is reason to recognize, in the spinal cord, the existence of two regions endowed with very distinct properties. Now, since these regions may be affected either separately or simultaneously, it follows that, in clinical practice, acute bed-sore and acute muscular atrophy will sometimes appear separately, and that they will sometimes, on the contrary, coexist in the same individual.

From what precedes, the influence of irritative lesions of the spinal cord upon the development of acute bed-sore seems to us placed beyond doubt. Herr Samuel has, however, advanced a contrary opinion; he thinks that the spinal cord does not play any part herein, and that the spinal ganglia or peripheral nerves are alone implicated. We shall mention elsewhere the arguments on which this theory is based; but we are now able to point out that it is in formal contradiction with what has been noted in the numerous cases of traumatic myelitis affecting an elevated part of the cord—the cervical region, for instance, or the superior portion of the dorsal region—cases where acute bed-sore supervenes in the sacral region, and certainly without the direct participation of the spinal ganglia, or of the peripheral nerves. The cases of hæmatomyelia, or of spontaneous central myelitis, followed by precocious eschars, are likewise adverse to the views of Herr Samuel.

It is not alleged, however, that the irritative lesions of the peripheral nerves, and perhaps also those of the spinal ganglia, may not sometimes have the effect of determining the rapid formation of eschars. No doubt, the examples published of dermal necrosis developed in consequence of a puncture, incomplete section, or compression of a nerve are rare enough; but many of them are thoroughly convincing.[2] In connection with this, I will relate the

[1] See *ante*, Lecture iii, § 1, p. 52.

[2] See amongst recent published facts, a case reported by Dr. W. A. Lanson ('The Lancet,' 30 Dec., 1871, p. 913), and two cases of Dr. Vitrac ('Union Médicale de la Gironde,' t. ii, p. 127, and 'Revue Phot. des Hôpitaux,' 1871).

case of a female patient which I have been recently studying at La Salpêtrière. She had, on the left side, an enormous fibrous tumour which compressed, in the pelvis, the roots of the ischiatic and crural nerves of the corresponding lower extremity. There had resulted a paretic state of this member, accompanied by acute pains running along the track of the principal nerve-trunks. One morning, shortly after the appearance of the first symptoms of compression, it was remarked that an eschar had rapidly formed near and to the left of the sacral region. The inner surface of the left knee, likewise, in a spot which had been pressed upon by the right knee for a considerable time during the night, in consequence of the attitude of the patient when asleep, presented some pemphigoid bullæ, full of a brownish liquid, which soon gave place to an eschar. Nothing of the kind was developed on the right knee. This is perhaps the place to mention that spontaneous zona which, in certain cases at least, is very probably connected with the inflammation of some nerve, may, according to the remark of Rayer,[1] occasionally issue in the more or less deep mortification of the skin. I have been often a witness to this fact, occurring among the aged persons in this hospital, and I have been many times able to satisfy myself that pressure on the spot occupied by the eruption did not here play an essential part. As to acute bed-sore of the breech, I am much inclined to believe that, in a certain number of cases, it should be attributed to an irritative lesion of the nerves of the cauda equina. A case recently published by M. Couyba, in his inaugural dissertation, may be cited as one of several examples of this class.[2]

III.

On Arthropathies of Cerebral or Spinal Origin.—Nutritive disorders consecutive on lesions of the nervous centres not unfrequently take up their seat in the articulations. The varieties presented

[1] Rayer, 'Maladies de la Peau,' t. i, p. 335.

[2] A young private in the Garde Mobile received a bullet wound, at the outposts of Clamart. The projectile had entered near the anterior extremity of the tenth rib on the left side, and had emerged on the right side of the vertebral column, about three inches from the spine, on a level with the second lumbar vertebra. Paresis, with acute hyperæsthesia of the lower extremities, followed. On the right gluteal eminence a bulla (which quickly gave place to an eschar) appeared on the fifth day after the accident. The eschar extended in a progressive manner, so as at last to cover the whole of the sacro-gluteal region. Death occurred on the nineteenth day.

Post-mortem.—A purulent mass covers the anterior and posterior surfaces of the cord, and extends from the cauda equina to the cervical region. The cord itself when examined, first, in the fresh state, next in numerous hardened sections, did not exhibit any alteration. On the other hand, a certain number of nerve-tubes in the nerve-filaments which form the cauda equina presented the anatomical characters of fatty granular degeneration.—Couyba, 'These de Paris,' 1871. Obs. xiii, p. 53.

by these articular affections, according to the nature of the cerebral or spinal lesions from which they arise, have led me to establish two principal categories.

A. The first comprises arthropathies of acute or subacute form, accompanied by tumefaction, redness, and sometimes by pain of a more or less severe character. This form was indicated for the first time, if I mistake not, by an American physician, Professor Mitchell,[1] who observed it in the paraplegia connected with Pott's disease of the vertebræ, in which, however, it is very rare, in my opinion.[2] It happens more frequently as a consequence of a traumatic lesion of the spinal cord, as we find from the sufficient evidence of the cases, above quoted, which have been recorded by MM. Viguès and Joffroy.[3] A case of concussion of the cord, related by Dr. Gull, supplies an analogous demonstration.[4]

Acute or subacute inflammation of the joints of paralyzed limbs may supervene also, in *spontaneous myelitis;* as examples of this class I may mention a case reported by Dr. Gull,[5] and another case which M. Moynier published in the "Moniteur des Sciences Médicales" for 1859. The second case relates to a young man, aged eighteen, who, after lodging for a long time in a damp place, and undergoing great fatigue, had presented all the symptoms of subacute myelitis. Paralysis of motion began to show itself on the 25th of January; it became complete on the 9th February. On the 23d of the same month, the skin of the sacral region presented an erythematous patch which gave place to an eschar, on the 5th of March. On the 6th of this month, there was severe pain in the right knee, which was swollen, and in which the sensation of fluctuation was perceptible. In addition, there was painful tumefaction of the tibiotarsal articulation of the same side. On the 9th of March, the knee had decreased in size, and on the same day, eschars made their appearance on the heels. The autopsy revealed a focus of ramollissement situated not quite two inches above the cauda equina.

Finally, in a case of central myelitis in a child, having its origin in the neighbourhood of a solitary tubercle situated in the cervical region of the cord, Dr. Gull records the formation of an intra-articular effusion, occupying one of the knees, at the time when the paralysis began to invade the lower extremities.[6]

It is remarkable to see these arthropathies, consecutive on the

[1] Mitchell, "American Journal of the Medical Sciences," t. viii. p. 55, 1831.

[2] I have, however, seen one knee become the seat of a subacute arthropathy in a woman suffering from paralysis consecutive on Pott's disease. This case has been recorded in the thesis of M. Michaud, "Sur la méningite et la myélite dans le mal vertébral," Paris, 1871.

[3] *Loc. cit.*, pp. 91, 92.

[4] Gull, "Guy's Hospital Reports," 3d series, t. iv, 1858. Case xxvii.

[5] Gull, *idem*, Obs xxvii.

[6] Gull, *loc. cit.*, Case xxii.

different acute and subacute forms of myelitis, frequently forming, when the muscles of the paralyzed limbs are beginning to waste away, or again when an eschar is being rapidly developed on the breech.

The *arthropathy of hemiplegic patients*, first described I believe in 1846, by Scott Alison,[1] afterwards by Brown-Séquard, and the

[1] Scott Alison, "Arthrites occurring in the Course of Paralysis," Note read before the Medical Society of London, Jan. 16, 1846, 'The Lancet,' t. i. p. 276, 1846. It is manifestly to the arthritis of hemiplegic patients, such as we have described it ('Arch. de Physiologie,' t. i), that the note of Dr. Alison refers. It is a characteristic of the affection to remain confined to the paralyzed limbs, and not to extend to the sound members. The affected joints are hot, swollen, and in some cases painful, either spontaneously or on movement made. The parts most frequently affected are the knee, elbow, wrist, hand, and foot. This form of arthritis seems to show itself chiefly in cases where the hemiplegia is consecutive on encephalitis or on brain softening. Two cases, selected from a number of others of the same kind, and cited as examples, deserve to be briefly recorded here:—

Case I.—A woman, aged 49 years, who had long enjoyed perfect health and had never suffered from any form of arthritic disease, was suddenly struck with hemiplegia; some days after, tumefaction and heat at the wrist of the paralyzed side set in, and a little later on, the knee and foot of the same side became swollen and painful in their turn. There was no œdema. The paralyzed limbs were rather rigid.

On *post-mortem* examination, partial softening of the brain was discovered. Each renal pelvis was filled with little calculi of uric acid.

Case II.—A man, aged 54, house painter, who had experienced several attacks of gout, was struck with sudden hemiplegia. Soon after the wrist, the hand, and the foot, became hot and swollen. The paralyzed limbs were rigid.

At the autopsy, the brain appeared softened, and a voluminous blood-clot was found in one of the lateral ventricles.

Dr. Alison endeavoured to explain the occurrence of arthritis in the course of (hemiplegic) paralysis, by showing that "the healthy relation between the living tissues and the materials of the blood was disturbed. Two morbid conditions gave rise to this disturbance, viz., a state of reduced vitality in the paralyzed parts, and the presence of exciting and noxious agents in the blood. In proof of this various facts were referred to, and the author related two singular cases of the inflammatory red line of the gums following the use of mercury, in paralysis of one side of the face, being strictly confined to the paralyzed side of the mouth. The paralyzed parts were in fact more delicate tests of poisons than parts in a state of health. In proof of the presence of exciting agents in the blood the gouty diathesis of the second case and the lithic acid calculi in the pelvis of the kidney of the first case, were adduced."

We, in our turn, would point out that, most certainly, these cases are altogether exceptional, as regards the question at issue, for most frequently, as may be understood from a perusal of the cases published in our work ('Archives de Physiologie,' t. i.) the arthritis supervenes in hemiplegic patients as a more or less direct consequence of the cerebral lesion, quite apart from all influence of gout, rheumatism, or other diethetic condition.

Hence, whilst acknowledging the accuracy of Dr. Alison's clinical descriptions, I am unable to endorse the pathogenic theory which he has proposed. I am, however, far from denying that the articulations of paralyzed members, in cases of hemiplegia of cerebral origin, may, as Dr. Alison holds, be particularly disposed to become foci of elimination for other agents previously accumulated in the blood. I myself communicated to the Société de Biologie, at the time of its occurrence, a case in which this particular disposition was very prominent. A woman, aged about 40 years, had been suddenly struck with right hemiplegia, three years before her admission into my wards. The paralyzed limbs were strongly

anatomical and clinical characters of which I have made known, belongs, if I mistake not, to the same category. In this second variety, as well as in the first, the arthropathies are limited to the paralyzed limbs and mostly occupy the upper extremities. They supervene, especially, after circumscribed cerebral ramollissement (*en foyer*), and, more rarely, as a consequence of intra-encephalic hemorrhage.

They usually form fifteen days or a month after the attack of apoplexy, that is to say, at the moment when the *tardy contracture* that lays hold on the paralyzed members appears, but they may also show themselves at a later epoch. The tumefaction, redness, and pain of the joints are sometimes marked enough to recall the corresponding phenomena of acute articular rheumatism. The ten-

contractured now and again, the several joints of these limbs, the knee especially and the foot, were the seats of tumefaction and pain. The patient, being aphasic, in a high degree, it was impossible to ascertain if she had been previously subject to gout or rheumatism.

At the autopsy, we found a vast ochreous cicatrix, the vestige of a focus of cerebral hemorrhage, situated exterior to the extra-ventricular nucleus of the corpus striatum. In most of the articulations of the limbs on the right side, which had been hemiplegic, the diarthrodial cartilages were incrusted towards their central parts with deposits of urate of soda, both crystallized and amorphous. The joints of the limbs, on the other side, presented no similar appearance. Some white striæ, which were found on microscopical and microchemical examination to be formed by urate of soda, were noticed in the kidneys.

It is undoubtedly most remarkable to find, in this case, that the gouty deposit forms exclusively in the joints of the paralyzed members; but, I cannot too often repeat that facts of this kind are exceptional, and, in any case, they have nothing in common, from a pathogenic point of view, with the ordinary arthritis of hemiplegic patients ('Cas d'Hubert,' see Bourneville, 'Etudes cliniques et thermometriques sur les maladies du système nerveux,' p. 58).

The merit is due to M. Brown-Séquard of having directed attention anew to the arthropathy of hemiplegic patients, and of having determined the organic cause, better than Dr. Alison had done. He thus expresses himself in a lecture published in 'The Lancet' ("Lectures on the Mode and Origin of Symptoms of Diseases of the Brain," Lecture i, Part ii, 'The Lancet,' July 13, 1861). After having admitted that the painful sensations, such as formication and prickling, which are experienced in the paralyzed members, in consequence of a cerebral lesion, result generally from a direct irritation of the encephalic nerve-fibres, he adds:—

"It is most important not to confound these sensations (which are referred sensations, like those taking place when the ulnar nerve has been injured at the elbow joint) with other and sometimes very painful sensations in the muscles or in the joints of paralyzed limbs. These last sensations very rarely exist when the limbs are not moved, or when there is no pressure upon them; they appear at once, or are increased by any pressure or movement. They depend upon a subacute inflammation of the muscles or joints, which is often mistaken for a rheumatic affection. This subinflammation in paralyzed limbs is often the result of an irritation of the vaso-motor or nutrition nerves of the encephalon."

Before M. Brown-Séquard, and before even Mr. Scott Alison, many physicians had already remarked the arthritis of paralytic patients, but without bringing out the interest connected therewith. Consult R. Dann, 'The Lancet,' t. ii, p. 238, 1841. Durand-Fardel, 'Maladies des Vieillards,' p. 131. Paris, 1854, Observation, Lemoine. Valleix, 'Guide du Médecin Praticien,' t. iv, 1853, p. 514. Grisolle, 'Pathologie Interne,' 2nd édition, t. ii, p. 257.

dinous sheaths are, indeed, often affected at the same time as the articulations.

I have shown that we have here a true synovitis with vegetation, multiplication of the nuclear and fibroid elements which form the articular serous membrane, and augmentation in number and volume of the capillary vessels which are there distributed. In intense cases, a sero-fibrinous exudation is produced, with which are mingled, in various proportions, white blood-corpuscles that may become abundant enough to distend the synovial cavity. The diarthrodial cartilages and ligamentous parts have not hitherto appeared to present any concomitant lesion perceptible to the naked eye. On the other hand, the tendinous synovial sheaths, in the neighbourhood of the affected joints, take part in the inflammatory process, and appear greatly congested.[1]

It is needless to insist upon the interest which pertains to these arthropathies as regards diagnosis,—articular rheumatism, whether acute or subacute, being an affection often connected with certain forms of cerebral softening, and one which, indeed, shows itself also, occasionally, after traumatic causes capable of determining shock in the nervous centres. On the other hand, many affections of the spinal cord are erroneously attributed to a rheumatic diathesis in consequence of the co-existence of these articular symptoms. The clinical characters which render it easy to recognize arthropathies correlated with lesions of the nervous centres, and which allow them to be distinguished from cases of rheumatic arthritis, are chiefly these:

1st. Their limitation to the joints of the paralyzed members.

2d. The generally determinate epoch in which, in cases of sudden hemiplegia, they make their appearance on the morbid scene.

3d. The coexistence of other trophic troubles of the same order, such as eschars of rapid formation; and (when the spinal cord is involved) acute muscular atrophy of the paralyzed members, cystitis, nephritis, &c.

B. The type of the second group is to be found in progressive locomotor ataxia. Allow me to fix your attention for an instant upon this species of articular affection, in which I take a paternal

[1] Charcot, "Sur quelques arthropathies qui paraissent dépendre d'une lésion du cerveau ou de la moelle épinière," 'Archives de Physiologie,' t. i, p. 396, Pl. vi, figs. 1, 2, 3, 4, 5, 6. Paris, 1868. The arthropathy in question should apparently not be confounded with the articular affection which has been described, in latter days, by Herr Hitzig of Berlin, "Ueber eine bei schweren Hemiplegien, Auftretende Gallenkaffection," in 'Virchow's Archiv,' Bd. xlviii, hft. 3 u. 4, 1869. This species appears, especially, when the hemiplegia is of comparatively old date, and the patients have been able to walk for some time; it chiefly occupies the shoulder-joint, and results principally from the displacement of the articular surfaces, occasioned by the paralysis of the muscles which surround the joint.

interest, all the more lively because the signification I attached to it has had to encounter many sceptics. And at first, a word as to the clinical characters of the *arthropathy of ataxic patients*.[1]

This disorder generally shows itself at a determinate epoch of the ataxia, and its appearance coincides, so to speak, in many cases with the setting in of motor incoördination.

Without any appreciable external cause, we may see, between one day and the next, the development of a general and often enormous tumefaction of the member, most commonly without any pain whatever, or any febrile reaction. At the end of a few days the general tumefaction disappears, but a more or less considerable swelling of the joint remains, owing to the formation of hydarthus; and sometimes to the accumulation of liquid in the periarticular serous bursæ also. On puncture being made, a transparent lemon-coloured liquid has been frequently drawn from the joint.

One or two weeks after the invasion, sometimes much sooner, the existence of more or less marked cracking sounds may be noted, betraying the alteration of the articular surfaces which, at this period, is already profound.[2] The hydarthus becomes quickly resolved, leaving after it an extreme mobility in the joint. Hence consecutive luxations are frequently found, their production being largely aided by the wearing away of the heads of the bones which has taken place. I have several times observed a rapid wasting of the muscular masses of the members affected by the articular disorder.

Ataxic arthropathy usually occupies the knees, shoulders, and elbows; it may also take up its seat in the hip-joint The anatomo-pathological information which we possess respecting it, is as yet very imperfect. However, one character is apparently constant, namely, the enormous wearing down which is exhibited in a very short space of time by the articular extremities. At the end of three months, this head of a humerus which I show you, and which belonged to a female patient in whom we were enabled to study the invasion of the arthropathy, was, as you may remark (Fig. 5), to a great extent destroyed. I would call your attention to the fact, that you do not find on this specimen, the bony burr around the worn articular surface, which would not fail to be present if this were a case of common dry arthritis.[3]

I now place before you in order to establish the contrast, a knee-joint also taken from a woman who presented the symptoms of ataxic

[1] Charcot, "Sur quelques Arthropathies," &c., p. 1. 'Archives de Phys.,' t. i, 1868.

[2] In some cases the cracking sounds have preceded, by several days, the appearance of the general tumefaction of the member; but, as a rule, the latter is the first symptom observed.

[3] Compare Charcot, "Ataxie locomotrice progressive, arthropathie de l'épaule gauche. Résultats nécroscopiques," in 'Archives de Physiologie,' t. ii, p. 121, 1869.

arthropathy, but in whom the articular affection was of much older date. Besides the wearing down of the articular surfaces which, as in the preceding case, is carried very far, you notice here the presence of foreign bodies, of bony stalactites, and, in a word, of all the customary accompaniments of *arthritis deformans*. These latter alterations, I repeat, were absolutely wanting in the first case. On this account, I am led to believe that they are nowise necessary, and that they are produced in an accidental manner, and to all appearance chiefly by the more or less energetic movements to which the patients sometimes continue to subject the affected members.

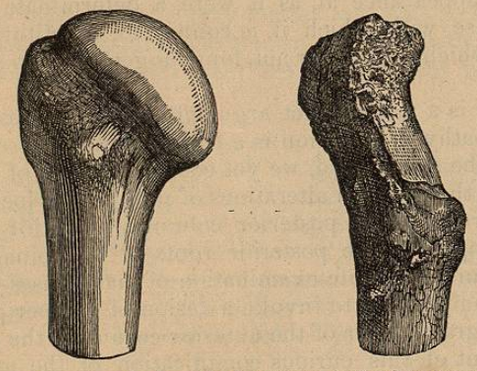

Fig. 5.—Upper extremity of a healthy humerus, and a humerus presenting the lesions of ataxic arthropathy.

I wish to confine myself at present to this indication of the most general features of the arthropathies of ataxic patients, for this is a subject which I propose to treat hereafter in more detail. What I have to say will suffice, I hope, to show that the articular affection in question is, itself also, the expression of trophic disorders directly dependent on the lesion of the spinal nerve-centre. But here are the principal arguments upon which I base my opinion.

I would point out, in the first place, the absence of all traumatic or diathetic cause of rheumatism or of gout, for instance, which might explain the appearance of the articular disease in the cases which I have studied. Herr R. Wolkmann[1] has said that the arthropathy of ataxic patients is simply the result of the distension of the articular ligaments and capsules, in consequence of the awkward manner of walking peculiar to this class of persons. The cases, which are now numerous, in which our arthropathy affected the upper extremities, and occupied either the shoulder or the elbow, are sufficient to prove that the interpretation proposed by Wolk-

[1] Canstatt's 'Jahresbericht,' 1868-1869, 2 Bd., p. 391.