

of the patient the dizziness is alone to be mentioned, which, however, even if the shaking movements were not present, in itself would be sufficient to keep him from doing anything.

As a result of this examination the diagnosis of multiple sclerosis was made, and will certainly be proved to be correct at the post-mortem examination. It is interesting, however, that in this case the course of the disease suggested in its initial stages Thomsen's disease (although not congenital), later tabes (with retained knee-kick), then spastic spinal paralysis (conjectural diagnosis), before the picture of insular sclerosis developed.

Diagnosis.—The diagnosis presents difficulties in almost every case, owing to the protracted course and the changes in the picture of the disease during the different periods. Even the most careful examination will not always keep us from

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Fig. 167.—SPECIMEN OF HANDWRITING OF A PATIENT (HAT-MAKER) WITH A MERCURIAL TREMOR.

errors, and we must never be surprised if the autopsy does not always confirm the diagnosis made during life. The case of Westphal, in which a multiple sclerosis was diagnosed, but where *post mortem* no lesions at all were found, has been alluded to before. In another instance, reported by Frey (cf. lit.), there was found, instead of the confidently expected foci of sclerosis, a leptomeningitis, and similar errors would not be difficult to find on a careful perusal of the literature. The possibility that we are dealing with hysteria, in a given case, must always be considered, and then, of course, great weight must be laid upon the presence of other symptoms which would indicate such a condition. The difference between intention tremor, as illustrated in Fig. 164, and other tremors, can be seen by a comparison with Figs. 165-168.

Pathological Anatomy.—The anatomical changes of multiple sclerosis are extremely characteristic. Even with the naked eye, here and there, grayish-white foci are seen in the brain, in the white matter of the hemispheres, in the walls of

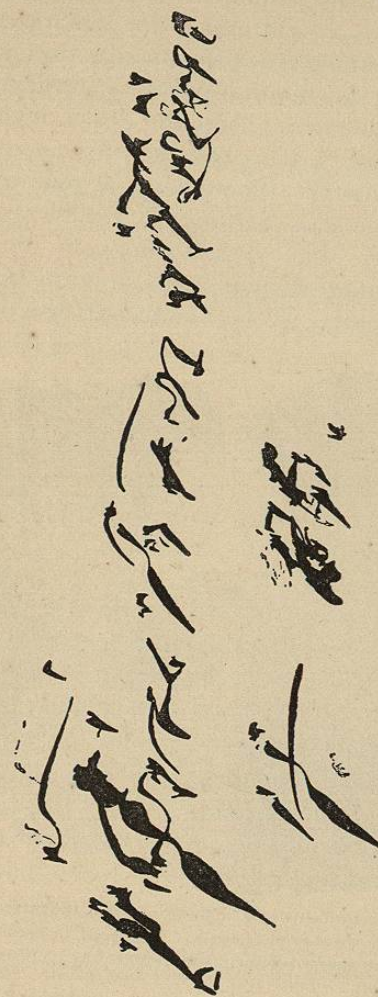


Fig. 168.—SPECIMEN OF HANDWRITING ILLUSTRATING THE TREMOR PRODUCED IN A CASE BY THE COMBINED ACTION OF ALCOHOL AND MERCURY. (V. FINKENSTEIN, 4, 86.)

the lateral ventricles, in the corpus callosum, in the pons, and on its surface, in the medulla oblongata, in the floor of the fourth ventricle, and in the spinal cord, where the white matter is decidedly more affected than the gray. The foci are distributed in a very irregular manner; sometimes they are more numerous in the brain, sometimes they are more numerous in

the cord, often they are found scattered equally over the entire central nervous system. If they are situated on the surface, they are seen through the pia, and are somewhat more prominent than the parts which surround them. They are generally harder and firmer than the rest of the substance, and on section they assume a light-pink color when exposed to the air. If they are examined microscopically, they are found to consist of reticulated fibrillary supporting tissue, and contain only a few intact nerve fibres; after the death of the medullary sheaths, the axis cylinders are preserved for an extraordinary length of time (Charcot). Secondary degenerations in the spinal cord are often absent (Strümpell), yet they are occasionally seen (Werdnig). The vessels show an increase in the nuclei, later a thickening of their walls, and are seen as yellow dots in the sclerotic foci. Whether the disease of the vessels

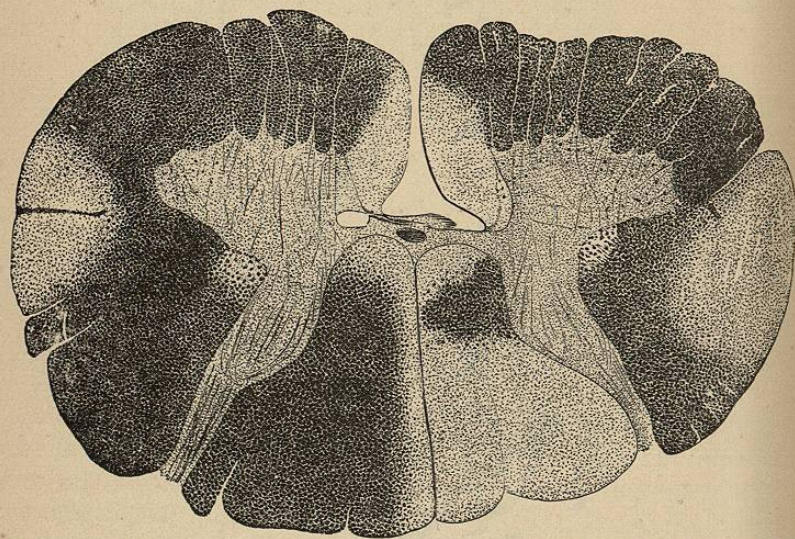


Fig. 169.—CROSS-SECTION THROUGH THE CERVICAL ENLARGEMENT OF THE SPINAL CORD IN A CASE OF MULTIPLE SCLEROSIS. Hardened in osmic acid. The lighter colored areas in the white matter represent the sclerotic foci. (After BRAMWELL.)

actually gives rise to the foci is not yet established (Fig. 169). Disease of the peripheral nerves has never been demonstrated in multiple sclerosis.

Ætiology.—About the ætiology we know practically nothing. It is possible that heredity deserves some consideration in this connection, but there are relatively many cases in which

this factor can positively be excluded. The influence of infectious diseases upon the development of insular sclerosis has recently again been dwelt upon by Marie (cf. lit.). Typhoid fever, variola (Sottas, *Gaz. des hôp.*, 1892, 44), scarlet fever, measles, whooping-cough, influenza (Massalongo, *Silvestri, Revue neurol.*, 1893, i, 23), and intermittent fever have repeatedly been known to precede the disease, although the material at our disposal is not as yet sufficient to prove a causative relation between the two. With regard to syphilis the connection here is by far less definite than, for instance, in tabes (cf. the case of Buss, lit.). Age and sex seem to be of some significance, inasmuch as children and aged people seem to be exempt. Westphal and others have only exceptionally seen it in children. Strümpell has observed it in a man of sixty. Both sexes seem to be attacked with about the same frequency. I have seen a case in which after a severe traumatism (fall from a ladder) the three cardinal symptoms of multiple sclerosis developed; nevertheless I am not convinced that the case was not one of traumatic neurosis. The question can only be settled by the autopsy.

Treatment.—An effectual treatment for multiple sclerosis does not exist. We possess no remedy which will arrest the development of the foci. The symptomatic treatment must always be tried, however, and the patient particularly seeks relief from the annoying tremor. For this we may administer, although without raising our expectations too high, veratrine, physostigmine, one to three milligrammes (grs. $\frac{1}{60}$ – $\frac{1}{20}$) daily, in pills, or solanin, recommended by Grosset and Sarda, and even termed by these authors "*médicament du faisceau pyramidal*" (*Progr. méd.* 1888, 27). It may be given in doses of from two to three centigrammes (grs. $\frac{1}{3}$ – $\frac{1}{2}$) from three to five times daily. In other respects the treatment is the same as in myelitis (cf. page 455).

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CHAPTER II.

TABES DORSALIS—LOCOMOTOR ATAXIA (POSTERIOR SPINAL SCLEROSIS—LEUCOMYELITIS POSTERIOR CHRONICA).

THE second of the diseases belonging to this group certainly deserves to be considered as one of the most important of those with which we are acquainted, not only because it is to be reckoned among the diseases of the nervous system which occur relatively frequently, and with which the general practitioner is not rarely brought face to face, but also because its clinical picture presents so many essential differences that it requires a large experience to feel at home with it on all occasions. Nobody questions the importance of the recognition of the disease in its early stages if only on account of its bearing upon the treatment, but many do not appreciate the difficulties which this early diagnosis entails. The more cases of tabes we see, the more we are surprised at the protean character of the symptoms, and the more are we convinced that almost every case offers some point of particular interest, and that occasionally even an expert can be sure of the diagnosis only after repeated examinations and long observation.

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Symptoms.—The symptomatology of tabes is so comprehensive that in order to get a clearer idea of it we shall in our description separate the cerebral from the spinal symptoms.

The cerebral symptoms which appear in the course of the