GLUCOSE TOLERANCE IN PATIENTS WITH LOCALIZED LESIONS OF THE CENTRAL NERVOUS SYSTEM*

J. P. SEGUNDO, M.D., E. BALEA, PH.D., AND R. ARANA, M.D.

Laboratorio de Neurofisiologia Clinica (Institutos de Neurologia y de Ciencias Fisiologicas), Facultad de Medicina, Montevideo, Uruguay

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Control of carbohydrate metabolism is fundamentally a function of the endocrine system. Neurohumoral mechanisms, though dispensable, are responsible for finer adjustments and for the integration of changes of glycemia in different patterns of response (sham rage, adaptation to cold, etc.14,15,18). Problems relating to this nervous regulation have been approached on different occasions by studying abnormalities exhibited by patients with neurological diseases.1,9,12,23,28,29,31,33–37,39 The present paper describes an attempt to find further proof of the participation of the central nervous system in the control of carbohydrate metabolism and, at the same time, obtain knowledge of the structures involved. As tumors appear at any age, in patients generally without other diseases and in practically any region of the central nervous system, as they present a relatively well-known pathological physiology5 and as they permit verification, they offer the opportunity of an analysis somewhat similar to that of a planned physiological experiment. We considered them, and a small number of other equally well-localized and verifiable lesions, as material better adapted to an investigation of the problem than that presented by other pathological entities and, therefore, proceeded to study them accordingly.

The correlation between localization of cerebral abnormality and associated disturbance of carbohydrate metabolism will necessarily have to be interpreted in a general way as neither radiologic nor operative observations are capable of elucidating, in a strict and detailed fashion, the entire extent and specific nature of neural structures that are involved. There exists the possibility, moreover, that these lesions may disturb function in the central nervous system through some mechanism that, as yet, remains obscure. These drawbacks, inherent in the handling of such clinical material, do not, in our opinion, proscribe its use for research analysis; nevertheless, they suggest that caution must be exercised in the interpretation of the available data.

MATERIAL AND METHODS

All patients in this series were under the age of 66 to avoid the influence of senility on the glucose tolerance curve.35 Also, patients who had a family

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history of diabetes were excluded as were those who had evidence of hepatic, endocrine or any other disease that could alter the glucose tolerance curve. The general condition of all individuals studied was adequate, none being terminally or severely debilitated.

As the study was undertaken to assess the influence of regional disturbances of the brain upon carbohydrate metabolism, the patients were divided into 2 groups. The first (Group I) contained only patients who had a disturbance of a lobe or part of the brain caused by the presence of a tumor mass that was later verified. The other (Group II) contained patients in whom a lobe or part of the brain was isolated or resected operatively. Primarily, the patients of Group I, therefore, were studied pre-operatively while those of Group II were evaluated postoperatively. In certain instances, however, postoperative determinations were made in patients of Group I (5 cases marked with asterisks in Table 1) and pre-operative examinations in cases of Group II for purposes of comparison.

Group I was made up of 43 patients, each with a localized lesion in the central nervous system that was both confirmed and localized by radiographic procedures (5 cases), by operation (34 cases) or by autopsy (4 cases). Most of these patients had tumors (34 cases) but 3 had hydatid cysts, 1 a giant and partially thrombosed aneurysm of the temporal fossa, 1 a spontaneous intracerebral hematoma, 2 platybasia with modifications of the cervical vertebrae, and 2 cervical spinal cord traumatic injuries.

Group II consisted of 7 leucotomized schizophrenics, 2 cingulectomized idiots, and a patient who, because of a large oligodendroglioma, was subjected to a right temporal lobectomy; this patient was included in Group II because she was not tested until after operation.

In making a classification of lesions or operative isolation according to localization, we accepted the gross anatomical divisions of the brain in main lobes and added those special regions that gave characteristic clinical syndromes (corpus callosum, third ventricle, pineal body, suprasellar region). Admitting a possible functional significance, we subdivided the major lobes as follows: frontal into precentral and prefrontal (convexity, orbital surface, white matter), temporal into convexity, basal surface, white matter, and deep (in the region of the basal ganglia and region of the third ventricle), parietal and occipital into convexity and white matter. Other lesions involved the cerebellum, the cerebellopontine angle or the cervical spinal cord. When, as frequently occurred, the lesion occupied more than one of the schematic regions described, it was included in each of the corresponding groups.

The same test was applied to all patients included in Groups I and II. The technique used for the glucose tolerance curve was that suggested by Levinson and MacFate. For 3 days prior to testing, the patient was placed on a special diet that included an abundant supply of protein and carbohydrate; no complementary medication was administered but the routine diagnostic measures were performed. In patients subjected to pneumoencephalo-