motor disorders in 13. The most striking psychic change consists of a hyper-excitation of the imagination, affecting the memory, the attention, reasoning, etc., with unconventional and immoral conduct. The author agrees that unless the evolution of the case is known, the diagnosis is often very difficult.

W. D. CHAMBERS.

[Jan.,

Action Tremor. (Journ. of Nerv. and Ment. Dis., July, 1926.) De Jong, H.

The author distinguishes action tremor from intention tremor, and therein differs from Kinnier Wilson, who considers them identical. An action tremor does not exist in rest but appears with movement. Any action may initiate the tremor. He considers all striated tremors to be action tremors. The frequency of action tremors is greater than that of intention tremors. Clonus is a phenomenon of rhythmical oscillations occurring in pyramidal hypertonia; tremor is a rhythmical phenomenon of extra-pyramidal rigidity. Tremor, then, is an extra-pyramidal clonus.

G. W. T. H. FLEMING.

A Clinical and Pathological Résumé of Combined Disease of the Pyramidal and Extra-Pyramidal Systems, with Especial Reference to a new Syndrome. (Brain, June, 1926.) Lhermette, K., and McAlpine, D.

A male, æt. 60. Weakness of lower limbs first noticed at 52. This progressed and rendered walking difficult. He had then a spastic paresis with evidence of a bilateral pyramidal lesion. There was considerable motor difficulty in the upper limbs, but no true paralysis in any of the limbs. At 56 he improved, and at 58 showed a typical paralysis agitans *sine tremor* and without involvement of the facial muscles. There was a bilateral extensor plantar response. Involuntary movements, choreiform in type, were present in the muscles of the face, pharynx, larynx, and at times in the left hand. There was considerable articulatory difficulty. He died æt. 60. The main histological features of the case were : In the putamen a marked reduction of the cells with neuroglial overgrowth. Degeneration of many of the fibres running to the globus pallidus and ansa lenticularis from the putamen. Many of the fibres having origin in the caudate nucleus were affected. In the globus pallidus a slight but definite reduction in the number of motor cells, neuroglial overgrowth and degeneration of some of the fibres rising in the globus pallidus, especially those which go to form the ansa lenticularis. Degeneration of the pyramidal tract was not evident above the level of the medulla. In the cord, degeneration of the crossed pyramidal tracts.

The condition has to be distinguished from Parkinson's disease, pseudo-bulbar palsy, progressive pyramido-pallidal degeneration, spastic pseudo-sclerosis and amyotrophic lateral sclerosis. The authors discuss the differential diagnosis.

From a pathological point of view we have to distinguish it from paralysis agitans, the Parkinsonian syndrome following epidemic encephalitis and syphilitic encephalitis of the corpus striatum. In discussing the physio-pathology the authors, after considering the histology of Huntingdon's chorea, in which there is a widespread cellular atrophy and demyelinization in the frontal cortex, together with disappearance of many of the cells of the caudate nucleus and putamen with neuroglial cell overgrowth, conclude that no theory