The writer some years ago pointed out the desirability of trying to limit the obvious causes working in Ireland which tend to produce insanity, crime, and degeneracy. Foremost amongst these is the continued and wilful neglect of the care and control of the feeble-minded. An effort ought, too, to be made to set up some form of specialised colony for the treatment of epileptics. Legislative changes must be initiated by those whose life interest has been the betterment of the insane, and whose experience and knowledge entitle them to deal with so difficult and specialised a subject.

C. W. FORSYTH.

*Luminal in Epilepsy. [Un Traitement efficace de l'Épilepsie : la Phényl-éthylmalonylurée ou Luminal.] (L'Endphale, July 10th, 1920.)* 
Maillard, G.

Those who discussed this paper at the Société de Psychiatrie of Paris agreed that luminal is very efficacious as a preventive of major fits, but appear to have spoken chiefly of its aptness to produce mental disturbance—alteration of character, irascibility, impulsiveness, excitement and violence. Rogues de Fursac said that at the Ville-Evrard asylum Ducosté had observed these effects, good and bad, even with minimal doses, never exceeding 15 cem., and in some instances a mental disturbance really grave—even delirium. Maillard showed that abrupt discontinuance of the drug is apt to provoke numerous fits.

SYDNEY J. COLE.

5. Pathology.

*The Histopathologic Findings in Dementia Precox. (The Amer. Journ. of Int., January, 1920.)* Rawlings, E.

The research occupied nine years. Precautions to prevent post-mortem changes and the inclusion of cases involving psychotic disease processes other than the one under review were as complete as possible. Any case conceivably open to doubt was eliminated. Only cases with a clear precox history were employed. And of 52 cases minutely worked out only 12 were made the basis of this article. The 40 remaining cases gave sufficiently clear indication of the pathological changes which the author regards as typical of this psychosis, although in this larger series masked by changes pathognomonic of other disease conditions. The areas investigated were usually the frontals, centrals, paracentrals, parietals, temporals and cerebellar; the staining methods and technique employed are indicated; the histories, clinical abstracts, and causes of death are outlined; the pathological findings for each case are well detailed and diagrams are appended. The following groups of cases were excluded:

(1) All over 60 years of age, to avoid senile changes other than Alzheimer's; (2) long-standing mixed manic-depressives; (3) paranoid involutionals, possibly due to chronic diseases; (4) imbecilities with frequent disturbance. Of cases under sixty, 10 were rejected for cortical arteriosclerosis (only 1, however, showing nerve-cell devastations). The series of 12 is advanced as probably presenting a disease entity. Ten showed macroscopical atrophies, chiefly frontal; 1 with heredity
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 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 lumina. 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. Amoeboid 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 lacunae 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 praecox.

John Gifford.


Sir F. W. Mott made a histological examination of the generative organs in many cases of dementia praecox, 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