SOME PRESENT-DAY VIEWPOINTS IN EPILEPSY*
A SYNOPSIS OF THE PROCEEDINGS OF THE GESELLSCHAFT DEUTSCHER NERVENÄRTZE HELD IN DÜSSELDORF IN SEPTEMBER, 1926
BY HENRY A. BUNKER, M. D.,
ASSISTANT DIRECTOR, NEW YORK STATE PSYCHIATRIC INSTITUTE

Spielmeyer deals briefly with the histopathological aspects of epilepsy, asking: What pathological changes do we find associated with epileptic convulsions, to what extent is it possible for research in this direction to elucidate their genesis, and do the results of such investigation throw light on the pathogenesis of the epileptic seizure?

The immediate brain changes which occur after a seizure are both of a generalized and diffuse and of a localized character. The former consist, according to Alzheimer, of acute ganglion cell changes, axis cylinder degeneration, nuclear division in the glia cells, and in especially severe cases a regressive transformation of the glial elements into amoeboid forms. But these changes have thrown no light on the pathogenesis of the seizure because we have not been able to interpret them. The localized changes, on the other hand, are capable of interpretation in this sense.

Spielmeyer has found circumscribed changes in the cerebellum and in the horn of Ammon. The former he has previously described as consisting of a shrub-like proliferation of the glia cells with numerous mitoses, this occurring in those places where the Purkinje elements have undergone destruction. With the increase, with successive convulsions, of the areas in which the latter have become replaced by glial proliferation, there come into existence areas in which the Purkinje elements are entirely lacking. These patchy and eventually sclerotic areas, which are often overlooked but which are nevertheless extremely frequent in epilepsies of all types, may be traced back to the cumulative tissue loss (Purkinje elements) associated with the seizures. It is Spielmeyer's conjecture that the Ammon's horn sclerosis may be analogous to these circumscribed areas of sclerosis in the cerebellum, and that their origin may possibly be ascribable to similar acute changes. Among 36 cases of status epilepticus he found acute changes in the typical regions of Ammon's horn in six, three of which were cases of idio-

* The first part of this synopsis appeared in the Psychiatric Quarterly for July, 1927.
pathic and three of symptomatic epilepsy. In these cases a process of fresh degeneration was encountered in those parts of Ammon's horn which in Ammon's horn sclerosis show definite tissue loss with gliosis. One can follow the diminution and disappearance of the ganglion cells, and the reaction of the glia in the form of proliferation and inclosure of the degenerating nerve cells.

Associated with epileptic seizures there occur, therefore, in addition to diffuse changes demonstrable with difficulty, localized areas of tissue loss in the cerebellum and in the horn of Ammon. After status lasting some days and after seizures which precede death by a few days, these can be demonstrated in the majority of cases, sometimes only in Ammon's horn or only in the cerebellum, sometimes in both. Spielmeyer, in his material, found changes such as these, associated with convulsions, in 80 per cent of cases of both idiopathic and symptomatic epilepsy.

For the demonstration of the part played by the pathological changes in the pathogenesis of the disease two methods are open: To discover the basis of the site of election of these alterations and to ascertain the manner in which these changes evolve in loco. Spielmeyer has already employed the first of these methods, having some time since reported that the areas of tissue loss in Ammon's horn in epilepsy correspond in their regional distribution with the similar areas conditioned by circulatory disturbances, such as those which occur as a result of arteriosclerosis or endarteritis. Hence the conditions with reference to the local circulation are the preponderant factor in the involvement of these areas, which, as we have seen, are about always the same in situation; and this is true even when there are certain variations in the latter respect. This follows also, indeed, from the investigations of Uchimura upon the blood supply in the region in question (diminished or inconstant blood supply in the situations above referred to, and an abundant blood supply in the more resistant areas). Thus it is possible to conclude that the site of election of the pathological changes is governed not by physico-chemical considerations, but by vascular and circulatory disturbances.

Regarding the character of the structural changes, the ganglion cell appearances are those of ischemic degeneration. Around the cell shadows glial rods later proliferate, in which lipoid catabolites become stored up and which take part in the removal of the waste