THE ROLE OF PSYCHICAL FACTORS IN THE PRODUCTION OF ORGANIC NERVOUS DISEASE.*

BY

ABRAHAM M. RABINER,

AND

MOSES KESCHNER, NEW YORK.

INTRODUCTION.

IN the course of routine neurological practice many patients are encountered who present no objective findings to account for their complaints. These cases are then designated as 'non-organic,' 'functional,' 'hysteric,' 'psycho-neurotic,' or 'psychasthenic,' and treated as such.

To the chagrin of the physician and discomfort of these patients many of them return after varying periods of time with undoubted evidences of organic nervous disease. The objective neurological findings which they then present can be readily correlated with the original complaints, and may be included in the concept of some well-defined organic nervous disease. It is then that one is confronted with the questions as to the relationship, if any, between these two phases in the patients' clinical course. In other words: (1) Is the later appearing organic phase independent, coincidental, or superimposed on the earlier, so-called non-organic phase? or (2) do both of these phases represent one and the same clinical entity, the second phase being present at the very onset of the disease, but that the objective manifestations were so slight or so overshadowed by the non-organic component as to escape clinical detection? (3) Is there any etiological relationship between the non-organic and organic phases?

Our clinical experience with some of these cases would seem to suggest that the evolution of some organic nervous diseases may occur in two phases; (1) the early, psychical, and (2) the later, organic phase. The transition between these two phases is imperceptible and can only be appreciated when the patients remain under prolonged observation by the same observers. We have had an opportunity to observe a series of such patients (*vide infra*) in whom after prolonged observation the possible occurrence of such a transition has suggested itself. These patients when last seen by us presented a symptom-

* Read by Title at the Fifty-fifth Annual Meeting of the American Neurological Association, Atlantic City, N.J., May 27, 1929.

* From the Neurological Services of Mount Sinai and Montefiore Hospitals, New York City.

complex with structural changes in the nervous system at the basis of it. The first question then arises: What, if any, is the relationship between these structural changes and the initial psychogenic manifestations?

In our opinion not only may change of structure influence function, but function may also influence structure. Our cases would seem to suggest that early functional disturbances in various segments of the body may be followed by changes in the anatomical structures at the basis of the neural mechanisms subserving these segments. Unfortunately in our present state of knowledge such transition does not lend itself to histological demonstration. General pathology and physiology, however, offer abundant evidence of the effect of disturbances of function on structure.

INFLUENCE OF FUNCTION ON STRUCTURE.

The increase in the size of cells of hypertrophied muscles and in hyper-functioning glands is too well known to require further comment. The experiments of Hodge¹ show what occurs in the morphology of nerve structures in cases of increased activity. This observer found changes in the size and shape of the nuclei as well as of the protoplasm of ganglion-cells of various animals (frog, cat, dog, sparrow, pigeon, swallow, and honey bee) after prolonged electrical stimulation, and after normal fatigue. Verworn² and Ziegler³ showed the occurrence of morphological changes in nerve elements during and after their prolonged activity. The views of these observers were later confirmed by Agduhr⁴, who showed that in mice after training there was an increase of the number of nerve-cells in the ganglia, and that the number of axons were increased, and the neurofibrillary mass in the nerve-cells was considerably larger in size. Beritoff⁵ believes that in the most active nerve paths of congenital as well as acquired reflexes the neurofibrillary mass and the neuroplasm of the nerve elements as well as the permeability of their plasma membranes are much greater than of those in the less active tracts—so much so that the electric resistance of the former is far less than that of the latter. Besides this a still more characteristic morphological change may occur during increased activity. We refer to the physiological development of nerve tracts, which has been shown by Kappers to exist during such increased activity. This author was able to show that during a continuation of stimuli in the nervous system there occurs an outgrowth of the principal dendrites and eventually a prolongation of the cell body in that direction from which the greatest number of stimuli come to that cell. Similarly the axis-cylinders actually grow in the direction of the stimulus. The physiochemical process at the basis of this phenomenon he designates as neurobiotaxis.

The effect of hypo- or afunction on morphology may be illustrated by what has been designated retrograde degeneration. Thus Berard⁶ found distinct atrophy of the ventral nerve roots supplying a limb that had been amputated some time before. Marinesco⁷ found that after amputation of a

limb, or after section of a peripheral nerve, there occur in the central part definite pathological changes. The younger the individual at the time of the amputation and the longer the time elapsing between the operation and death, the more marked are the alterations. The degeneration in the central stump of a divided nerve, although it appears much later than in the distal stump, presents similar morphological appearances, and is apparently an analogous process. The motor fibres of the central stump gradually diminish in number and, in some instances, appear to vanish entirely, and a large number of the motor cells of the ventral horns dwindle in size and may after a time be actually lost.

Gudden, Forel, Maper, and Mendel experimented by tearing spinal or cerebral nerves away from their connections with the central nervous system, especially in newly born or very young animals. These animals were allowed to live for many months, then killed. Histological examination of the central portion of the nerve involved, together with the group of cells corresponding to it, revealed marked changes in the nucleus of origin. The cells present showed distinct atrophic alterations and many of them had entirely vanished, so that enumeration of the cells of the group concerned revealed a decided discrepancy in the count on the two sides.

Nissl⁸ states that even if the peripheral nerve is not cut through, but is rendered temporarily incapable of functioning, regressive alterations can be made to appear. Flatau, in accord with Nissl's investigation, obtained similar results from a study of the brains of four cats in which the third nerve had been cut intracranially. This has also been confirmed by Sodovsky⁹ and Biedl¹⁰. Warrington¹¹ has shown that if the impulses coming to a cell by way of the dendrites and the cell body be cut off there appear changes in the whole neuron. He found that section of several posterior nerve roots is followed by a considerable degree of cell change. He believes that that is the result of the withdrawal of the afferent impulses which normally impinge upon the cornual cells. Hence the anterior horn cells must be in a highly altered functional state. He concludes that these results are in harmony with those of other observers, and, therefore, accepts the general law that in a cell loss of continuity of its axon is followed by definite structural changes.

As a matter of fact Cassirer states that every dysfunction, whether due to a disturbance in the afferent or the efferent path, or in the processes which are supposed to exist between them, leads to an anatomical change in the structure of the nerve cell. The final outcome of this change depends on whether we are dealing with an irreparable or only a transitory disturbance of function. Similarly, structural alterations may be produced in cases of hyperkinesia of psychogenic origin. One must recognize that merely the pattern of an abnormal movement proves nothing as to its genesis, and will not permit its classification as psychogenic or organic. Epidemic encephalitis has produced many syndromes of hyperkinesia and akinesia which only a few

years ago were classified as purely psychogenic. Studies of extrapyramidal syndromes have been of great interest to neurologists, especially as regards the types of disturbance that seem to form a link between organic and psychogenic affections.

ABNORMAL LIPOID METABOLISM AND THE PRODUCTION OF LESIONS.

The precise underlying factors entering into the production of this phenomenon of transition from psychogenic to organic must in our present state of knowledge remain speculative. That the basis for this phenomenon may have to be sought in disturbances of metabolism may perhaps be inferred from a statement by Adolf Meyer¹² that "Every mental adjustment must be in keeping with the laws of anabolism and catabolism; it has its somatic components" (p. 487). Modern researches seem to point to abnormal lipid metabolism as a genetic factor in the production of degenerative lesions in the central nervous system. Given a patient who has sustained a psychological trauma, is it not conceivable that the latter may influence directly or indirectly the vegetative nervous system, giving rise to metabolic changes which may affect lipid metabolism and so initiate pathological changes in the nerve structures? This hypothesis would seem, on theoretical grounds, to find substantiation in the clinical fact that most of these cases present features of extrapyramidal involvement. The close proximity of the vegetative centres to the basal ganglia which are apparently the site of predilection for the more common lesions of extrapyramidal disease may be more than a mere coincidence. It will remain for the biochemists to prove or disprove this hypothesis.

The relationship of the cortex and the basal ganglia has been stressed by all observers. The influence of the cortex on extrapyramidal motor disturbances is manifested clinically by diminution and at times temporary abeyance of the latter following suggestion, hypnotism and re-education. The relationship of affective processes to lesions in the midbrain and basal ganglia would seem to be of some significance in this connection. The close similarity of many of the symptoms of the various psychoses, endogenous or exogenous, such as the catatonias of dementia præcox, or the stupor of manic-depressive insanity and other psychoses, to similar states observed in organic lesions of the basal ganglia, as has been stressed by Henneberg, Jakob, Schilder, and others, is also suggestive of the close functional relationship between this component of the central nervous system and the psyche. We need only mention the clinical resemblance of the rigidity of catatonic dementia præcox ('rigidity of thought'), to that of Parkinsonism ('motor rigidity'). The distractibility and psychomotor restlessness of the manic patient may well be likened to extrapyramidal hyperkinesia. In this connection we wish to recall that most neurologists have time and again seen anxiety states in patients suffering from encephalitis, even when they were afebrile, which could not be distinguished from such states observed in the anxiety neuroses.

It seems to us that the evidence adduced hitherto is at least suggestive of the important role that psychical factors may play in the genesis of organic disease of the central nervous system.

PERSONAL CASES.

To illustrate our thesis we have selected at random the histories of some of our patients who began their disease with a psychogenic picture, and later showed evidence of definite organic nervous disease which could be well harmonized with the original complaint.

CASE 1.

J. S. At the age of sixteen this patient experienced great difficulties in her attempts at social adjustment. During this period she was continually subjected to various psychical traumas, which greatly influenced her behaviour. The details of these are not essential for the purposes of this paper. In January 1921, at the age of seventeen, following an unusually severe emotional shock she made an unsuccessful attempt at self-destruction. Immediately thereafter she developed movements of the muscles of the face, trunk and all her extremities. These movements, apparently involuntary, did not assume the pattern of any of the well-known dyskinetic phenomena usually associated with organic nervous disease. She was observed for three months at the Lenox Hill Hospital and after a stay in the country her condition improved. In May 1929 she was observed again, this time at Bellevue Hospital. Her movements later became intensified and she was then studied for nine weeks at the Neurological Institute. In March 1922 she entered the Mt. Sinai Hospital, where she was very noisy and excited and disturbed the other patients. She was finally admitted to the neurological wards of the Montefiore Hospital on May 13, 1922. Here the movements were found to be generalized and began to assume the pattern of a violent chorea. As time went on, however, she developed contractures at the ankles and toes and the movements became typically dystonic. There is no question now but that hers is a classical case of dystonia musculorum deformans.

CASE 2.

L. B. In August 1927, while she was swimming in the Hudson River with a friend both were swept away by the undertow. Her playmate was drowned and our patient was barely saved. She then developed generalized choreiform movements; these have persisted and are always aggravated by any emotional strain. She has never had any sore throats, joint pains, or other rheumatic manifestations.

When she was first admitted to the Mt. Sinai Hospital, on November 16, 1927, her entire clinical picture was attributed to hysteria. The psychiatrist in that institution interpreted her movements as a "reactivation of an old conflict between infantile exhibitionistic tendencies and her more mature and realistic ego ideal." As time went on, however, the posture of the feet assumed a definite pattern of dystonia and now there is no question that she is suffering from dystonia musculorum deformans.

CASE 3.

R. C. This is the case of a female of 26 years. At the age of fourteen, while in the country, she was greatly frightened during a thunderstorm. She immediately developed jerky movements in all parts of the body, especially the face, shoulders, hands and lower extremities. During the following three years she was under observation in several hospitals, in all of which her case was diagnosed as hysteria. She was then admitted to the Montefiore Hospital. During the first three years of her stay in this institution her case was regarded as one of disordered motility of psychogenic origin, although some

of the observers were of the opinion that there might be an underlying organic substratum for her movements. The dyskinetic phenomena were at various times associated with attacks of spontaneous laughter and crying. The movements of the lower limbs were so violent that she was unable to walk until plaster casts were applied under general anaesthesia. She then was able to walk fairly well and left the hospital wearing braces. One year later she was readmitted with the same complaints. Observations at this time left no doubt that the case was one of typical dystonia musculorum deformans.

SUMMARY.

1. The evolution of some organic nervous diseases may occur in two phases: (a) The early psychical, and (b) the later organic phase.
2. The transition between these two phases may be imperceptible and only appreciated after prolonged observation by the same observers.
3. We would suggest that during this transition there may occur structural changes in the nervous system resulting from disturbances of function during the first or non-organic stage.
4. Evidence is adduced that not only may structure influence function, but also that function may influence structure.
5. A hypothesis is offered that psychical factors may, through the vegetative nervous system, affect lipid metabolism and so produce structural changes in the central nervous system at the basis of the organic clinical picture.
6. Three cases are cited in which the patients, following psychical trauma, developed disorders of motility that did not conform to the patterns of any known organic nervous disease. Later the patients developed evidences of organic nervous disease, undoubtedly due to structural changes in the nervous system.

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