1971

Year Book OF NEUROLOGY AND NEUROSURGERY

DE JONG
SUGAR

THE YEAR BOOK *of* NEUROLOGY *and* NEUROSURGERY 1971

NEUROLOGY

EDITED BY

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NEUROSURGERY

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YEAR BOOK MEDICAL PUBLISHERS

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There are twenty YEAR BOOKS in various fields of medicine and one in dentistry. Publication of these annual volumes has been continuous since 1900. The YEAR BOOKS make available in detailed abstract form the working essence of the cream of recent international medico-scientific literature. Selection of the material is made by distinguished editors who critically review each year more than 500,000 articles published in the world's foremost journals.

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NEUROLOGY

RUSSELL N. DE JONG, M.D.

INTRODUCTION

A large number of neurologic diseases have, in the past, been called "degenerative" because of lack of specific information about their etiology and mode of production. Some of these were inherited in a known manner, but in most there was no familial pattern. Some came on late in life, and were presumed to be associated in some way with the aging process, but others came on early, even in infancy or childhood, and still others in middle age. The pathology of most of them was known, but the changes could not be explained on the basis of vascular disease, known toxin or infection, or known deficiency or metabolic alteration. With the passage of time, however, and one by one, etiologic information has been gained about some of them—the vitamin deficiencies, the disturbances secondary to hepatic or renal disease or associated with diabetes mellitus, and the endocrinopathies, among others.

During recent years, explanations have been reached for increasing numbers of those diseases which were formerly called degenerative. Wilson's disease, or hepatolenticular degeneration, is now known to be the result of a genetically determined disturbance of copper metabolism, and it can be treated effectively by drugs that decrease the concentration of copper in body tissues. Creutzfeldt-Jakob disease and subacute sclerosing panencephalitis (Dawson's inclusion body encephalitis) have been shown to be caused by a slowly growing or maturing viral agent. Many of the progressive dementing diseases of infancy and early childhood are now known to result from deficiency or absence of specific enzymes, often with secondary accumulation of abnormal biochemical products. It has recently been shown that Parkinson's disease is a metabolic disturbance with a deficiency of dopamine and other catecholamines in the basal ganglions. For some of these conditions treatments are available, such as replacement of the deficient enzyme or other substance or removal of the offending accumulated product. Unfortunately, however, many of them are still untreatable. Current research has contributed a great deal, nevertheless, in aiding our understanding of the underlying mechanisms of these disorders, and gives a ray of hope for some treatment in the future. A ma-

jor question facing neurologists today is whether these advances may help us to ascertain the etiology of still-devastating and high-incidence diseases such as multiple sclerosis and amyotrophic lateral sclerosis.

During the time period reviewed in this YEAR BOOK, L-dopa has become accepted as the treatment of choice in Parkinson's disease and the parkinsonian syndromes in all stages—from the patient with barely perceptible difficulties to the bedfast patient. Its effectiveness in relieving the symptoms of these disorders far surpasses that of previously used agents. With the understanding of the possibility of and mechanism for side effects associated with its use, most of which are dose related, these can be kept to a minimum. Unfortunately, it has not been shown to be helpful in other extrapyramidal disorders such as chorea and dystonia. Amantadine has also proved useful in Parkinson's disease, especially for those patients who cannot tolerate therapeutic doses of L-dopa, and in some cases a combination of the two seems to work.

The association of hydrocephalus with dementia, the syndrome of so-called normal-pressure hydrocephalus, has continued to arouse interest, and it has now become standard neurologic practice to consider this syndrome in the evaluation of all patients with progressive organic dementia. The well-defined clinical picture that has emerged includes memory impairment, apathy, unsteadiness of gait and urinary incontinence. In severe cases the patient becomes mute and helpless and appears totally demented. If the diagnosis is confirmed by pneumoencephalography and RISA scanning, ventriculovenous shunting should be carried out. The results of this procedure have been gratifying in some cases but not in others. No beneficial effect can be anticipated from shunting in those patients in whom the picture is not complete and where there is no evidence of impaired cerebrospinal fluid flow over the cerebral convexities. The procedure has been tried without success in patients with Alzheimer's disease and other degenerative conditions who have dementia and evidences of cerebral atrophy as shown by the finding of both enlarged ventricles and increased cortical air on pneumoencephalography. It is important to bear in mind that surgery should be limited to those patients who presumably will benefit.

The definition and delineation of cerebral death are assuming increasing importance. In most large hospitals where transplant surgery is done there is a committee on cerebral death of which

neurologists and electroencephalographers are members. The criteria are now quite well established, except for some question of how long the electroencephalogram should be isoelectric before the patient is declared dead. With the widespread use of drugs of all kinds, especially by members of the younger generation, questions have been raised about effects on the nervous system, possibly permanent ones. Several articles do give some suggestions that marijuana does have some effect on memory and does cause some minor changes in the electroencephalogram. One might conclude, then, that prolonged use would cause even more changes, some of which might be permanent. The dangers of using the stronger drugs, especially in unusual doses, are well known.

We must always be on the alert to the possibility of the development of hitherto unrecognized side effects or toxic reactions associated with the use of our therapeutic agents. It is an old adage that there is no medication that may not produce some side effects in susceptible patients. We are constantly learning of new complications associated with the use of some of our common medications. Diphenylhydantoin and some of the related anticonvulsants are examples. We have long known of the possibility of the development of sedation, skin rash, gingival hyperplasia and occasionally ataxia and nystagmus in association with the use of the hydantoinates. Some years ago fairly conclusive evidence was brought forth that massive doses may cause destructive changes in the cerebellum, which would explain the ataxic syndrome. There is still, however, controversy about this. Now we learn that the hydantoinates may cause changes in the hemopoietic system, with the development of lymphadenopathy and even malignant lymphoma, or of folate deficiency and associated anemia. They may also cause the development of disseminated lupus erythematosus or a syndrome closely resembling it, and they have recently been shown to cause the inhibition of antidiuretic hormone secretion. We must continue to be on the alert.

The books dealing with neurologic subjects which were published during the past year are, in the main, devoted to specialized subjects rather than to general neurology. One general text is *Brain Diseases* by Prof. Arie Biemond of the University of Amsterdam (New York: Elsevier Publishing Co., 1970), which summarizes the author's extensive clinical experience over many years. In *Studies in Neurology* (New York: Oxford University

Press, 1970), Sir Charles Symonds reprints some of his most important contributions of the past 50 years, and adds autobiographical comments. *Aphasiology and Other Aspects of Language* by Macdonald Critchley (London: Edward Arnold, Ltd., 1970) is an interesting collection of the author's writings on problems of speech. *Neuropathology: Methods and Diagnosis,* edited by C. G. Tedeschi (Boston: Little, Brown and Co., 1970) deals not only with traditional neuropathology but also with neurochemistry and enzyme histochemistry. Two volumes of the multivolume *Handbook of Clinical Neurology,* edited by P. J. Vinken and G. W. Bruyn (Amsterdam: North-Holland Publishing Co.), were issued in 1970. These are Vol. 8: *Diseases of Nerves, Part II,* and Vol. 9: *Multiple Sclerosis and Other Demyelinating Diseases.*

The advances in the treatment of Parkinson's disease are brought up to date by Andre Barbeau and Fletcher McDowell in L-*Dopa and Parkinsonism* (Philadelphia: F. A. Davis Co., 1970) and in a symposium *Pharmacologic and Clinical Experiences with Levodopa,* chaired by William E. O'Malley and published as a supplement to the December, 1970, issue of *Neurology*. Several books were published which deal with various aspects of cerebrovascular disease, among which are *Cerebral Circulation and Stroke* edited by Dr. K. J. Zülch (New York: Springer-Verlag, Inc., 1970), *Atlas of Cerebral Angiography* by T. Nomura (New York: Springer-Verlag, Inc., 1970), *The Physiological Mechanisms of Cerebral Blood Circulation* by A. I. Naumenko and N. N. Benua (Springfield, Ill.: Charles C Thomas, Publisher, 1970), *Recent Advances in the Study of Cerebral Circulation* edited by Juan M. Taveras, Herman Fischgold and Domenico Dilenge (Springfield, Ill.: Charles C Thomas, Publisher, 1970) and *Cerebrovascular Dynamics* by Robert F. Rushmer (Philadelphia: W. B. Saunders Co., 1970). A new periodical *Stroke: A Journal of Cerebral Circulation* was also introduced in 1970 by the American Heart Association; Clark H. Millikan is editor-in-chief and the associate editors are A. B. Baker and Fletcher McDowell. Three books dealing with different aspects of the neurology of childhood are *Pediatric Neurology* by Ingrid Gamstorp (New York: Appleton-Century-Crofts, Inc., 1970), *Neurology of Early Infancy* by Anatole Dekaban (Baltimore: The Williams and Wilkins Co., 1970) and *Neuromuscular Diseases of Infancy and Childhood* by K. F. Swaiman and F. S. Wright (Springfield, Ill.: Charles C

Thomas, Publisher, 1970). For the historically minded there are the second and enlarged edition of *The Founders of Neurology* by Webb Haymaker and Francis Schiller (Springfield, Ill.: Charles C Thomas, Publisher, 1970) and *Weir Mitchell* by R. D. Walter (Springfield, Ill.: Charles C Thomas, Publisher, 1970).

RUSSELL N. DE JONG

NEUROANATOMY AND NEUROPHYSIOLOGY

Origin of the Alpha Rhythm. Olof Lippold[1] (London) observes that it is generally accepted that the alpha rhythm originates mainly in the occipital cortical neurons, which are presumed to beat in unison at a frequency of about 10 Hz when the cortex is "resting." There is no convincing evidence of a "physiologic" function of the alpha rhythm, however; 2-5% of normal persons never exhibit the waves. There are no perceptual or motor concomitants despite the large amplitude of the waves and their wide distribution over the head. The site of origin has been postulated to be in a dipole in the gray matter of each occipital lobe, where phase reversal points are located, but frontal alpha waves, farther from the supposed site of origin and smaller in amplitude, tend to lead the occipital waves by a variable interval, usually about 5 msec.

The extraocular muscles have feedback control, with oscillation at about 10 Hz when the loop gain exceeds unity. Usually this occurs with the eyes closed or when a sharp retinal image is lacking. The standing potential across the eye is the source of current for generating the wave form of alpha rhythm. It is conducted back into the cranium by the extraocular muscles, which act as variable shunts. The electric record of alpha rhythm closely resembles the mechanical record of a limb tremor. Both rhythms vary similarly with age, and in Parkinson's disease with eye muscle involvement, the predominant cortical rhythm, like the tremor, is often at 5 per second. Studies have shown excellent correspondence between the amplitude of the alpha waves and the magnitude of the corneoretinal potential when illumination of the eye is altered.

It is concluded that occipital alpha waves are generated by the standing potential across the eyes. The likely mechanism in-

(1) Nature, London 226:616-618, May 16, 1970.

volves some form of modulation of this potential by oscillation in the extraocular muscles at about 10 Hz when there is no sharply focused image on the retina.

► [The source of the alpha rhythm has never been entirely understood. This article may raise controversy, but the author does appear to report a carefully carried out study. If it can be authenticated, it may bring about a reappraisal of our present concept of electroencephalography, because it suggests that the wave form that is found in human subjects when their eyes are closed does not originate in brain tissue.—Ed.]

Somatosensory Thresholds: Contrasting Effects of Postcentral Gyrus and Posterior Parietal Lobe Excisions. In view of the disagreement on the contributions of different parts of the cortex to somatosensory discrimination, Suzanne Corkin, Brenda Milner and Theodore Rasmussen[2] (McGill Univ.) obtained quantitative measures of such function in patients with well-localized unilateral cortical excisions. The hand was examined for pressure sensitivity, two-point discrimination, point localization and position sense in patients with focal epilepsy.

Most data were obtained from 127 patients studied in a 6-year period. Fifty patients had parietal cortex excisions. Four patients having postcentral gyrus removals and 4 having posterior parietal removals were retested postoperatively. The lesions made produced a more or less localized area of brain damage, subsequent healing with gliosis and with more severe brain destruction, gross cicatrix formation. All the patients had static, long-standing brain lesions. Removals were carried out by subpial suction dissection. Study was also made of 20 normals.

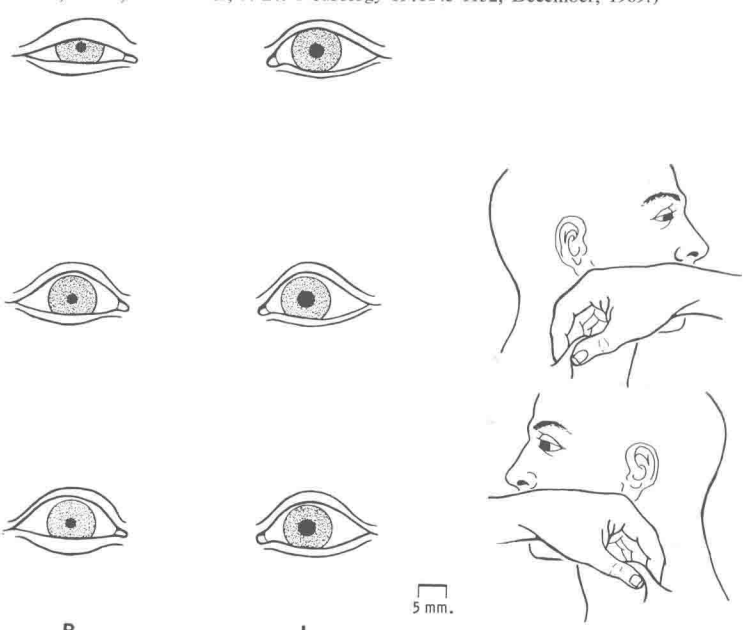
Abnormally high sensory thresholds and impaired position sense were clearly related to excision in the postcentral gyrus, not the posterior parietal lobe, precentral gyrus, frontal-granular cortex or temporal cortex. Within the postcentral region, certain sensory functions of the hand appeared to be focally represented in the hand area. Deficits in tactual object recognition were always accompanied by defects in one or more sensory tests, although not all patients with sensory loss had difficulty with tactual object recognition.

These findings unequivocally reaffirm the role of the postcentral gyrus in discriminative sensibility. The critical area is bilaterally symmetrical and limited to the postcentral gyri. Impaired tactual recognition of common objects reflects the sensory status of the hand; no object recognition deficits were found that were disproportionate to the sensory loss.

(2) Arch. Neurol. 23:41-58, July, 1970.

Ciliospinal Response in Man. Clinical evidence has indicated that the ciliospinal reflex requires an intact brain stem, but other evidence indicates that the brain stem is not required. The relative significance of sympathetic excitation and parasympathetic inhibition in reflex pupillary dilatation is presently unclear, as is the importance of central sympathetic pathways for its existence. Alexander G. Reeves and Jerome B. Posner[3] (Cornell Univ.) studied 20 unanesthetized patients with neurologic disease to resolve these problems and to determine whether the reflex is clinically useful. The patients had lesions of the peripheral sympathetic pathways in the upper mediastinum or cervical region, or both. All had miosis, ptosis and ele-

Fig. 1.—Ciliospinal response after a peripheral lesion of sympathetic pathway in woman, 43, with extensive carcinoma of lung involving right apical and superior mediastinal regions. Miosis, ptosis of upper lid, elevation of lower lid and decreased facial sweating were present on right. There were no other neurologic abnormalities. Pinching of either right or left side of neck produced no pupillary change on right but a brisk 1- to 2-mm. dilatation of left pupil. Upper lids were manually retracted during stimulation for ease of observations. Drawings were made to scale indicated from 35-mm. transparent photographs taken during ciliospinal test. Slides were projected on a screen and traced by hand. (Courtesy of Reeves, A. G., and Posner, J. B.: Neurology 19:1145-1152, December, 1969.)



(3) Neurology 19:1145-1152, December, 1969.