that the patient had experienced, was followed on the same night by death. In the case of N—, whose record was compiled in my wards by M. Joffroy, five hours merely after the invasion of an apoplectiform attack, with incomplete loss of consciousness and general resolution of the members, the rectal temperature stood at 40.3° C. (=104.54° F.), and the pulse at 120. Next day the apoplectiform symptoms were dissipated, and at the same time the pulse and the temperature had returned to what they were in the normal state.[1]

If I have dwelt with some tenacity on the changes which the temperature of the body presents, in the apoplectiform and epileptiform seizures of general paralysis, and of some other cerebro-spinal affections, it is because, in my judgment, we find a characteristic therein which may, in certain cases, be profitably used in diagnosis. It is not necessary, I think, to enter into a long discussion in order to show how difficult it is, in presence of a patient who has just been stricken with apoplexy, accompanied or not by convulsions, to decide from the mere contemplation of external symptoms whether we have to deal with *true apoplexy*, resulting from the actual formation of a focus of cerebral hemorrhage or of ramollissement, or whether, on the contrary, we have before us a simple *congestive attack*. Well, an examination of the central temperature would supply, in such cases a decisive test. I have, in fact, demonstrated by repeated observations[2] that in true apoplexy, especially when it depends upon cerebral hemorrhage, the temperature constantly diminishes, some moments after the attack, and afterwards remains, generally for at least twenty-four hours, below the normal standard, even when intense and reiterated convulsive fits occur. Now, we have just seen that, in the so-called congestive attacks, the temperature, on the contrary, from the invasion of the first symptoms, rises above the physiological standard and tends to become gradually more and more elevated during the whole continuance of the attack.

Periods and Forms of Disseminated Sclerosis.

Gentlemen, after having considered, one by one, the different elements which compose the symptomatology of multilocular sclerosis where we have to deal with a complete case, one which has already arrived at an advanced stage of its evolution,—it is next proper to show, in a general view, how these elements are grouped

[1] 'Société de Biologie,' t. i, 5 série, 1869-70, p. 145.

[2] Charcot, "Note sur la température des parties centrales dans l'apoplexie liée à l'hémorrhagie cérébrale et au ramollissement du cerveau," in 'Comptes Rendus des Séances de la Société de Biologie,' t. iv, 4e série, 1867, p. 92. See also Charcot, 'Leçons sur la thermométrie clinique, publiées dans la *Gazette hebdomadaire*,' 1869, pp. 324, 742, 821; Bourneville, 'Études cliniques et thermométriques sur les Maladies du Système Nerveux,' Paris, 1870–73.

and arranged in the different phases and forms of the disease. The affection is, in truth, far from presenting itself clothed in all its attributes, at every epoch of its course. At the outset it may be constituted by the union of two or three symptoms only; and, besides, there are cases where the symptomatic series remains incomplete until the fatal end. Now, it is, especially, when the disease is yet at an early stage, or when it assumes an imperfect form, that it is important to know how to recognize it by the slightest indicia.

I have proposed to establish three periods in the progressive development of the disease. The first extends from the moment when the first symptoms appear to the epoch when the spasmodic rigidity of the members reduces the patient to almost absolute impotence. The second comprises the space, usually of considerable length, during which the patient, confined to bed, or barely able to take a few steps about the room, still preserves the integrity of his organic functions. The third commences at the moment when, all the symptoms of the disease becoming simultaneously aggravated, the functions of nutrition suffer in a manifest manner. We will take occasion, as regards this ultimate period, to notice the disorders which, in the common order of things, mark the last phase of the disease and accelerate its fatal termination.

I.

First period.—The mode of invasion and of concatenation of symptoms presents certain varieties which deserve to be pointed out to your notice.

Sometimes, the drama is begun by the cephalic symptoms. Thus, the patients commence by complaining of habitual giddiness, and more or less transient diplopia; little by little, difficulty of enunciation, and finally nystagmus, show themselves. The union of these symptoms would already constitute a sufficiently characteristic group, one which, even if tremor provoked by movement and paresis of the limbs were not superadded, should of itself enable us to establish a diagnosis, on strong probabilities.

But such is not the most common mode of invasion. Generally, the spinal phenomena first reveal themselves, and so common is this circumstance that during many months—nay, even for years—the patients may present no symptoms other than an enfeeblement, a more or less marked paresis of the lower extremities, displaying a tendency to become aggravated, in a slowly progressive manner, and to extend to the upper extremities. In such a case, the position of the clinical observer is necessarily an extremely difficult one. For, in short, paresis of the lower limbs is a somewhat trite symptom, one common to a crowd of different diseases; still, it shows itself in multilocular sclerosis, as you remember, with some peculiar

features which may indicate the right path to follow. Thus, however marked it may be—setting aside exceptional cases where the lesion predominates in the posterior columns—it is not accompanied by any trouble of sensibility, nor by any perceptible disorder of nutrition in the muscular masses. Add to this that, as a rule, there is no functional derangement of bladder or rectum. Finally, it is not rare to meet with *remissions*, and even with complete *intermissions*, which give rise to hopes of a decided cure.[1] But it is clear that these indicia, even with the aid of all the others, only supply very vague data. Certainty can hardly be secured unless the peculiar tremor, or some of the cephalic symptoms, are superadded to the spinal symptoms.

Hitherto, gentlemen, I have shown you the invasion and ulterior concatenation of accidents as slow and progressing in a uniform manner. That, in fact, is by far the most usual case; but it is important you should know that, in certain exceptional circumstances, the disease may set in suddenly and unexpectedly, or after a few preliminary symptoms, of little significance.

Thus, vertigo and diplopia having suddenly shown themselves, paresis and titubation may follow in a few days, so that the disease is thus, as it were, immediately established. This, to take one case amongst several, is what happened as regards the young woman named Vinch—, whom some of you may have seen in our wards.

Sometimes the beginning is marked, as in the case of one of Valentiner's patients, by an abrupt invasion of paresis in one of the lower extremities; or again, as occurred in M. Léo's case and in that of one of my patients, whose history M. Vulpian has related,[2] the invasion is inaugurated by an apoplectiform attack, preceded for some days or weeks, by vertigo and cephalalgia, and followed by temporary hemiplegia.

Finally, gentlemen, there is yet another variety, to which I must call your attention, where the invasion is marked by an affection

[1] In our memoir, we summarized a certain number of cases in which remissions were found so complete as to enable the patients, who had been paralyzed, to resume their occupations. (See *loc. cit.* obs. iv, ix, x, xi, &c.) In an observation recorded by M. Vulpian, which we also quoted (p. 139), there was a series of alternate ameliorations and aggravations. We shall briefly indicate them:

When the disease was still recent, there supervened, after an attack of small-pox, a quasi-complete recovery. This improvement lasted for three years. At the end of that time, the menses were suppressed; new, but slight, symptoms showed themselves, which disappeared on restoration of the catamenia. Two years after, the patient had an attack of jaundice, followed by new symptoms. These improved, but on bronchitis supervening, the paresis of the limbs reappeared in a more marked form, and, after successive remissions and recrudescences, became permanent.

Sometimes the remission is incomplete, and only affects certain symptoms, particularly incontinence of urine and of feces. In a patient, whose case was noted by Herr Baerwinkel, there was also a brief remission. (B.)

[2] Vulpian, "Note sur la Sclerose en Plaques de la Moelle Épinière," Obs. ii, 'Mémoires de la Société Médicale des Hopitaux,' 1869.

which is mostly regarded as foreign to the principal disease, although it is, in my opinion, intimately bound up with it, on the contrary, by a link not recognized until now. I allude to the *gastric* or *gastralgic crises*, whichever you please to call them, that are occasionally very severe, and are accompanied by lypothymia, by repeated vomiting, &c. These crises have often opened the drama, and been quickly followed by the usual symptoms of multilocular sclerosis; it is not rare, also, to find them several times recurring and intermingling with these symptoms, during the early stage of the disease. Of this class, a case reported by M. Liouville[1] and that related by Herr Zenke furnish good examples. These accidents are all the more worthy of notice inasmuch as we shall find them again, with nearly the same characters, in other forms of sclerosis of the spinal cord, and particularly in fasciculated posterior sclerosis (*locomotor ataxia*), but chiefly in its initial phases. In such a case, these gastric crises, coinciding or alternating with the fulgurant pains of the limbs, may actually be, along with diplopia and perhaps a little titubation when the eyes are closed, the only symptoms of the disease in question, whose true nature is then too often misunderstood.[2] These same gastric crises are found, as my friend Dr. Duchenne (de Boulogne) and I have observed, in the form of *subacute* or *chronic central myelitis*, which reproduces the symptoms of *general spinal paralysis*. But I do not wish to delay any longer on this subject, which I intend soon to resume and to discuss in detail, as its importance deserves.

II.

Second period.—In general, from the close of the first period, multilocular sclerosis shows itself arrayed in most of the symptoms which characterize it. These symptoms become aggravated and intensified during the second period, and spasmodic contraction of the limbs is superadded, either with or without the accompaniment of spinal epilepsy, in consequence of which the patients who, until then, had been able to walk or hobble, with more or less difficulty, are thenceforth rendered almost quite powerless, and definitely confined to their rooms or beds. The contracture which marks the commencement of this period is almost always a very tardy symptom; it seldom shows itself till two, four, or even six years after the appearance of the first accidents of multilocular sclerosis.

[1] 'Mémoires de la Société de Biologie,' 5e série, t. i, p. 107, Paris, 1870.

[2] See what M. Charcot has said, in reference to this subject, in his lectures delivered at La Salpêtrière in 1868 (Dubois, 'Etude sur quelques points de l'ataxie locomotrice,' Paris, 1868, "Des crises gastriques," p. 56; 'Leçons sur les anomalies de l'ataxie locomotrice,' 1873, leçon ii, p. 32).

III.

Third period.—The commencement of this final period is marked, as I mentioned to you, by the progressive enfeeblement of the organic functions; inappetency becomes habitual, diarrhœa frequent, and soon a general emaciation supervenes which grows more and more evident.[1] At the same time, there ensues an aggravation of all the symptoms proper to this disease, the obnubilation of the intellect proceeds even to dementia, the difficulty of enunciation is carried to its extreme, and the patient can only utter an unintelligible grunting; then the sphincters become paralyzed, and it is not rare to find the mucous coat of the bladder affected with ulcerous inflammation. Then, on the sacral region and on all points of the lower limbs submitted to prolonged pressure, eschars appear which occasionally assume enormous dimensions, and, consecutively, comes the whole series of accidents which depend on this complication, purulent burrowing sores (*fusées*), purulent or putrid poisoning, etc. Death follows without delay.

In most cases the patient's existence may be abridged by some intercurrent disease;[2] pneumonia, caseous phthisis, and dysentery may be numbered amongst the most frequent of these terminal affections.[3]

[1] At this period of the disease, especially, we notice the supervention of disorders which may, perhaps, be classed among trophic troubles. Such are—1st, softening of the vertebræ, of the trochanters, of the head of the tibia, of the bones of the tarsus, &c. (Bourneville et Guérard, *loc. cit.*, cas du Docteur Pennock, p. 83); 2d, a cyphosis and (right) scoliosis, mentioned in one of Friedreich's cases (B. et G., *loc. cit.*, pp. 213 and 214); 3d, an effusion of liquid into the two femoro-tibial articulations (Obs. de M. Malherbe). (B.)

[2] In the cases which have since been published, we most usually find the terminal diseases indicated by M. Charcot. It follows from the statistics we have collected that pulmonary diseases (pneumonia, purulent pleurisy, tubercular phthisis) are by far the most prominent. We should also mention the occurrence of *acute bed-sore*, of *pyelo-cystitis* (one case), and of *œdema glottidis* (one case). (B.)

[3] In this manner, the patient Vauthier (the subject of the preceding lecture) succumbed, and the patient Bezot, who long occupied bed No. 10, Salle St. Luc. We shall rapidly summarize the principal facts of their clinical history:—

I. Vauth—(Josephine C.) was admitted March 21st, 1867, to M. Vulpian's wards, and died January 7th, 1871 (aged thirty-two), in M. Charcot's charge. From fourteen to twenty-one years of age, she suffered from vertigo followed by vomiting. Pregnancy, at twenty-one, put an end to vomiting. Disseminated sclerosis showed itself at the age of twenty-three years, six months: weakness of the lumbar region, very great fatigue of the lower limbs, lancinating pain in the right leg, enfeeblement of the sight, diplopia. At twenty-five years, feebleness of the arms, which are occasionally affected by pains.

1867.—Nystagmus, diplopia. Integrity of the muscular masses, loss of idea of position as regards lower limbs. Paresis and tremor of the upper extremities. Tactual sensibility largely lost everywhere. Momentary improvement under nitrate of silver.

1868.—The patient can no longer stand erect; the symptoms are more marked on the right side than on the left; the tremor of the upper extremities has augmented. Frequent fulgurant pains, especially in the left half of the face. Fits of giddiness coming on at close intervals. Nystagmus more marked. In May,

I have reserved for special mention the appearance of some symptoms of *bulbar paralysis*, because they may, by an abrupt aggra-

M. Vulpian administered two pills of 0.025 gram. (or nearly $\frac{1}{3}$ grain) of extract of Calabar Bean. Soon after, a fit of weakness, tremor exaggerated, cold sweats, pallor of the face (these phenomena are, perhaps, due to the Calabar Bean). From July, three pills of Calabar Bean. In November, M. Vulpian suppresses the Calabar Bean, and as incontinence of urine has latterly supervened, he prescribes three pills of 0.03 gram. (or nearly $\frac{1}{2}$ grain) of extract of Belladonna. The incontinence of urine, after presenting some transient improvements, ceased altogether in the course of December.

1870 (January).—Psychic disorders (see *ante*, p. 160). In the course of this year the symptoms noted augmented in severity; and, besides, symptoms of bulbar paralysis made their appearance. These became rather rapidly worse, and the patient died, as it were asphyxiated, Feb. 7th, 1871.

Autopsy.—Numerous sclerosed patches found to exist in the brain and spinal cord. On account of the *ataxic symptoms* presented by the patient, the lesions of the spinal axis deserve mention. There were sclerosed patches throughout the whole length of the lateral columns. As to the *posterior columns*, they are affected nearly throughout, but, principally, from the lower extremity of the dorsal region upwards. Fig. 15 represents the lesions observed on a section taken from the upper part of the lumbar region. At this level the posterior columns are completely invaded (fig. 15, *c*), but especially affected in the mid-region. The lateral columns are comparatively less injured.

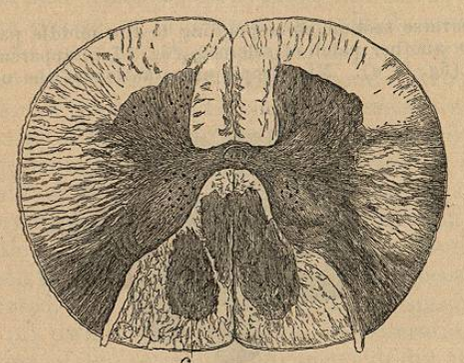

Fig. 15.—Representation of the lesions observed on a section taken from the uppermost portion of the lumbar region. The posterior columns are invaded throughout their breadth, and the lesion predominates in their middle region.

II. Bez—(Pauline), aged thirty-five, child's nurse, admitted Feb. 17th into M. Charcot's wards. To the ordinary symptoms of disseminated sclerosis were added, about the month of May, dyspnœa and dysphagia. The difficulty of deglutition compelled the patient to eat very slowly. Return of food, through the nasal orifices, was not observed until near the end. The patient died of asphyxia, June 12th, without any râles having been noticed in the lungs.

Autopsy.—Sclerosed patch on the chiasma of the optic nerves, invading the tractus opticus. Sclerosed patch in the ventricles and in the centrum ovale. In a section made a centimètre above the inferior border of the protuberantia annularis, on a level with the apparent origin of the trifacial nerve, a large and irregular patch of sclerosis is found (fig. 16, *b' b'*).

vation, precipitate the course of events, and induce the fatal termination, even before the manifestation of the phenomena of the final period. Contemporaneously with increased difficulty of utterance, there appears a difficulty of deglutition which, though transient at first, soon becomes permanent. Then, from time to time, paroxysms of dyspnœa show themselves, of less or more gravity, and death may supervene during one of these fits. I have recently observed two cases which terminated in this manner. On a post-mortem examination it was seen, in both these cases, that a patch of sclerosis had invaded the floor of the fourth ventricle, where it enveloped the originating nuclei of most of the bulbar nerves. (See foot-note 3, p. 176.)

After the details which I have laid before you, it seems useless to undertake the particular description of the different *forms* which multilocular sclerosis may assume. The *cerebral* and *spinal* forms correspond to an incomplete invasion of the nervous centres by the sclerosis; it is if you like, the disease arrested in its development, in its progress either of ascent or of descent. The symptomatic series is, therefore, as it were, curtailed; but the symptoms, considered singly, are not modified. The first, or cerebral, form is very rare; the second, or spinal, is, on the contrary, very frequent; but

Another transverse section, corresponding to the middle part of the corpora olivaria, reveals another patch of sclerosis (fig. 17 *c*) apparently involving the pneumogastric (fig. 17 *a*). Microscopic examination of the nerves showed the

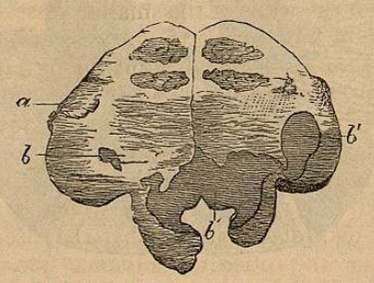

Fig. 16.—*a*, Pneumogastric; *b*, small sclerosed patch; *b'*, large sclerosed patch.

existence of numerous fatty degenerated tubes in the hypoglossal nerve, and traces of irritation in Schwann's sheath in the pneumogastric nerve. As to the other organs, and particularly the pharynx, the larynx, and the lungs, they were all healthy.

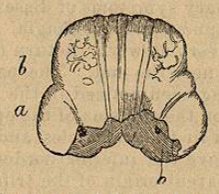

Fig. 17.—*a*, Pneumogastric nerve; *b*, hypoglossal nerve; *c*, sclerosed patch.

on the whole the *cerebro-spinal* form constitutes the normal type, that which we most often meet with in practice.

Cerebro-spinal multilocular sclerosis completes, generally speaking, its whole evolution in the space of from six to eight years;[1] this establishes another contrast between it and paralysis agitans, the normal duration of which is much longer. The spinal form usually gives the most lengthy respite; it may not terminate its course for a space of twenty years, or even longer still.[2]

Pathological Physiology; Etiology; Prognosis, and Treatment.

In order to conclude this study, gentlemen, it remains for me to discuss the pathological physiology, the etiology, and finally the therapeutical treatment of multilocular sclerosis of the nervous centres. Unfortunately, the facts and documents which refer to these different subjects are few in number, and as yet mostly imperfect, so that I shall be compelled to confine myself to a few summary observations.

A. The cause of the very singular mode of arrangement under which the sclerosed islets are distributed in different parts of the central nervous system, is, at present, completely unknown to us. Herr Rindfleisch[3] has stated that the starting-point for the formation of sclerosed foci resides in the vascular system. According to him, the inflammation of the walls of the smaller vessels, always to be met with in the centre of foci during the process of formation, would be the initial fact; from this central point the irritation is propagated to the reticulum of the neuroglia, whence it radiates in all directions. It is evident, however, that this explanation only sets the difficulty a little farther back. Besides, this predominant part accorded to vessels in the evolution of the morbid process is anything but demonstrated. I am even very much disposed to believe, judging from my own observations, that the alterations of vascular tissue and those of the reticulum advance side by side in parallel lines, without acting reciprocally on each other. Be this as it may,

[1] It is rather difficult, at present, to ascertain the mean duration of disseminated sclerosis. In a first collection (Bourneville et Guérard, *loc. cit.*, p. 148), comprising seventeen cases, we found a mean of from eight to ten years. In another, including thirteen new cases, we found a mean of seven and a half years. The minimum of the duration of the disease was one year (case of M. Malherbe, in 'Journal de Médecine de l'Ouest,' 1870, p. 168, and Buschwald, "Ueber Multiple Sklerose des Hirns und Ruckenmarks," in 'Deutsches Archiv für Klin. Medicin,' c. x, fas. iv und v, p. 478, 1872). The maximum is from sixteen to seventeen years. (B.)

[2] In three cases of disseminated sclerosis, with predominance of lesions of the posterior columns, the disease lasted fourteen, twenty-one, and twenty-eight years. (Bourneville, 'Nouvelle étude sur quelques points de la sclérose en plaques disséminées,' 1869.)

[3] Rindfleisch, "Histol. Detail zu der Grauen Degeneration von Gehirn und Ruckenmarks" ('Virchow's Archiv,' 1863, t. xxvi, p. 474).

the question arises whether, if the position of the sclerosed islets, in the different portions of the nervous system, be given, we can deduce therefrom the production of phenomena which, in their totality, constitute the symptomatology of disseminated sclerosis? This is, at least to some extent, possible. We have already shown you that the motor inco-ordination, the loss of sense of position and the fulgurant pains which are found in a certain number of cases, may, in such cases, be attributed to the sclerous invasion of the posterior columns of the cord for a certain altitude. On the other hand, the customary predominance of sclerosed patches along the course of the antero-lateral columns accounts, as I shall soon demonstrate to you, for the almost constant existence of the paresis or of the paralysis of the limbs, which is sooner or later followed by permanent contracture. The nystagmus and the difficulty of enunciation are correlated with the habitual localization of nodules in the substance of the protuberantia annularis and the bulbus rachidicus. But there are a large number of other symptoms, the interpretation of which presents much greater difficulty. Such, for instance, is the peculiar tremor which is manifested during certain attitudes of the body, and in the execution of intentional movements. I have expressed the opinion that the long persistence of the axis-cylinders, deprived of medullary sheathing, in the midst of the foci of sclerosis, probably plays an important part here. The transmission of voluntary impulses would still proceed by means of the denuded axis-cylinders, but it would be carried on irregularly, in a broken or jerky manner, and would thus produce the oscillations which disturb the due execution of voluntary movements.

This resistance of the axis-cylinders is certainly not a phenomenon exclusively pertaining to multilocular induration; but it is here manifested in a more marked manner than in other forms of sclerosis of the nervous centres. It may also be quoted, I think, to account for the slowness with which the paretic symptoms advance in disseminated sclerosis, and for the long space of time which elapses before they give place to complete paralysis and permanent contracture.

B. What is known in reference to the conditions that preside over the development of disseminated sclerosis comes to very little. It seems, however, to be established at present, that the disease is far more common in females than in males. Thus, of all the instances which I collected in my first treatise, only three or four related to men. The cases which have since been published have not modified this result in any perceptible manner. Adding to the eighteen cases which are mentioned in the monograph of MM. Bourneville and Guérard, sixteen new cases, we get a total of thirty-four cases, of which nine relate to males, and twenty-five to females.

Judging from the same records, it follows that this is a disease of youth, or of the first half of adult age. It has been observed in patients aged fourteen, fifteen, and seventeen years,[1] but it seems most frequently to set in between twenty-five and thirty years. It rarely makes its appearance after thirty. On the other hand, forty years seem to be the ultimate limit of life for patients affected by disseminated sclerosis.

With respect to the influence of hereditary predisposition, we have only one case to mention in which it seems to have played a certain part. This example has been communicated to us by Dr. Duchenne (de Boulogne).

In the pathological antecedents of the patients themselves we generally find nothing but vague indicia. Hysteria was present in some cases, but in most we only find mention made of ill-determined neuropathies, occasional hemicrania, or neuralgias.[2]

Amongst *occasional causes*, we frequently find mention made of the prolonged action of moist cold.[3] In one case, the first symptoms are alleged to have appeared a short time after a fall.

[1] In a work by M. Leubé ("Ueber multiple inselformig Sklerose der Gehirns und Ruckenmarks," in 'Deutsches Archiv,' 8 Bd., 1 heft, 1870, p. 14) we find the record of the case of a child who presented the first symptoms of disseminated sclerosis at the age of seven years. She died aged fourteen years and six months. Summary: slight nystagmus; right facial paralysis; marked ataxia of the extremities, especially on the left side; tremor of the head; difficulty of utterance; atrophy of the legs. *Autopsy*: sclerosis of the pons Varolii and annexes, almost general on the right, disseminated on the left. The cerebrum and cerebellum present, in their cortical layers, a double degeneration, whitish-yellow, or steel-gray, partly diffused, partly disseminated in patches. In the cord, and principally in the medulla oblongata, the sclerosis occupies, firstly, the posterior columns, next the lateral columns, and finally the anterior columns. (B.)

[2] There is, however, an etiological cause which deserves mention, namely, the influence of certain acute diseases on the development of sclerosis. The following facts are given in support of this assertion.

1st. In a case given by Erbstein ('Deutsches Archiv für Klinische Medicin,' t. x, fasc. 6, p. 596) disseminated sclerosis set in during convalesence from typhoid fever. The patient, then, suffered from debility of the lower extremities, and a difficulty of enunciation; the words were scanned, and the pronunciation was somewhat indistinct and monotonous.

2d. A patient in M. Charcot's charge, Nic—(Julie) noticed a certain degree of weakness in her lower limbs, on recovering from an attack of cholera. Some short time after, she had an attack of typhoid fever, after which the feebleness of her limbs augmented in a slow but continuous manner, to such an extent that she was soon obliged to use a cane. (A. Joffroy, 'Mémoires de la Société de Biologie,' 1869, p. 146.)

3d. In the case, recorded by MM. Fontaine and Liouville, it is mentioned that the first signs of sclerosis were preceded by copious bilious vomitings, which lasted from ten to fifteen days. (H. Liouville, in 'Mémoires de la Société de Biologie,' 1860, p. 107.)

4th. Finally, we will mention the case of a woman named Dr—(Hortense), in whom the first symptoms of disseminated sclerosis appeared soon after she had had a severe attack of smallpox. (B.)

[3] A patient, according to Herr Baerwinkle, experienced a difficulty in executing movements with the right leg, three days after having fallen into water. The action of moist cold has a reality in this case, because the patient allowed his wet clothes to dry upon him. (B.)

But the circumstances most commonly assigned as causes of this disease, by patients, appertain to the moral order—long-continued grief or vexation, such, for instance, as may arise from illicit pregnancy, or the disagreeable annoyances and carking cares which a more or less false social position entails. This is often the case as regards certain female teachers. Having said so much with respect to women,[1] the question of male sufferers arises. These are, for the most part, persons who have lost caste, and who, thrown out of the general current, and too impressionable, are ill-provided with the means of maintaining what, in Darwin's theory, is called the "struggle for life." In short, the etiology is a somewhat trite one, such as may be met with again, as it were, at the beginning of all the chronic diseases of the central nervous system.

C. The *prognosis* has hitherto been of the gloomiest. Shall it be always thus? It is to be hoped that, when the disease has become better known, the physician will learn how to take advantage of that spontaneous tendency to remission which has been noticed in a great number of cases. Nor must it be overlooked that, at the present time, the real nature of the disease is not recognized until the lesions have become well marked, and are consequently but little amenable to the influence of therapeutic agencies.

D. After what precedes, need I detain you long over the question of treatment? The time has not yet come when such a subject can be seriously considered. I can only tell you of some experiments which have been tried, the results of which, unfortunately, have not been very encouraging.

Chloride of gold and phosphate of zinc appear to have rather exasperated than improved the symptoms. Strychnine has sometimes caused cessation of the tremor, but its influence has always been transient. The same is to be said of nitrate of silver. In several cases, which I have noted, it seems to have had a very favorable influence over the tremor, and the paresis of the limbs, but this influence was not of long duration.

The exhibition of this drug is formally contra-indicated by the existence of permanent contracture, and especially of spinal epilepsy; its employment, in such cases, would almost certainly have the effect of exasperating these symptoms. The hydropathic treatment seems to have produced a temporary amendment in one case; in another, on the contrary, it completely failed.

Arsenic, belladonna, ergot of rye, and bromide of potassium have been likewise administered, in disseminated sclerosis, without any marked benefit. The same may be said of faradization and of

[1] The 'Lancet' (1873, vol. i, p. 236) has published the summary of a case of disseminated sclerosis recorded by Dr. Moxon, at Guy's Hospital, in which we find the following causes mentioned—*a*, febrile disease, accompanied by diarrhœa, which lasted for several weeks; *b*, a violent moral emotion experienced by the patient on seeing her husband in bed with another female. (B.)

galvanism. As regards the employment of the continued current, however, further experiments are required before we can form a definite judgment.[1]

[1] Other drugs have also been employed, without better success than resulted from the use of those enumerated by M. Charcot. Such are, phosphorized oil, iodide of phosphetylamine, and Calabar Bean. Since the-publication of the first edition of these Lectures several works or observations concerning disseminated sclerosis have appeared. As they only confirm the descriptions traced by M. Charcot, we confine ourselves to a simple catalogue. 1st. Timal, "Etude sur quelques complications de la sclérose en plaques disseminées." Thèse de Paris, 1873. 2d and 3d. H. Schule, "Beitrag zur multiplen Sklerose des Gehirns und Ruckenmarks," in 'Deutsches Archiv für Klinische Medicin,' 1870, Bd. vii, p. 259. "Weiterer Beitrag zur Hirn Ruckenmarks Sklerose" (ibid., 1871, Bd. viii, p. 223). Baldwin, "A Case of Diffused Cerebral Sclerosis" ('Journal of Mental Science,' 1873, July, p. 304). 5th. Moxon, "Two Cases of Insular Sclerosis of the Brain and Spinal Cord" ('The Lancet,' vol. i, p. 471, 609, 1875). 6th. Buzzard, "Disseminated Cerebro-Spinal Sclerosis," (ibid., vol. i, p. 45). 7th. Moxon, "Eight Cases of Insular Sclerosis of the Brain and Spinal Cord" ('Guy's Hospital Reports,' 3d series, t. xxi, London, 1875).