

the foregoing acute form. Death occurs early on account of refusal of food. There may be periods of expansive ideas. A succession of expansive and depressed states constitutes Mendel's circulatory paresis, which Salgó refuses to recognize as a distinct entity, considering it without practical and scientific value. The third form is characterized by a longer duration of ameno-mania and delusions of grandeur and a more regular general course. Nevertheless, cases of this kind may run a sufficiently rapid course, dependent in this particular upon the paralytic symptoms. Remissions are frequent, and these naturally increase the duration of the disease. Remissions can only be expected when there is embarrassment of speech. Death is usually consecutive to marasmus following subacute attacks of exaltation and violence. Cerebral œdema, apoplectiform, or epileptiform attacks are also frequent causes of death. Often between a period of remission and reprisal there is an intermediate state of *folie raissonante* that bears a striking resemblance to the period of termination in cases of long-standing mania. The fourth is that form of paresis of longest duration, in which dementia and paralytic symptoms show parallel development and in which there are no delusions and no ideas of grandeur. Cases of this kind are common, and there is not the slightest need of isolating or incarcerating them. They are quiet, not dangerous, and easily cared for at home. This fourth form presents the typical course of paresis. Its clinical facts are confirmed by autopsy.

Salgó insist upon the appropriate analogy existing between paresis and chronic brain changes due to local lesions or to general constitutional diseases, such as senile changes in the brain substances and meninges; cerebral hemorrhage, local softening, cranial and cerebral traumatism, chronic alcoholism, hematoma of the dura mater, and cerebral syphilis. These pseudo-paretic or symptomatic groups run a very different course from true paresis, have a distinctly different pathological anatomy, and must not be confounded in any way with general paralysis.

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OBJECTIVE SYMPTOMS OF NEURASTHENIA.

Among the objective symptoms of neurasthenia which we have gradually learned to recognize in addition to the many subjective ones, the following deserve

special mention: Pale complexion and emaciation induced by derangement of sleep and nervous dyspepsia; pronounced redness of the conjunctivæ and the ears; dilatation and frequently transient inequality of the pupils; incomplete closure of the eyelids when directed to stand with closed eyes; fibrillary tremor in the orbicularis oris and in the musculature of the tongue; weakness in convergence of the eyes; unconscious and aimless movements of the extremities; increase of the skin and tendon reflexes (loss of knee-jerk was not observed); pronounced mechanical irritability of the facial nerve; increased electrical irritability of nerves; weakness and indistinctness of speech; manifestations of paraphasia and verbal amnesia; changes and errors in writing; a disposition to abnormal laughing and yawning; acceleration and irregularity of heart's action; abnormal prominence of the temporal arteries in consequence of vasomotor disturbances; nervous dyspepsia with anomalies of the motor and secretory functions of the stomach, with eructations and vomiting; nervous constipation and diarrhoea; polyuria, phosphaturia, and oxaluria, moreover a uric acid diathesis of long duration. The latter may be associated with a neuropathic condition either congenital or acquired, or with the neurasthenic state (Löwenfeld, *Neurolog. Centrbl.*, 1892, No. 17). W. M. L.

A CASE OF HUNTINGTON'S CHOREA, WITH AUTOPSY.

The following case is reported by Drs. Kronthal and Kalischer in the "*Neurologisches Centralblatt*," Nos. 19 and 20, 1892. The patient was a woman, forty-five years of age. The chorea began in her thirtieth year. One sister was similarly affected at the same age. Her grandmother, mother, and mother's cousin also suffered from chorea. Her father died of phthisis. The patient showed all of the symptoms of the disease, together with endocarditis. Eight days before death she fell, striking her head, and producing a fracture at the base of the skull. The autopsy was made forty-eight hours after death.

After an exhaustive and elaborate description of the anatomical findings, and a review of the literature of the subject, the authors summarize the result of their histological examination, which was made in Mendel's laboratory (excluding those conditions found at the autopsy which were the immediate cause of death).