

Post-Hemiplegic Disturbances of Motion. B. GREIDENBERG. *Arch. für Psych.*, xvii., 131.

This article contains the most exhaustive discussion of the various symptoms which develop after a hemiplegia, and is noticeable on account of its masterly summary of the facts recorded by various authors on the subject, and by its complete bibliography. It deserves careful study and suffers in any attempt at condensation.

The first symptom discussed is *post-hemiplegic contractures*. These are classified as (1) such as appear at the time of the apoplexy or follow it immediately. They may be (a) temporary, lasting only during the attack; (b) intermittent, in the form of tetanic spasms; (c) stationary, continuing till death; (d) alternating with spasms from time to time. These contractures are due to (1) a laceration of the fibres of the motor tract in their passage through the centrum semiovale or internal capsule, which produces such an irritation in the tract as to cause muscular action. They are very frequent in those cases in which a hemorrhage has torn its way into the lateral ventricles, but it is to the injury to the tract, and not to irritation within the ventricles that this form of contracture, termed apoplectic, is due. These contractures are due (2) to irritation of the cortex in the motor area, such as occur in meningeal apoplexy; and (3) to injury in the crus or pons, which involves the pyramidal tract. Any lesion of a destructive nature which involves the motor tract may therefore cause an apoplectic contracture.

(2). The second form of contracture is termed primary or early contracture, and appears from two to five days after the apoplexy. In these cases the limbs are flaccid for a time, but then the muscles become rigid, both extremities assuming a position of semi-flexion, the flexors prevailing over the extensors. This form of contracture is easily overcome by passive motion, passes off soon as a rule, but occasionally goes on to the secondary contracture. Its cause is supposed to be an irritation of the motor tract produced by the inflammatory process set up by, and in the vicinity of, the initial lesion. This irritation is conveyed to the muscles and results in an increase of normal muscular tone and consequent contracture.

(3). The third form of contracture is secondary or late contracture. This is very common, and is usually permanent. It appears from six to eight weeks after the apoplexy, although occasionally it appears earlier (twenty days). It is not a simple rigidity of the flexors, but affects all the muscles of the limbs, and is not easily overcome by passive motion. As a rule the arm is flexed, the leg extended; but occasionally the arm is extended, and the hand may then be fixed in various positions. It is rare for the leg to be extremely flexed, and as a rule the leg is affected to a much less degree than is the arm. Contractures of the face, neck, and thorax have been noticed, but are very rare, and occasionally the opposite extremities have been involved, and a para-

plegia has resulted. Such contractures are diminished by rest, by warmth, by sleep, or by bandaging, and are increased by effort, cold, and mental emotion, or mechanical irritation. Although occasionally subsiding under favorable conditions, they are apt to return suddenly, and have therefore been called latent contractures. They are not unfrequently followed by atrophy of the contracted limbs. This form is most fully developed in infantile spastic hemiplegia. Various hypotheses have been offered to explain this form. It is due to the development of secondary degeneration in the pyramidal tract, which is supposed by some to cause an irritation of the fibres in this tract, an irritation which being conveyed to the motor cells of the spinal cord produce the contracture. Continued irritation of a normal cell, however, exhausts the cell and results in a temporary suspension of its action; it is therefore supposed by others that the degeneration extends to the cell itself and affects its function in such a manner that it responds to all reflex impulses more readily than before, the least excitement leading to excessive muscle tone, and thus to contracture. The contracture is therefore ascribed to continued irritation of the anterior cells of the spinal cord. This is the theory most widely accepted. If these cells are actually destroyed as well as altered in function then an atrophy of their muscles results.

The second symptom discussed is the *increased tendon reflexes*, on the paralyzed side. This symptom is very constant. The statement of Meulen's, that the reflexes are slightly exaggerated during the first month after the attack, are greatly exaggerated from the second to the seventh month, and then gradually return to the normal, has not been confirmed by other observers. On the contrary, a rapid and permanent increase of the reflexes is found to occur in the very large majority of cases. The symptom has been noticed within one hour of the attack, but it usually develops from the seventh to the twenty-first day (Westphal). The theories which are offered to explain this symptom are (1), that the inhibitory influence exerted by the cortex upon the subcortical centres is removed by the rupture of the motor fibres which conduct these impulses (Westphal); (2) that the secondary sclerosis in the motor tract produces an irritation of the fibres, and this being transmitted to the cells of the anterior horns of the spinal cord increases their irritability (Charcot); (3) that the two factors already mentioned are supplemented by a third, which is the result of the first two, viz., a tension of the muscles and tendons with increased irritability of the peripheral nerve-endings (Ross); (4) that the lesion itself produces an irritation which is sent along the motor tract and is communicated to the motor cells of the cord (Schwarz). The latter theory is preferred by the author, who cites in its favor the recent experiments of Adamkiewicz on brain compression. When the cortex was compressed the tendon reflexes increased, but they diminished again to normal when the compression ceased. From this experiment it is evident

that permanent degeneration is not necessary to the production of the symptom, and that temporary irritation is sufficient to cause it.

Associated movements of the paralyzed and unaffected halves of the body are then discussed. Motions of the face as evidence of emotion, such as occur in laughing or crying, are often found to be not only perfectly performed on the paralyzed side, but even excessive. They are only defective where the optic thalamus is outraged (Nothnagel). So too movements of the arms in yawning are usually well executed, although voluntary motion in one arm is imperfect. Frequently, when the unaffected arm is moved, the paralyzed arm makes, or at least begins, a corresponding movement. This is seen chiefly in those cases in which voluntary motion is entirely impossible and in cases of infantile hemiplegia. Westphal has offered in explanation the theory that voluntary impulses starting from one hemisphere of the brain may be transmitted by commissural fibres to the opposite hemisphere, when inhibition is impaired, and thus give rise to these associated movements. There is another form of associated motion which Benedikt considers characteristic of infantile hemiplegia. It is well known that when the flexors are forcibly contracted there is also a slight action of the extensors, which serves to fix the points so that the maximum effect of flexor action is possible. The same is true of many sets of antagonistic muscles. In infantile hemiplegia this normal contraction of antagonistic muscles may be excessive, so that any desired motion is performed with difficulty on account of the internal resistance as it may be called. This is produced by the unbalanced action of antagonistic muscles. Such unbalanced action is not observed when the seat of the lesion is in the motor tract. It is quite characteristic (according to Benedikt) of cortical disease. Charcot and Strümpell, however, regard such movements as reflex in their origin.

Posthemiplegic tremor, resembling that of either paralysis agitans or multiple sclerosis, has been observed, but is rare, occurring in less than five per cent. of the cases. Posthemiplegic chorea is more frequent. The motions are usually more severe and constant in the arm than in the leg, and they increase on voluntary movements. The symptom is often associated with hemi-anæsthesia in the affected limbs. The same is true of hemi-ataxia, and both symptoms are characteristic of lesions which affect the posterior part of the internal capsule, through which the co-ordinating tracts pass. Posthemiplegic athetosis has been observed in about one hundred and fifty cases since its first mention by Hammond. The athetoid movements are usually slow and regular, but occasionally they may be rapid and jerky. They are usually so uniform as to be called mechanical, and have been likened to those of a machine. The hand-muscles are the ones involved, though the entire arm and the leg may take part in the movements. Hammond's statement that the motion continues during sleep has not been confirmed by other observers. Hemi-anæsthesia is not associated with athetosis as often as with chorea,

but may be present. While some cases with autopsy support the theory that this symptom is due to lesion of the optic thalamus, others fail to confirm it, and the cause is therefore still undetermined.

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Paralysis of the "Glottis-Dilators" as the Initial Symptom of Tabes Dorsalis. Prof. A. WEIL, of Heidelberg. *Berl. klin. Wochenschrift*, March 29, 1886.

Prof. Weil gives a full account of a case of tabes dorsalis, which set in with peculiar laryngeal symptoms. The patient is a man, æt. forty-nine, and a pilot on one of the large rivers of Germany. About a year before he consulted W. he had passed through intense excitement from the fact that his boat was in danger of colliding with another. He issued his commands in a very loud tone of voice, and immediately thereafter had an attack of extreme dyspnœa, which lasted about ten minutes with crowing respiration, etc. Eleven months later he had a second attack of the same sort. From that time the least exertion produces a loud snoring respiration. Laryngoscopic examination showed that the margins of the glottis were two to three *mm.* apart during expiration, but during inspiration there was nothing but a cleft-like opening left. Phonation was entirely normal. These symptoms differ from those of ordinary laryngeal crises in several particulars: 1. They are not distinctly paroxysmal: there is a *permanent morbid condition* which is aggravated by physical exertion or mental excitement. 2. There is no cough. In regard to the question whether these symptoms are due to paralysis of the *abductor*- or spasm of the *adductor*-muscles, the author is not prepared to give a definite answer, but he inclines to the view that there is paralysis of the dilator-muscles. The author gives a list of published cases in which symptoms of this sort were observed. He refers very properly to the necessity of examining for tabes in all cases of sudden paralysis of the vocal cords.

On a Case of Locomotor Ataxia with Laryngeal Crises and one of Primary Sclerosis of the Columns of Goll, Complicated with Ophthalmoplegia Externa. By JAMES ROSS, M.D., LL.D., *Brain*, April, 1886.

Dr. Ross' first case is of greatest interest when compared with the one we reviewed in the preceding paragraph. Although Dr. Ross speaks of "laryngeal crises" the symptoms are remarkably like those which Weil's patient described. Dr. Ross says of his patient, "About two years before admission, the patient began to experience a crowing noise *along with inspiration*. He suffered *almost continuously* from this noisy inspiration; but in addition he had paroxysms of difficulty of breathing, each of which lasted several minutes. * * * he was told that the noise which accompanied breathing was always present in a marked degree when he was asleep."