field labourer forty-three years old (apparently of normal intelligence (Eichler), and a boy twelve years old of average intelligence, but colour-blind and disliking music (Klob).

The case described by Malinverni is put aside. This man, who had been a soldier, said to have been at one time melancholic, was thought of normal intelligence during most of his life. These results are confirmed by cases of destruction of the trabs through disease, and show that this structure is not necessary to the performance of the mental processes of ordinary life. Several of the individuals in whose brains the corpus callosum was found absent had reached an age between forty and fifty years. One man was as old as seventy-two.

It is to be regretted that those careful researches have not yet revealed to us the function of this great white tract of nerve-matter which holds the two hemispheres together. WILLIAM W. IRELAND.

A Contribution to the Study of the Cerebral Localisation of Chorea and Epilepsy [*Contributo allo studio delle localizzazioni encefaliche nella corea e nella epilessia*]. (*Riv sper. di Freniat., vol.* xxix, *fasc.* 3). *Ravena, E.*

The author describes a case of chorea followed by epilepsy, a somewhat rare association, and gives a detailed account of the macro- and microscopic examination of the brain. Before bringing forward his own case he makes an excellent summary of what has been published on the subject, dwelling more especially on the work done on chorea alone. He believes that the association of this latter with epilepsy is due more to the intensity or extension of the primary originating lesion than to any difference in the causation of either. He mentions among other theories as to the pathogenesis of chorea that of Koch—that all cases are due to a "chorea virus" acting on the nerve centres. Berkley held that the origin was infective, and found in a case of chorea with acute endocarditis lesions of the cerebral vessels and nerve elements similar to those found in diphtheria. This view is held by many. Cesaris-Demel has published a case of chorea caused by an encephalo-myelitis arising from infection by the *Staphylococcus pyogenes aureus*, in which marked lesions of the pyramidal cortical cells were present. Several investigators found atrophy of the convolutions, especially in the parietal region. Murri, from his examination of two cases of polyclonia and four of chorea, came to the conclusion that the motor region of the cortex is the seat of the mischief in both diseases. The author holds the same opinion, and this is pretty generally accepted.

The author's case was an idiot girl, æt. 11 years, on admission into the asylum. Her mental development was very small and her habits extremely degraded and destructive. She was extremely choreic in her movements. Chorea appeared at the age of four, and was attributed by her parents to fright caused by some cows. Her face, head, and all her limbs were a prey to continuous choreic movements. There was no marked defect in her physical development. Eighteen months after her admission into the asylum epilepsy supervened. Her

fits, at first frequent, became rare, but three years after she died in status epilepticus.

At the necropsy, the skull was found much thickened, especially over the frontal lobes. The dura mater and pia arachnoid were also thickened and hyperæmic. The brain weighed 32 ozs. There was marked atrophy of the frontal lobes on either side. Starting from the Rolandic fissure the frontal lobe could be divided into two parts. The first part, comprising the commencing portion of the three frontal gyri, was triangular in shape, and here the thickness of the convolutions was normal. The second part, comprising the rest of the frontal lobes and extending round the anterior curve of the brain back as far as the optic chiasma, was in a state of atrophy. The convolutions were only from 1 to 2 mm. in thickness, of gelatinous consistence and of a whitish colour. The atrophy was symmetrical, and there was a well-defined line of demarcation between the normal and the atrophied part.

On microscopical examination, the atrophied area seemed to be composed of fibrillar tissue of a neuroglia type. Bundles of fibrils could be seen running in different directions: in some places their direction was exactly opposite to the usual one. In the apparently normal part of the frontal lobes the ganglionic cells were small but preserved their contour. They presented an incipient hyaline degeneration, and the nuclei stained diffusely, the chromatic network being observed with difficulty. Here and there round the capillaries in this layer were seen small round uninuclear cells which stained diffusely and were not neuroglia cells, but gave rather the appearance of a connective-tissue arising from the adventitious coat of the vessels, and were apparently of recent origin.

In the atrophied region, with a higher objective, the fibrillar bundles could be seen to form a reticulum, the spaces of which were empty and increased in size the nearer they approached the surface. The fibrils constituting their walls contained numerous nuclei presenting the characteristics of neuroglia tissue. A few larger nuclei could be seen with traces of a chromatic reticulum and retaining a slight amount of irregular protoplasm surrounding them. These were probably the remains of much atrophied nerve-cells, and were only found at the margin of the atrophic region. To sum up briefly, what the author found is as follows:—

A diffuse and well-marked gliosis, and almost complete disappearance of the nerve-cells in the atrophied portion of the frontal lobes on either side: Hyaline degeneration of the protoplasm and nuclear atrophy of the nerve-cells in the part of the frontal lobes not suffering from atrophy, and in the precentral gyrus.

The author holds that the clinical symptoms can be easily explained by what has been found.

Where the atrophy was most marked and where the cells had almost completely disappeared no functionating power was possible. This was the condition of the greater part of the frontal lobes. Hence the true idiocy presented by the patient. In the non-atrophied portion of the frontal lobes and in the precentral gyrus, a moderate degree of protoplasmic degeneration and nuclear atrophy was present in the ganglion cells. The functions dependent on these cells were not

suppressed but merely altered. Hence first the chorea and then the epilepsy. The next question is, What was the primary cause of these cerebral lesions? It is not probable that it could have been due to the thickening of the calvarium, as the atrophy was not limited to the superior surface of the brain but extended round the base as far as to the optic chiasma. The glioses in the frontal lobes should rather be attributed to that anomaly in the development of the nerve centres which is termed microgyria. Different opinions are held as to the pathogenesis of this condition. The most likely hypothesis is that a morbid process, probably of an inflammatory nature, at an early date, attacked a great part of the frontal lobes, causing degeneration and disappearance of the nerve tissue and substitution of neuroglia tissue in its place, as always happens in similar cases. Resulting from this alterations of a less pronounced character occurred secondarily in the cortical motor area which produced, first choreic phenomena, and later either from increase in severity or by extension, the epileptic attacks.

A. J. EADES.

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3. Physiological Psychology.

On the Pathology of the Consciousness of the Ego [*Zur Pathologie des Ich-Bewusstseins*]. (*Arch. f. Psychiat.*, *B.* 38, *H.* 1.) *Pick, A.*

In 1873, Krishaber, under the title of *De la Névropathie Cérébro-Cardiaque*, described a derangement of the recognition of personality of which Taine, in his work on the *Intelligence*, declared that he had found more instruction in it than in a whole metaphysical volume on the substance of the ego. It is not uncommon in asylums to meet with patients who affirm that they have become another person that they are not, or, as in the old song, they say, "This is no me." They find their feelings and their tastes all altered, or they have no feelings at all in some parts of their body. Their own voice is strange to them. Conflicting influences or unusual desires disturb their mind. Thus the string of sensations and thoughts recognised as belonging to themselves is so altered that it is only a slender thread of memory, or the repeated recognition of others, which sustains the sentiment of continued personality. More rarely this sentiment is wholly lost, so that they insist that they are some quite different person. Professor Pick has described an instance of this kind. The patient was the wife of an inn-keeper. She was thirty-three years of age. She had for some time been in weak health, when suddenly there came over her a feeling as if she had lost the current of her thoughts; it seemed as if the thoughts were not her own. When she walked she knew that her legs carried her, but it seemed as if they moved of themselves. Her actions and dealings did not seem to proceed from her own agency. It was not her mind—her thought (she had the sentiment of not being the same person)—her very dreams were altered. When she did not go about or do something she did not know that she was in the world. She said, "I do not at all recognise myself." This condition was worse in the afternoon. It