have here, gentlemen, to deal with a spasm which occupies simultaneously and with almost equal strength the antagonistic muscles, for, when the limbs are flexed, it is almost as difficult to extend them as it is to bend them when they are extended.

When the extremity of one of the feet is grasped by the hand, and somewhat abruptly extended on the leg, there ensues almost immediately throughout the whole extent of the corresponding limb a sort of convulsive trembling, which recalls the tremulation determined by strychnine poisoning. This tremulation, which must not be confounded with the peculiar shake that supervenes on purposed movements, is not always limited to the limb in question; it is sometimes propagated to the other limb, and then the agitation may occasionally become so intense as to shake the whole body, and even the bed on which the patient reclines. It persists in some cases for several minutes, and even much longer, after cessation of the act which set it going. You may cause it to stop at once, as M. Brown-Séquard has shown, and as I have often since observed, by grasping, with the hand, one of the great toes of the patient and flexing it suddenly and forcibly. Immediately after this operation the tetanic rigidity and convulsive trembling cease in both members, which become temporarily "perfectly supple and pliable as after death, before rigor mortis supervenes."[1] The convulsive tremulation may be determined by faradization, by pinching the skin of the leg, or, more rarely, by kneading the limb, by the influence of cold, or by tickling the sole of the foot. It also comes on sometimes spontaneously, or at least apparently so, sometimes because of an effort made by the patient, as in vomiting, defecation, raising himself in bed, or getting out and placing his foot upon the floor. It is also provoked by an attempt to walk, for permanent rigidity does not always absolutely prohibit this act; the patients can sometimes hobble along on their toes, the heel being raised from the floor. Finally, this tremulation may also be temporarily produced, along with rigidity, even during the course of the paretic period, under the influence of one or other of the several modes of excitation which we have just reviewed.

Gentlemen, the phenomenon, whose principal characters I have here sketched, is nothing other than the *spinal epilepsy* described by M. Brown-Séquard. We observe it present in the case of Mademoiselle V— in what I have proposed to call the *tonic* form. This form, which is the type most commonly met with in gray multilocular induration, may be placed in opposition to the *saltatory* form, which predominates, on the contrary, in progressive locomotor ataxia and in some other spinal affections.

Permanent contracture of the limbs and spinal epilepsy must not any longer detain us. These symptoms, in fact, do not exclusively

[1] Brown-Séquard, 'Archives de Physiologie,' t. i, p. 158.

belong to multilocular sclerosis of the nervous centres. Far from it. They shall, therefore, be studied apart, both generally and in their relations with the different affections of the spinal cord in which they show themselves.

LECTURE VIII.

APOPLECTIFORM SEIZURES IN DISSEMINATED SCLEROSIS. PERIODS AND FORMS. PATHOLOGICAL PHYSIOLOGY. ETIOLOGY. TREATMENT.

SUMMARY.—Apoplectiform seizures. Their frequency in disseminated sclerosis. General considerations on apoplectiform attacks in general paralysis, and in cases of circumscribed cerebral lesions of old standing (hemorrhage and ramollissement). Pathogeny of apoplectiform seizures; insufficiency of the congestion theory. Symptoms: state of the pulse; elevation of the central temperature. Apoplectiform seizures in old cases of hemiplegia. Importance of temperature in diagnosis.

Periods in disseminated sclerosis. First, second, and third periods. Symptoms of bulbar paralysis. Forms and duration of disseminated sclerosis.

Pathological physiology: relation between symptoms and lesions.

Etiology. Influence of sex and age. Hereditary predisposition. Previous nervous affections. Occasional causes: prolonged action of moist cold; traumatism; moral causes.

Prognosis. Treatment.

GENTLEMEN: I purpose calling your attention to-day, in the first place, to certain cerebral accidents which may happen to complicate the symptomatology of cerebro-spinal disseminated sclerosis. I refer to *apoplectiform seizures*, which are occasionally encountered several times in the course of the disease, and which sometimes close the fatal scene. These attacks have not hitherto appeared in the case of Mademoiselle V—, whose clinical record is otherwise so complete in most respects; but nothing assures us that they will not some day show themselves. In fact, this is not a rare complication; I find it mentioned in about a fifth of the cases which I have collected, and I have personally observed it in at least three instances.[1]

[1] Case III of the memoir of M. Vulpian, communicated by M. Charcot. Case of the patient Byr (Charcot); case of Nicolas, presented to the Société de Biologie, by M. Joffroy.

The group of symptoms, which constitutes an apoplectiform seizure, does not exclusively belong to multilocular sclerosis. It is found in a number of affections which involve several points of the cerebro-spinal axis at once, and particularly in progressive general paralysis. It is, indeed, in the latter disease that these *congestive attacks*—as they are commonly called, at least in France—have been specially studied on account of their frequency. They are met with there in all the various forms which they assume. The description of such attacks, in progressive general paralysis, has given rise to numerous divisions and subdivisions. But, in point of fact, all the varieties of form which clinical observation has revealed—I mean the graver kinds—may be classed as belonging to two fundamental types, namely:—

1st. *Apoplectiform attacks* (the "pseudo-apoplexy" of British authors), and

2d. *Epileptiform*, or *convulsive attacks*.

The characteristics of both types may, however, be intermingled and confounded in the same paroxysm. The first type only has been, up to the present, met with in disseminated sclerosis; but it cannot be doubted that, when observations relating to this disease shall have accumulated, they will enable us to fill up the picture.

Among the other organic diseases of the nervous centres in which apoplectiform or epileptiform attacks are frequently observed, I shall confine myself to certain circumscribed cerebral lesions of old standing, and accompanied by permanent hemiplegia. Such are *cerebral hemorrhage* and *brain-softening* when occupying regions of the encephalon, the lesion of which has the effect of almost certainly determining the cerebro-spinal alterations known under the name of *descending fasciculated scleroses*.

Between these partial lesions of the brain and progressive general paralysis it seems, at first glance, that no point of contact exists. However, gentlemen, here is a character which brings them together: the observations of M. Magnan and those of Herr Westphal have shown that, in general paralysis, there is very often superadded to the lesions of periencephalitis a sclerous alteration, sometimes diffuse and sometimes fasciculated, which occupies the crura-cerebri, pons Varolii, medulla oblongata, and certain regions of the spinal cord, at the same time. Now, these cerebro-spinal lesions (as much on account of their mode of distribution as because of the peculiar nature of the morbid process) deserve to be assimilated to the descending fasciculated scleroses consecutive on hemorrhage or softening of the brain. We know, on the other hand, that, in multilocular sclerosis, the sclerosed patches occupy not only the spinal cord and the brain proper, but are likewise very commonly found in different parts of the isthmus cerebri,

and particularly in the bulbous rachidicus. You see, by this, that the existence of irritative lesions, disseminated nearly everywhere in the cerebro-spinal axis, but always present in the isthmus cerebri, is a character common to all those affections, so different in appearance, in which the so-called *congestive* attacks supervene. I would especially point out to your attention the constant existence of the bulbar lesion, which is, in all probability, a predominant element in the production of these attacks.

However this may be, gentlemen, we have here permanent alterations of slowly progressive evolution. They cannot, consequently, without the assistance of other lesions, explain the development of accidents which are, for the most part, suddenly produced, and which may rapidly disappear without leaving any trace. I am not unaware that many physicians, even at the present day, put forward the theory of a partial sanguine congestion—a fluxion which, according to the needs of the case, should affect this or that portion of the encephalon. As regards myself, I cannot endorse this hypothesis. In order to justify my scepticism in this matter, I will appeal to the reminiscences of those among you who, in this hospital, were attached to the department for the insane. How many times have they not been disappointed in not finding, on post-mortem examination, the congestive lesion, which they expected? But I shall appeal, above all, to the cases which I have had opportunities of collecting in my accustomed field of study. Many a time have I had occasion to see patients, long suffering from hemiplegia, the result of brain-softening or intracephalic hemorrhage, succumb to epileptiform or to apoplectiform attacks. Now, in such cases, no matter what attention I gave to the autopsy, I have ever found it impossible to discover, whether in the nervous centres or in the viscera, any recent congestive lesion, œdematous or other, which could explain the grave symptoms that had characterized the fatal termination of the disease. I have never met with any but old lesions—ochreous foci, yellow patches, or foci of cellular infiltration—on which depended the hemiplegia, and the secondary degenerations of the mesocephalon and of the cord, which are the consequences of these partial lesions of the cerebral hemispheres. In short, I believe that, in the present state of science, the absence of proper lesions is, anatomically speaking, a common characteristic of these attacks, whatever be the form they assume or the disease with which they are connected.

In what relates to the symptomatology of the apoplectiform and epileptiform attacks, in order not to enter upon the details of a regular description, I shall confine myself to mentioning the following peculiarities. The scene generally opens unexpectedly, without any marked preliminaries, sometimes by rapid and more or less intense obnubilation of the intellectual faculties, sometimes by profound coma, suddenly supervening. In certain cases con-

vulsions are added, which recall those of ordinary epilepsy, but which are usually localized in one side of the body (*epileptiform attacks*). In other instances there are no convulsions (*apoplectiform attacks*). In both cases it is frequent to find, developed from the outset, a more or less complete hemiplegia, sometimes with flaccidity, sometimes, but more rarely, with rigidity of the paralyzed members. The symptoms may gradually grow worse in the course of a few days and induce death. This is usually heralded by the rapid development of eschars on the sacral region. If, on the other hand, the patient is destined to survive, the disappearance of the symptoms soon becomes manifest, hemiplegia is the only one that holds out for some time, but sooner or later it also dissipates without leaving any trace of its existence.

These attacks usually recur several times, generally after long intervals, during the course of the disease. So far as disseminated sclerosis is concerned, they have been noticed thrice in Case III of M. Vulpian's memoir, thrice in Zenker's case,[1] and up to seven times in that recorded by M. Léo[2]. In every instance, these fits left after them a notable and persistent aggravation of all the symptoms of the original disease.

The sketch which I have given you, gentlemen, would be too imperfect if I did not call your attention to the troubles of circulation and temperature which, as a general rule, show themselves in the course of these attacks. The *pulse* is always more or less accelerated; but, besides (and this is the important point), the *temperature* of the central parts rises rapidly; it may, in the hours immediately following the invasion, reach 38.5° C. (= 101.3° F.), or even 39° (= 102.2° F.), and frequently, at the end of twelve or twenty-four hours, it rises to 40° (= 104° F.), and remains at this elevation for some hours, without necessarily entailing a fatal result. But if the patient is to survive, the temperature soon diminishes rapidly. An increase above 40° C. is almost always followed by a fatal termination.

These modifications of central temperature have been studied by Herr Westphal in the epileptiform and apoplectiform attacks of *progressive general paralysis;* I have met with them again in the attacks which supervene in patients suffering from *hemiplegia of old standing*, consecutive on *hemorrhage* or on *softening of the brain*. In order the better to settle your ideas upon this subject I think it will be useful to summarize the details of two cases relating to the last-named species.

The first case is that of a woman, aged thirty-two years, affected by hemiplegia of the right side, dating from childhood. There existed general atrophy, rigidity, with shortening of the limbs,

[1] Bourneville et Guérard, *loc. cit.*, p. 112.
[2] *Ibid.*, p. 112.

and paralysis, such as are generally found in like cases. This woman was subject to epileptiform attacks. She was brought to the infirmary some hours after a more than usually severe attack. On the evening of her admission her temperature was above 38° C. (=100.4° F.); next day it had reached 4° C. (=104° F.). The fits became subintrant; they were repeated about a hundred times a day. Eschars formed rapidly on the sacral region, and death supervened the sixth day. On that day the rectal temperature stood at 42.4° C. (=108.32° F.). On post-mortem examination there was found, at the surface of the left cerebral hemisphere, a considerable depression answering to a yellow patch, the remnant of a vast focus of ramollissement. The whole hemisphere, moreover, was atrophied. No trace of a recent lesion could be found, neither in the nervous centres nor in the viscera.

The second case is that of a woman, aged sixty years, afflicted with right hemiplegia consecutive on cerebral hemorrhage, dating from two years previously. This patient had already experienced several epileptiform or apoplectiform attacks, which, however, were generally slight. One day an intense and prolonged epileptiform attack supervened, which was followed by an apoplectiform condition. Two hours after the setting in of these accidents, the rectal temperature was 38.8° C. (=101.84° F.); five hours later, it rose to 40° C. (=104° F.). Next day, in spite of the cessation of convulsions, the temperature was 41° (=105.8° F.); and the day following, being the day of her death, it reached 42.5 C. (=108.5° F.). The autopsy showed two ochreous foci, one occupying the corpus striatum, the other the substance of a convolution. There existed no recent lesion capable of explaining the accidents which had determined death.

I have as yet had no opportunity of following, day by day and at different periods of the day, the changes of central temperature in a case of *apoplectiform seizure* supervening in a patient affected with *disseminated sclerosis*. Nevertheless, we can gather partial results from different cases, which leave no doubt that, even in this respect, matters proceed exactly in the same way in multilocular sclerosis, as in progressive general paralysis and in circumscribed lesions of the cerebral hemispheres. Thus, the patient whose history has been related by Herr Zenker was, towards the close of his life, taken with an apoplectiform attack, followed by hemiplegia of the right side. Now, on the day of the seizure, his pulse being at 136, the temperature reached 39.6° C. (=103.28° F.). Next day, the thermometer marked 40° C. (=104° F.). The day after, the paralysis had ameliorated and the temperature had fallen back to the physiological figure. In the case of the patient Nolle, narrated by M. Léo, an apoplectiform attack came on in the evening. Next morning early, the pulse numbered 144, and the temperature stood at 38.5° C. (=101.3° F.). This attack, the seventh