THE INFLUENCE OF FUNCTIONAL CIRCULATORY DISTURBANCES ON THE CENTRAL NERVOUS SYSTEM.*

By Prof. Dr. W. SPIELMEYER,
München.

IF I may be allowed to speak to you about the importance of functional impediments to the circulation, I would like to do so from the point of view of the anatomist. I shall not speak about the clinical and physiological observations in neuropathology and internal medicine; I want rather to begin with the deductions to which the anatomist is led by his own methods, independent of clinical facts and questions.

I mean to discuss those alterations of the brain which are recognized by pathogenetic analysis as results of disturbances of the circulation. These alterations are not such that we can base a definite diagnosis upon them or delimit a process with exactitude. By these means they can be considered only as results of identical or similar causes, and can be explained in this way on a patho-physiological basis. Anatomy has lately made progress in this direction which may be useful for the understanding of some mental and nervous diseases.

Anatomical investigation shows that a variety of changes can be of circulatory origin, without, however, there being any change in the size of the lumen of the vessel nor any pathological changes in the vessel wall. It can also be proved anatomically that a non-organic obstruction to the circulation may cause a degeneration of the central nervous system—in other words, a disturbance of the circulatory *function* only may produce similar conditions to those which are caused by an organic occlusion of circulation.

The first set of photographs will show you this. The lesions appear the same, and yet one has resulted from a distinctly recognizable occlusion of the vessel, and the other has developed without such

* Paper, illustrated by lantern demonstration, read at the Annual Meeting, Oxford, July 2, 1930.

https://doi.org/10.1192/bjp.76.315.641 Published online by Cambridge University Press

an obstruction. In cases of arterio-sclerosis with hypertension, I have long observed that foci of alterations are found not only within the reach of vessels narrowed by the arterio-sclerotic process, but also in parts of the brain which are supplied by intact arteries.

In the first slide one focus is the result of an artery very much narrowed by arterio-sclerosis. In the other the pial vessel belonging to the focus is not altered, and its branches also are normal. On the next slide we see another form of degeneration—a pallor of the cortex extending over several convolutions of the occipital lobe, the acute nervous disease having existed only four days, in a case of arterio-sclerosis with hypertension. This alteration could be explained by no organic abnormality of the blood-flow. In the adjacent photograph the pallor of the cortex is caused by an embolus.

It is not necessary to-day to bring forward fresh proof for these similarities in the effects of organic and of functional alterations in the circulation. To-day it is more important for me to show how from the most varied causes damage, strikingly similar in appearance, can result to the brain.

As a complement to the photographs of arterio-sclerotic dementia with hypertension previously shown, the next slide will show you a softened area in the brain of a youthful patient, with hypertension, without any arterio-sclerosis. The triangular focus which fills the whole width of the cortex proceeds as a widely extended strip into the subcortical white matter. The softening, characterized by destruction of granular cells and by proliferative processes, especially in the mesenchyma, occupies only the marginal zones; the main part of the focus shows only necrotic tissue changes.

The next slide is from an *eclamptic pseudo-uræmia*. Since, according to our experiences, the cerebellum here is very often involved, I am showing a photograph of that structure. It shows the final state of necrobiosis extending over several adjacent lobules.

The next slide is from a case of *eclampsia of pregnancy*. The photograph shows the characteristic focal and streaky discolourations. They conform perfectly to the pictures produced by other causes, but in pathogenetically identical conditions. This can be proved by the following photograph. This picture shows about the same alteration. This is from a case of *eclampsia in whooping-cough*, in which I believe that the lesions described in our Research Institute are due to functional circulatory alterations. I think that the same pathological mechanism acts in all epileptic attacks. As an example of this I bring a picture of a *symptomatic epilepsy* in *infantile cerebral*

paralysis from my former studies on epilepsy. Here one sees in the cornu Ammonis, at the typical sites—that is, in Sommer's sector and in the end plate—the characteristic nerve-cell defect, just as in other epilepsies. This circumscribed defect is, as I have shown before, identical with that which we found in *coarse substantial obstructions* to the circulation.

Further, I bring only one picture of intoxications. Here the character and spread of the alterations is the same, in spite of the quite different original cause. This preparation is from a case of acute *intoxication by pantopon* (*omnopon*).

The next photograph, finally, demonstrates the essence of the alterations. A man, æt. 63, previously healthy, suddenly became ill with rapidly increasing mental confusion. Some days later he became inactive, drowsy and finally somnolent. The day before his death he had epileptic attacks. The heart and kidneys showed nothing abnormal. The necropsy also showed an intact vascular system. Microscopically there were found widely extended fresh necrobioses, partly in the form of pale spots, partly with beginning phagocytosis and organization.

These are the stated facts. What do they mean? The conclusion is evident that for diagnosis or differential diagnosis these lesions are quite useless. We find the same alteration in a traumatic lesion, CO-intoxication, chloroform narcosis, hypertension, pseudo-uræmia, and after other spasmodic conditions. We see always the same alterations. They vary only according to their age and intensity. When they seemed to be different in their localization, and to have something really typical in them, further experience showed that this was an error.

So these findings are of no value to the definition and classification of disease entities. Their fundamental value rather consists in showing something common to all those diseases ætiologically and clinically so very different; not anatomical forms of diseases, but pathogenetically similar symptom-complexes. The essential nature of these showings is the predominance of central circulatory alterations in brain injuries caused by the most varied noxa, such as trauma, intoxications, infections, hypertension or eclampsia. It can be seen from this how the most disparate injuries by a more or less long chain of causes finally lead to the same pathophysiological process.

From this we may conclude as to what meaning these determinations have in neurological and psychiatric questions. I think they give us an understanding of the essence of these brain alterations.

And besides, the pathogenetic analysis leads us, through the knowledge of the development of brain conditions, into pathophysiology, and so to the understanding of many clinical symptoms.

We try to make use of the microscopic showings in the interpretation of clinical symptomatology. After all I have said before, this can only be done in co-operation with the clinician. He tells us if focal symptoms of the aforementioned kind are present, such as apoplexies, paresis and fits in youthful hypertonics, in eclampsia, eclamptic intoxications and traumatic conditions. The findings demonstrate the anatomical background of such circumscribed cerebral manifestations. For long we have neglected to consider the fresh necrobioses which appear only as a faint pallor, but many cases of so-called "hemiplegia without anatomical findings" can be explained by these pallors, which are visible only in Nissl preparations. It is the same with general symptoms of cerebral origin—sudden drowsiness and coma, or excitement with anxiety and signs of acute delirium. Such general cerebral symptoms can be in relationship with widespread vasomotor disturbances, which have acted on large parts of the brain-tissue. In most of the cases a special vessel district, as, for instance, the district supplied by the arteria cerebri media, was especially involved on one side. Sometimes the same region on the opposite side also showed some damage, and in a few cases there were alterations in very different vessel districts.

We have also known psychic symptoms, for instance in intoxications, which according to our experience we connected with circulatory damage, but which showed no visible morphological substrate. We must suppose that here the central circulatory disturbances were not yet strong enough to destroy the nervous tissue. To produce visible alterations in the tissue, a certain intensity of circulatory deficiency is needed.

Such cases explain the possibility of a total recovery, and of the disappearance of menacing symptoms.

In morphine intoxication, for instance, these findings are very impressive. Dr. Weimann has demonstrated that this latter acts mainly by disturbances to the circulation, in spite of their being accompanied by direct toxic influences. From the appearance and disappearance of symptoms, as well as from the often observed increase and decrease of their intensity, we can conclude that the angiospasms or stases are varying in degree. This may explain hemiplegia, its varying intensity and its final disappearance, as well as the different degrees of unconsciousness, its increasing into

coma and its final recovery. The angiospasm and angioparalysis can be followed by a recovery of the vasomotor functions.

I think I need say no more about the connections between clinical symptoms and anatomical statements. I hope I have shown how our anatomical results can be used in clinical questions.

As I have already said, the search for the ætiology of disease processes can only be done in co-operation with the clinician, who sees much better with what other symptoms the vasomotor processes are connected.

There is, in general, no other topic in neurology and psychiatry for which the anatomist is so much in need of the help of the clinician in order to analyse his findings successfully.