may have been agenesic. A defective endowment could be ruled out almost positively in 8 cases, though 7 had hereditary history; but the conclusion that the lesions were acquired could only be absolutely adopted in 1 case. Of the remaining 4, 2 had a background defective + heredity = agenesia + aplasia, and 2 had insufficient history.

The pathologic findings were uniform and essentially chronic. The duration varied from 4 to 33 years, with an average of 13·6. There resulted atrophy of the nerve-cell body and its nucleus, disappearance of stainable substance, attenuation with partial fragmentation of neurofibrils, and atrophy with distortion of protoplasmic prolongations, the termination being either (1) extreme pyknotic atrophy, the cell and prolongations covered with incrustations; or (2) the fragmentation of the cell to a more or less shadowy outline. The neurofibrils were also fragmented, the changes being unlike those in general paralysis of the insane, senile dementia, or Alzheimer's disease. The more acutely altered nerve-cells showed fatty deposits which even filled the cells and extended to the prolongations. The glial nuclei, especially in the molecular and infrastellate layers, showed irregular stippling with fine fat granules. Lipoidal matter was largely manifest in the adventitial cells of blood-vessel walls or in their luminæ. Regressive alterations occurred generally in glial structures—in the glial nuclei of both grey and white matter atrophy or varied stages of fibre-formation leading to foci of gliosis; and increase in the surface mat. Amœboid glial cells were of special type diverse from recognised acute terminal manifestations. Satellitosis was negligible. Where the process was most acute neurophages were frequent in all layers closely applied to nerve-cells or in lacunæ of their protoplasm.

In three cerebella nervous tissues were destroyed, especially at the convolution summits where Purkinje cells were extremely atrophic, pyknotic or patchily lost.

Regionally the frontal areas, but occasionally the central, suffered most severely, and the changes in the right hemisphere were surprisingly less established. Statigraphically the greatest involvement was in the first three nerve-cell layers, decreasing and less diffuse toward the third stratum. The stellate layer was singularly fragmented. The process is probably initiated as moderate swelling of both cell-body and nucleus, succeeded by gradual disintegration of the chromatin framework. It is concluded that these changes may be assumed pathognomonic of dementia præcox. JOHN GIFFORD.

Studies in the Pathology of Dementia Præcox. (*Proc. Roy. Soc. Med.*, 1920, *vol. xiii., Sect. Psych., pp.* 25–63.) *Mott, Sir F. W.*

Sir F. W. Mott made a histological examination of the generative organs in many cases of dementia præcox, and his investigations showed a varying degree of regressive atrophy of the seminiferous tubules in all the cases. Corresponding changes have also been found in the ovaries of female patients. The author, however, does not consider that this regressive atrophy of the reproductive organs is the cause of the mental changes by its disorganising influence on the chemical balance of the body, though he admits that auto-intoxication may act as

an important contributing factor in certain cases. In his opinion the atrophic phenomena in the sexual glands and the degenerative changes occurring in the brain cells are concomitant manifestations of a lack of durability germinal in origin.

It is essential to recognise two morbid conditions affecting the neurones which are related to functional disorders or organic disease—the one, characterised by changes in the arrangement and quantity of the basophil substance (chromatolysis), indicates *suspension* of function; while the other, showing biochemical changes, points to degeneration or even death of the cell (*suppression* of function).

Mott made personal observations of the brain in eight cases of dementia præcox and found widespread alterations in the neurones. Not only were there morphological and biochemical changes in the nucleus, but there were also morphological and biochemical changes in the cytoplasm of the cell and its processes. The cortical cells were most affected, but less marked changes were also noted in the basal ganglia, the stem of the brain, and the medulla oblongata. Importance is attached to the fact that the changes mentioned were observed in the stellate cells of the layers of granules, as these have not been noted by the majority of authors. These cells are the intercalary neurones and constitute the second type of Golgi, which form a synapse with the first type of Golgi. In some cases deficient development of many neurones was observed, notably in the small and medium-sized pyramids in the frontal lobe. The neuroglial cells showed proliferation. In none of the cases was there thickening of the membranes of the brain or changes in the vessels.

The author demonstrates how these morbid changes in the brain and reproductive organs may be correlated with the characteristic symptoms of dementia præcox. It is not uncommon for psychic abnormalities to precede the real development of dementia præcox ("dementia præcossissima"). Probably such cases would be found to present not only deficient neuronic development but also arrest of spermatogenesis: "and at puberty, when the stress of productive energy of the reproductive organs occurs, nuclear neuronic decay sets in." When the mental symptoms first appear at puberty or early adolescence the progressive deterioration which ensues may be correlated with regressive atrophy of the testes. This failure of the reproductive organs points to a germinal defect.

Suspension of neuronic function, indicated by chromatolytic changes caused by auto- or hetero-intoxication, is capable of remission and even of recovery; but *suppression* of function, characterised by biochemical changes brought about by a germinal lack of durability, shows no remission, the psychic deterioration being continuously progessive (dementia simplex). Those cases of dementia præcox in which remissions or partial remissions occur, or sudden changes from apathy to impulsiveness, would probably be found to present both these conditions associated in the same subject.

As is well known, cases occur presenting all or many of the clinical symptoms of dementia præcox, but which, nevertheless, recover. It is assumed that in such cases the neurones would show chromatolytic changes, implying suspension of function. In regard to the morbid

changes in the intercalary neurones, Mott says: "The affection of the stellate intercalary cells which enter into the synapse . . . suggests that a hypofunction or suspension of function of these neurones would lead to a synaptic dissociation and thereby account for psychic dissociation and the coming and going of symptoms; or where there is a permanent morbid change, to a suppression of their function with permanent dissociation."

In one of the cases (a monorchid) investigated by the author the suprarenal gland on the side of the absent testicle was found to weigh one-half that of the opposite side, and the cortex adrenalis was much diminished in quantity. This deficiency of the cortical lipoid substance was also observed in other cases of dementia præcox. These findings are significant in view of the fact that there is much evidence to show that the adrenal cortex influences the genital function, though it is not certain whether it acts alone or in conjunction with other endocrinic glands, *e.g.*, the thyroid and pituitary. Among the functions attributed to the suprarenal cortex are the manufacture of lipoids of the body and the building up of myelin of the nerve-fibres of the brain.

It is clear that the further investigation of the *rôle* played by the glands of internal secretion should not only prove to be of considerable scientific interest, but should, at the same time, be able to throw much light on the ætiology of dementia præcox.

NORMAN R. PHILLIPS.

Epidemic Encephalitis and Katatonic Symptoms. (*The Amer. Journ. of Ins., January*, 1920.) *Bond, E. D.*

The writer through illustrative cases points out the relationship of epidemic encephalitis to the katatonic psychoses, and propounds the plausibility of cerebral disturbance as a basis for katatonic episodes. There proved to be many features in common, and especially abnormalities of muscle function. He asks for a unified and definitive terminology, rendering possible the exact description of postures, tensions and other activities of muscle, as found, *e.g.*, in katatonia. No observer, however careful, can convey with the existent generalized vocabularies any exactitude of clinical visualization to the reader. Granting this as achieved, it is further essential that there should be methods of examination and diagnosis standardized in succinct fashion, so that the findings of various investigators can be relied on to express the same phenomena. Control and description of the surrounding circumstances is also necessary. He instances Langelaan's method of recognising hypotonia as a case in point, while admitting that in the hands of different workers lack of thoroughness may result in this term being used for very varying degrees or even actually diverse conditions. He pleads for the institution in mental hospitals of conditions which will permit of thorough repeated examinations, even, be it noted, in long-standing chronic cases. And he foresees therefrom a rich harvest of reward. Mild transient but definite symptoms are usually missed in excited, seclusive, or indifferent patients. Obscure explanations are only justified, and should only be sought, when the above methods fail to elicit causation. He supports his contention by a graduated series