of an inch. He controlled this by examination of fibres from the flexors of a normal adult forearm which varied from 1-350 to 1-1000 of an inch.

It is to be noted that Jacoby, from his earliest researches, doubts whether these muscle changes can be regarded as peculiar to Thomsen's disease—a doubt which may be borne in mind in the consideration of its etiology.

Pathology.—Since Thomsen's day many theories have been offered to explain the condition. So far as the writer knows the necropsy by Dégérine and Sottas still remains the only one on record, so that any discussion can only be speculative. Thomsen's own view seems to have been that the affection was a neurosis, and he called special attention to the heavy incidence of nervous disease in his family group. It has been ascribed to a congenital, faulty development of the pyramidal tracts. Jacoby has regarded the disease as a congenital malformation of the muscular fibres—each fibre containing too many sarcous elements—hence the hypertrophy of the fibres, and the increased duration of their contradictions. Dana says: the disease is probably a primary muscular dystrophy. There may be, however, a peculiar defect in innervation, resulting from a congenital anomaly of the motor tracts. The views of Hale White are thus stated:—"It would appear that each individual affected is from his birth faultily constructed, so that some of his muscular fibres all through his life grow abnormally, and in consequence of this abnormal growth contract in an abnormal manner. This is more in harmony with what we know of other diseases than to believe as Dégérine and Sottas apparently do, that the abnormal contraction of the muscle leads to its abnormal growth. All who have written on the subject agree that it is a disease solely of the muscular system." (Albutt's System, Ed. 1905).

The writer ventures to dissent from the last statement as the possibility of the nervous system being seriously at fault seems to be held by a number of most competent observers.

Hale White calls attention to the similarity of the muscle tracings in Thomsen's disease to those induced in animals poisoned with veratria; and also to the well known experiments of Ringer and Sainsbury with phosphate of soda on curarized animals, from which it appears that the myotonic contractions produced on stimulation of the sciatic nerve are due to the action of phosphate of soda on the muscular fibres themselves.

Danilla in reporting a Russian case (1886), expresses the view that it is a functional disturbance of the cerebral psycho-motor centres.

Cook and Sweeten have suggested the following explanation of their