THE PATHOGENESIS OF SOMATIC DISEASE IN MENTAL DEFECTIVES.

By L. CROME, *M.C.*, M.R.C.P.Ed.

From the Department of Neuropathology, The Fountain Hospital, London.

THE problems of the interdependence and unity of the brain and body have been put on a scientific basis by Pavlov and his successors. Bykov (1947) has, for example, been able to demonstrate that the cortex plays a leading part in the regulation of somatic processes, such as secretion of urine, blood pressure, peristalsis and metabolism. It is therefore reasonable to argue that lesions of the central nervous system will be reflected in the pathogenesis and course of morbid processes in the body. It does not follow, however, that this influence will necessarily be in the direction of greater lability, more rapid pathogenesis or more extensive destruction. The outstanding feature of the central nervous system is its plasticity and power of compensation. It is therefore possible and probable that those parts of the nervous system which remain intact will take over and compensate for the function of the lost ones. Emotion may, for example, lead to polyuria, but it does not follow that urinary secretion will be impaired in a leucotomized patient. The brain may well play an important part in the infective processes of a normal person, but the defence against infection in a microcephalic idiot may remain perfectly adequate, and may even be more effective than in a normal person, provided that the mechanism of the immunity and phagocytosis had been more fully mobilized in the course of his previous life.

In assessing the part played by the brain in any alteration of pathogenetic processes, it is important to realize that the nervous system acts not merely by virtue of its purely physiological, i.e., mechanical and humoral, connections with other organs. The brain is also the organ of the mind, and it is the mind which enables man to live normally in society. Disease is not only an altered physiological state, but also a social one. Since it is the brain which enables man to live in society, it is obvious that mental derangement following cerebral disease will alter the patient's social position. It may well be that in some cases it is this social change which plays the greatest part in the altered pathogenesis of physical disease.

The morphological appearances of pneumonia in idiots are often different from those seen in mentally normal children. The lungs of such idiots show a more varied pattern, consisting of lobular collapse, emphysema, inhalation pneumonia, along with patches of early purulent infiltration of the terminal and respiratory bronchioles. It may be that the defective brain contributes to the production of this characteristic picture through impairment of the nervous control, but it is more likely to be the result of the patient's

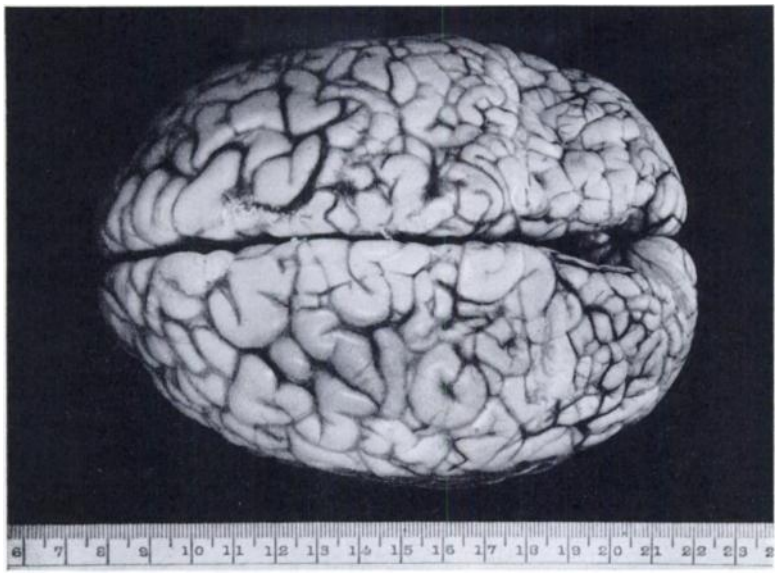

FIG. 1.—Dorsal view of the brain. Ulegyria of posterior part of the cortex.

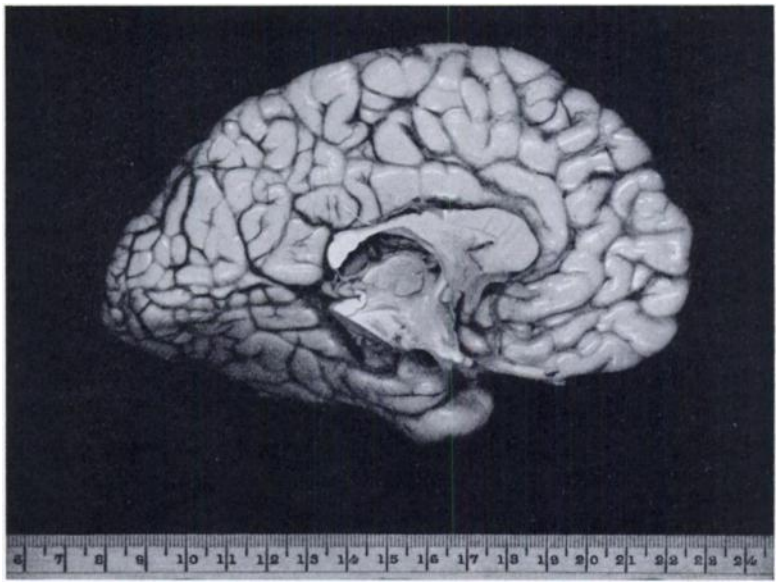

FIG. 2.—Medial surface of the right hemisphere.

https://doi.org/10.1192/bjp.97.409.792 Published online by Cambridge University Press

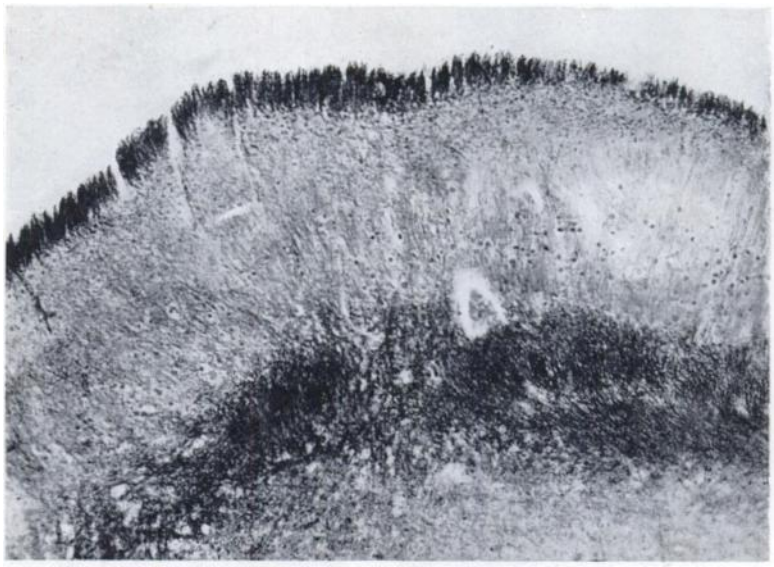

FIG. 3.—Marginal and laminar gliosis of the cortex. Holzer stain. × 60.

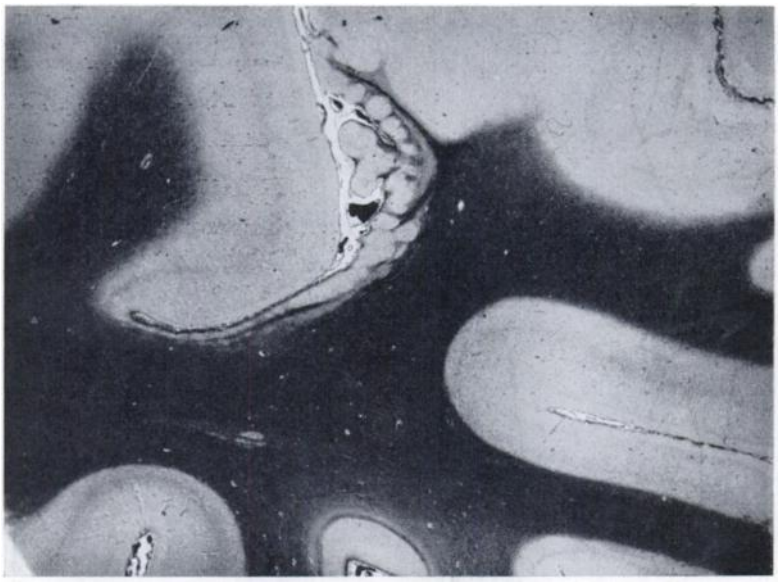

FIG. 4.—Granular atrophy and status marmoratus of cortex. Phosphotungstic acid haematoxylin. × 24.

https://doi.org/10.1192/bjp.97.409.792 Published online by Cambridge University Press

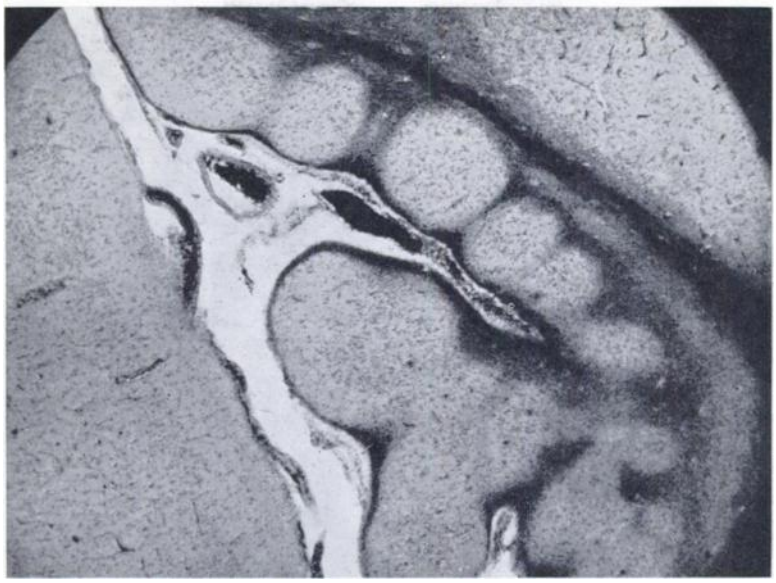

FIG. 5.—Status marmoratus and plaques fibromyeliniques. Phosphotungstic acid haematoxylin. × 50.

FIG. 6.—Periventricular gliosis. Holzer stain. × 60.

https://doi.org/10.1192/bjp.97.409.792 Published online by Cambridge University Press

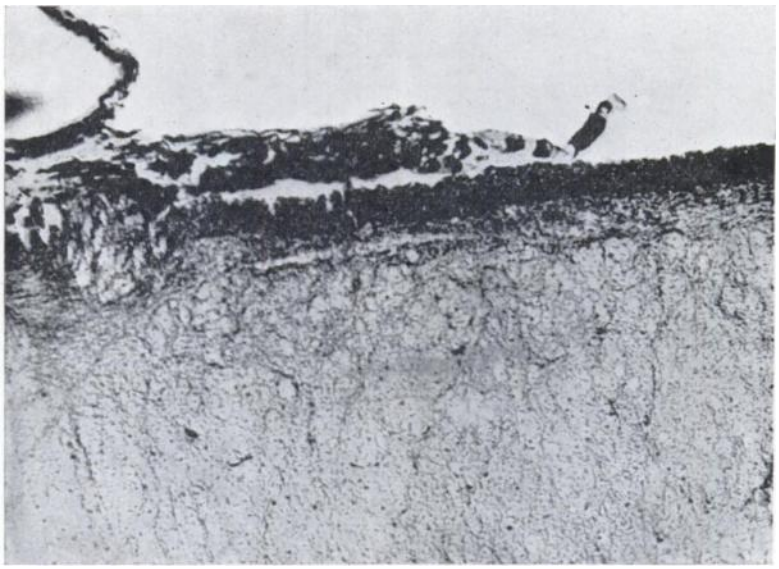

FIG. 7.—Marginal gliosis of brain stem. Holzer stain. × 60.

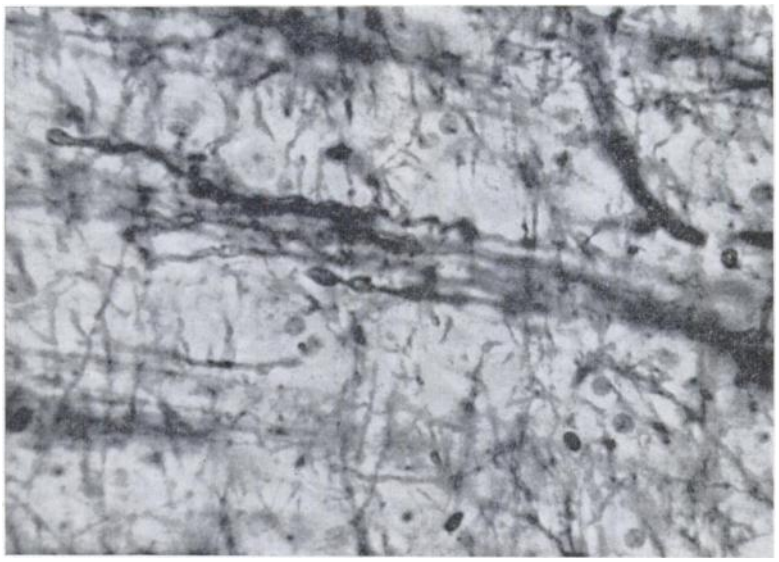

FIG. 8.—Beading and varicose dilatations of myelin sheaths. Heidenhain's haematoxylin. × 700.

https://doi.org/10.1192/bjp.97.409.792 Published online by Cambridge University Press

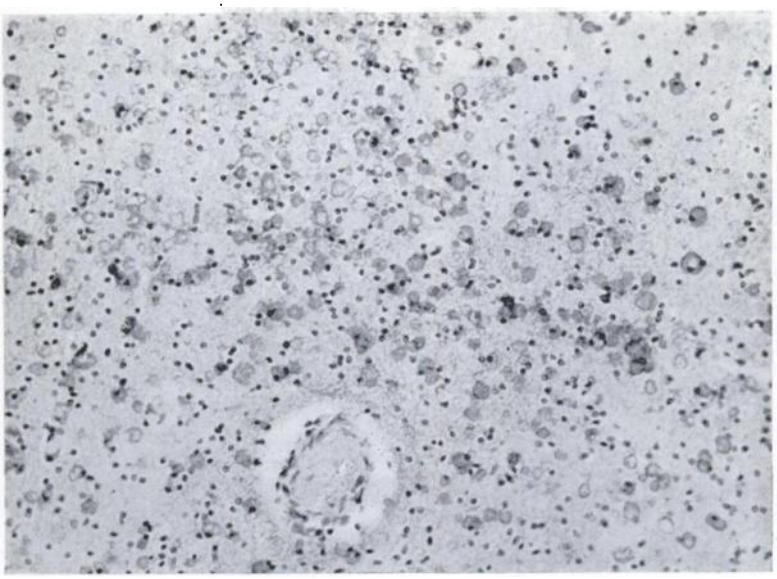

FIG. 9.—Metachromatic bodies in white matter. Mucicarmine and iron haematoxylin. × 280.

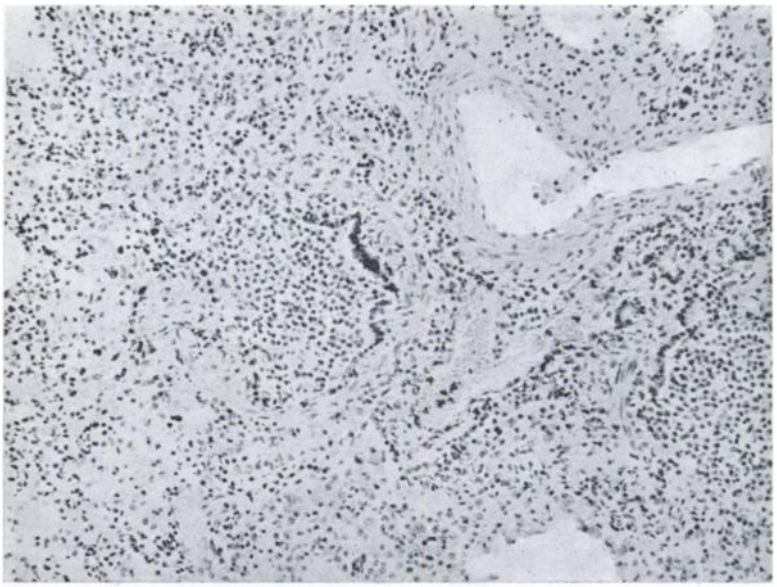

FIG. 10.—Lungs. Early broncho-pneumonia. H. and E. × 280.

https://doi.org/10.1192/bjp.97.409.792 Published online by Cambridge University Press

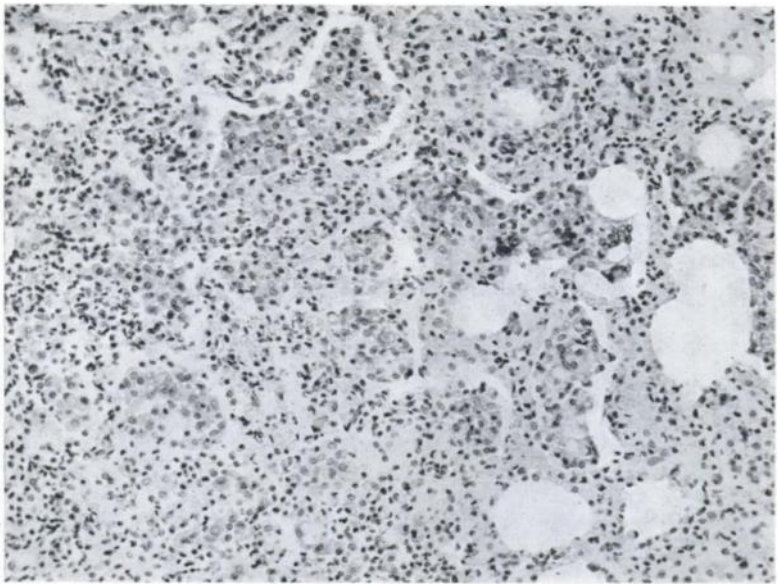

FIG. 11.—Lungs. Inhalation pneumonia. H. and E. × 280.

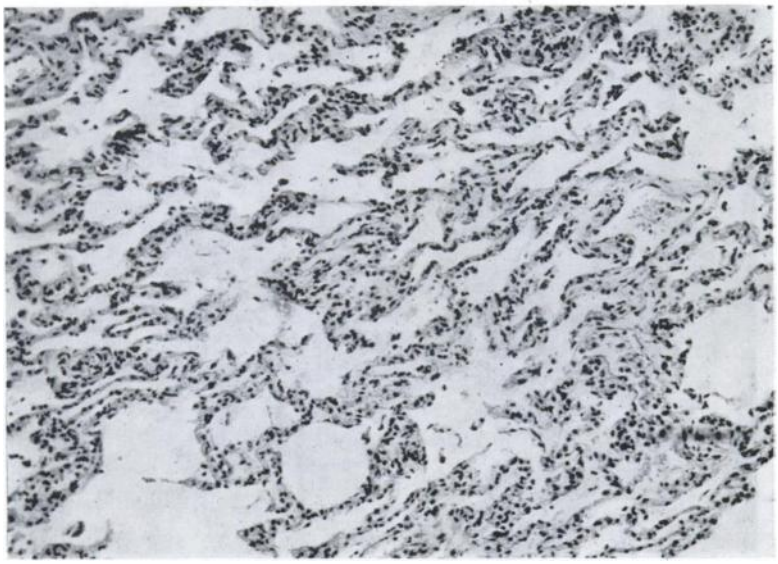

FIG. 12.—Lungs. Partial collapse. H. and E. × 280.

https://doi.org/10.1192/bjp.97.409.792 Published online by Cambridge University Press

immobilization, difficulty in coughing, inadequate nursing and lack of maternal care. The following case illustrates this position.

Case Report.

The patient was the tenth child in a family in which the preceding four pregnancies of the mother resulted in stillbirths. He was delivered by Caesarian section after a seven months' pregnancy. The patient's abnormality was noticed soon after birth, and he was later admitted to the Fountain Hospital with cerebral diplegia, epilepsy and idiocy. He died from bronchopneumonia at the age of four.

Examination of the brain, which was normal in size, showed that the gyri in the posterior half of the brain were narrower and more convoluted than in the anterior half (Fig. 1). The corpus callosum was short and its body thin. The transverse fissure was wide (Fig. 2). Microscopical examination showed widespread cortical gliosis of the narrow gyri in the posterior half of the brain. This gliosis was both marginal and, in places, laminar in distribution, involving layer III of the cortex (Fig. 3). In addition, there were patches of granular atrophy and marbling of the cortex with the presence of plaques fibromyeliniques (Figs. 4 and 5). There was also extensive periventricular gliosis (Fig. 6), and marginal gliosis of the brain stem (Fig. 7). The myelin of the white matter did not appear to be affected when examined with the naked eye in appropriately stained sections, but microscopically it showed beading and varicose dilatations of the myelin sheaths (Fig. 8). Diffusely scattered metachromatic bodies were seen throughout the white matter (Fig. 9).

The lungs showed in a few places the characteristic changes of early bronchopneumonia (Fig. 10). In other areas the alveoli were filled with masses of mono- and multinucleated phagocytes containing debris of inhaled material (Fig. 11). There were also areas of lobular collapse (Fig. 12), emphysema and oedema.

Discussion.

The significance of metachromatic bodies in the white matter of the brain is still uncertain (Smith, 1949). The remaining structural changes are, however, familiar findings in the brains of idiots. They are suggestive of the occurrence of some destructive process during the later stages of intra-uterine life or at birth.

The pulmonary lesions in this case are consistent with bronchiolar infection combined with inhalation pneumonia and lobular collapse following bronchiolar obstruction.

It may be supposed that the gross cerebral damage present in this case had contributed to the establishment of some defect in the autonomic innervation of the lungs and to the impairment of defence mechanisms in it. Such a possibility cannot be excluded, but there is no positive evidence to support it. On the other hand, an acquaintance with institutional life and the knowledge of the patient's physical and mental disability afford a simpler and more plausible explanation of the changes in the lungs. They can be best explained by the patient's difficulty of coughing and clearing the obstructed bronchioles,

along with inhalation of food and a superadded bronchiolar infection, or, in other words, by factors closely related to the specific social environment of the patient.

The modifying influence of an anomalous social position on the pathogenesis of somatic disease may be more apparent in some cases, while physiological processes may be more evident in others. This variation in the degree of their relative impact cannot justify their separation. The two remain interrelated. Thus the immobilization of a patient with quadriplegia or in extreme catatonia may lead, even in the absence of cortical damage, to as much change in social status as microcephaly.

It can be concluded that lesions of the nervous system may modify the course of physical disease in the body either by their effect on physiological processes or through the mediation of social factors. It may be more the one or more the other in different cases, but usually it is a combination of both. Both form an integral part of the natural history of disease. Society is, after all, no less a product of life than phagocytosis or bacterial immunity. Social factors therefore rank equally with physiological ones in the complex of pathogenetic change.

Summary.

Lesions of the nervous system may influence the pathogenesis of somatic disease not only through their effect on physiological processes, but also by the alteration of the patient's social environment. The two sets of processes are not independent of each other. Both form an integral part of the natural history of disease. The effect of a characteristic social environment on the pathogenesis of lesions in the lungs is illustrated by a case report.

References.

Bykov, K. M., *Kora golovnogo mozga vnutrennii organi*, 1947. 2nd ed. Moscow and Leningrad : Medgiz.

Smith, M. C., *J. Neurol. Neurosurg. Psychiat.*, 1949, **12**, 100.