Slight changes of temperature are insignificant, especially with cold, a difference of 5 deg., making no practical difference in the reactions. From 0 deg. up to 23 deg. or 24 deg. there are cold reactions, intense below 6 deg. or 8 deg., and growing less marked up to 15 deg. At 22 deg. hot reactions begin to come in but are not marked until 40 deg. or 42 deg. are reached. At between 49 deg. and 54 deg. the heat passes over into pain which arises from all points of the skin alike. The range of cold reactions is much greater than that of hot.

The relation between "hot" and "cold" seems to be one of mutual independence. They are neither coincident nor complementary, but seem to overlap without law. The personal factor was prominent.

CHRISTISON.

143. LA TOSSICITÀ DEL SUDORE NEGLI EPILETTICI (The Toxicity of the Sweat of Epileptics). Cabitto (Revista Sperimentale di Freniatria, 23, 1897, p. 36).

Dr. Clemente Cabitto experimented with the perspiration of epileptics upon rabbits and came to the following conclusions:

I. The sweat of epileptics in the prodromal period of the attack injected into the circulation of rabbits provoked a decisive toxic action and a very strong convulsive attack.

II. The toxic and convulsive power of the sweat increases as the attack advances, and decreases in the period after the paroxysm relatively with the duration of the post epileptic state.

III. The attacks preceding the day of experimentation do not exert any influence over the toxicity of the sweat.

IV. The action of the sweat of epileptics some time before the attack does not differ from that of a healthy subject. KRAUSS.

144. LE SENS DE L'ORIENTATION. (The Sense of Position). P. Bonnier (Revue Scientifique, 1898, p. 108).

In a short and interesting article the author discusses the general problem of the sense of position. To the ampullæ of the semi-circular canals he attaches much importance, in that there is a memory sense connected with the displacement of their contents. VOGEL.

145. LA SENSIBILITÉ MUSCULAIRE DES YEUX (Muscular Sensibility of the Eyes). B. Bourdon (Revue Philosophique, 22, 1897, No. 10).

In the estimation of space this muscular sense is of value, and the results of previous investigations having given such contradictory results, the author was led to perform some experiments.

The results of these experiments would be contrary to the hypothesis that our conception of space is due to muscular sensibility of the oculomotorius. The movements of the eyes are, therefore, not controlled by muscular sensibility, but by the retinal impressions. Muscular sensibility of the oculomotorius could be markedly diminished, yet visual space perception suffer no loss. JELLIFFE.

PATHOLOGY.

146. DEGENERATIONEN DER VORDERHORNZELLEN DES RÜCKENMARKS BEI DEMENTIA PARALYTICA (Degenerations of the Cells of the Anterior Horns of the Spinal Cord in Dementia Paralytica). H. Berger (Monatsschrift f. Psychiatrie u. Neurologie, 3, 1898, p. 1).

Berger has examined the cells of the anterior horns of the cord in twelve cases of dementia paralytica. He has found pigmentary degeneration, karyolysis, destruction of the dendrites, tumefaction of the cells, chromatolysis, vacuolation, changes in the nucleus and

nucleolus, etc. The division of the nucleus of the nerve cell into two parts, reported by him, each with a nucleolus, without division of the cell body, is worthy of special note. Berger regards this as an incomplete regeneration of the nerve cell. He has observed changes in both horns of the cord in man after destruction of one pyramidal tract, but he was unable to find that lesions of this tract in the dog and cat, or of a posterior root in the dog, had any effect on the anterior cells of the cord. He explains the muscular atrophy seen in *dementia paralytica* by the cellular changes which he has observed, and states that such cellular alteration occurs in the anterior horns of the cord in 83 per cent. of all cases of general paralysis, and is most frequent in the lumbar region. He believes that the disease of the anterior cells is primary; that it is, to a certain extent, independent of degeneration of the white columns; and that there is no constant relation between the degree of these cellular changes in the cord and the degeneration in the brain.

SPILLER.

147. THE MORBID ANATOMY OF A CASE OF HEREDITARY ATAXIA. A. Mcyer (Brain, 20, 1897, p. 276).

The author had an opportunity to study a case already reported by Dr. Sanger Brown. The following general statements are offered:

1. There is no circumscribed cerebellar lesion, nor does the cortex show a marked decrease of the number of the Purkinje cells.
2. Parts of the spinal cord and medulla which are known to have relations with the cerebellum were found affected.
3. The spinal cord, as a whole, shows increase of the superficial neuroglia, and a remarkably large number of corpora amylacea, similar to what is seen in very old people, in paralysis agitans, etc.

JELLIFFE.

148. FAIT RELATIF A L'ÉTUDE DE LA PATHOGÉNIE DES ARTHROPATHIES ET DES FRACTURES SPONTANÉES CHEZ LES TABÉTIQUES (The Pathogeny of Arthropathies and Spontaneous Fractures in Tabetics). A. Pitres et G. Carrière (Archives Cliniques de Bordeaux, 5, 1896, p. 483).

The authors give the clinical history and pathological findings in a case of tabes in a man of 58, in whom the disease had existed since his twenty-sixth year. At 38, there was spontaneous fracture of the right tenth rib; at 42, arthropathy of the left knee. The autopsy and microscopical examination showed the usual sclerosis of the posterior columns, no change in the antero-lateral columns or the cells of the anterior horns; advanced atrophy of the posterior roots, the anterior roots normal; diffuse alterations in the ulnar, intercostal and nerves of the lower extremities, especially marked in the nerves of the left knee-joint, and the tenth right intercostal—supplying the fractured rib. They then discuss the two theories proposed to account for arthropathies and spontaneous fractures.

The one theory makes the trophic disturbance of the bones and joints dependent upon atrophy of the cells of the anterior horn; the other refers it to degeneration of the peripheral nerves, especially of those supplying the diseased part. Collecting all available observations, they find that in only four cases alterations in the cells of the anterior horn are reported, and these cases were studied before the development of our present technique.

On the other hand, between 1882 and 1896, seventeen cases have been examined, and in none of these was found alteration of the cells of the anterior horns. In eleven of these cases the peripheral nerves