their counterpart in such psychic abnormalities as fixed ideas, obsessions, etc. A tic may thus arise from an obsession if the besetting idea provokes a motor reaction; or, inversely, a tic may engender an obsession. The mental basis is similar in the two cases, and it is not rare to see obsessions and tics alternate or coincide in the same individual. The different varieties of phobia, the *délire du toucher*, hypochondriacal doubts, etc., are mentioned as forms of obsession, common in the subjects of tic. The authors urge that it is important to distinguish the tics which belong to the fundamental state of psychic infantilism from those which are related to these secondary mental disorders. In the latter, which are harder to eradicate, it is necessary to direct treatment specially to the mental condition.

W. C. SULLIVAN.

Differential diagnosis between Hysteria and Katatony [*Beiträge zur differential Diagnose der Hysterie und Katatonie*]. (*Allgem. Zeitsch. f. Psychiat., B. lviii, H.* 5 *and* 6.) *Kaiser, O.*

He describes at considerable length two patients in the Asylum of Alt-Scherbitz, one, which he calls a typical case of katatony, becoming finally *dementia præcox;* the other, a young student with hysterical convulsive attacks and hallucinatory states and delirium. Kaiser regards hysteria as an abnormal mental susceptibility of the nervous system, by which it becomes prone to yield either to outward suggestions or to fanciful notions formed within the mind of the patient. Through this hyper-suggestibility, whole association systems are diverted from their functions, and the activity of others heightened. The differential diagnosis between katatony and aggravated hysteria is stated to be, that in the former there is a childish mental weakness, a state of depression with few ideas, passing into dementia, which contrasts with the selfish caprice, cunning, and persistence of purpose in the hysterical patient.

In my opinion, katatony is a formal distinction into which it is difficult to squeeze a sufficient number of cases of insanity. To find katatony one must hold Kahlbaum's description in mind, and step into the asylum to seek for examples. It is like looking for faces in the fire.

WILLIAM W. IRELAND.

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6. Pathology of Insanity.

Changes in the Cerebellar Neuroglia in Progressive Paralysis [*Die Gliaveränderungen im Kleinhirn bei den progressiven Paralyse*]. (*Arch. f. Psychiat. u. Nervenkr., B. xxxiv, H.* 2, *p.* 523.) *Raecke, Dr.*

Fifteen cases in which the changes in the cerebellar neuroglia were specially studied are given in some detail. The results correspond generally to those of Weigert. In the molecular layer, Bergman's fibres are increased in numbers, but unevenly. Most of the new fibres run vertically, but some obliquely or transversely, the last often forming bands at two levels, viz. along the outer margin of the cortex and at the

boundary of the granular layer. In the latter position they form basketworks enclosing Purkinje's cells. The transverse fibres are mostly delicate, but a number of large spider-cells at the border of the granule layer give off coarse fibres, running to the surface. The largest collection of glia-nuclei is in the same situation. In the granule layer also the changes are of unequal degree. They consist in loss of granules, the place of which is taken by hypertrophied neuroglia fibre and nuclei. In the medulla the hypertrophy is rarely of great extent, and appears to prefer the immediate neighbourhood of the vessels, where large, coarse-fibred spider-cells are also found. Fibres and nests of glia-nuclei occur, however, between the nerve-bundles. In general, the rule is that in progressive paralysis the molecular layer is most involved, then the granule layer, more in spots, and last and least the medulla. Hence it is the dendrites of Purkinje's cells which appear to be chiefly affected in this disease, and their bodies also vanish in advanced cases. The morbid process thus seems to advance from without inwards. Little clinical value is claimed for these results, owing to the irregular distribution and frequently slight degree of the foci of disease; but it may be supposed that the changes contribute to the ataxy and incoordination. The paralytic seizures are more likely, from these cases, to be connected with diseased foci in the thalamus. No relation could be detected between the cerebellar changes and absence or increase of the reflexes.

W. R. DAWSON.

The Topography of Degeneration in the Cortex of Paralytics in relation to Flechsig's Association Centres [*Die Topographie der paralytischen Rinden Degeneration und deren Verhältniss zu Flechsigs Associations-Centren*]. (*Neur. Cbl., No.* 2, 1902.) *Schaffer, Karl.*

Dr. Karl Schaffer, of Budapest, gives the results of his examination of the brains of three general paralytics. His paper is illustrated with five lithographs, showing sections of brain stained by Weigerts-Wolter's method. The degenerated parts take on the stain poorly. Schaffer finds the most degenerated parts in general paralysis to be the anterior and basal portions of the frontal lobes, the whole parietal lobes, the posterior median convolutions, the insula, and the temporal gyri, and the occipital lobes and the upper surface of the cerebellum. Less affected were the anterior median gyrus, the margins of the calcarine fissure, and the inferior occipito-gyri. This showed that degenerative process most affected the association centres of Flechsig, his sensory spheres being very much less touched. Schaffer holds that the degeneration of the cortex in general paralysis is not haphazard but selective. He upholds Flechsig's views, and considers that they have been confirmed by the recent researches of Ramon y Cajal, who has made an original study of the nerve-tissues in the fœtus and in the newly-born child. The latter describes a specific plexus of centripetal nerve-fibres, which terminate in the motor area of the cortex, in the sphere of bodily sensibility, and in the visual area. It is significant that this plexus does not pass into Flechsig's association centres, confirming Schaffer's observation of the posterior median convolution being, in general paralysis, much more degenerated than the anterior. These considera-