Book Review


The present volume is the result of an international congress organized in March 1985 as part of the annual OHOLO conferences sponsored by the Biology Research Institute of Israel. The large volume contains 710 pages, with the integral text of 73 communications, divided into six parts. The first part is dedicated to anatomical, pathological and biochemical aspects. It begins with an analysis of anatomical organization and cholinergic function (Butcher et al.). It then continues with an excellent synthesis of pathological and biochemical aspects of Alzheimer’s disease (AD) (Wisniewski). The importance of neuronal loss in the nucleus of Meynert in the pathogenesis of AD is questioned by Edwardson et al., who find the same phenomenon in Parkinson’s disease (PD), not connected with dementia. In a discussion of the basic mechanisms leading to AD, Wurtman et al. consider the possibility of cellular ‘autocannibalism’, where the neuron utilizes the choline in the phospholipids of its own membrane. A number of communications are devoted to neurotransmitter and neuropeptide deficits, in particular of serotonin, noradrenaline, muscarinic cholinergic receptors (M1, M2), somatostatin and vasopressin, Ap-pel et al. discuss the idea of a lack or functional deficiency of nerve growth factors caused by toxic, immunologic or other mechanisms directed against them.

The part of this volume dealing with clinical aspects and noninvasive diagnostic approaches is rather heterogeneous. In a strictly clinical approach, Mayeux and Stern, basing their argument on a 4-year longitudinal study, distinguish 4 types of AD: a typical form, a benign form, AD accompanied by extrapyramidal signs, and AD with myoclonia. The two latter types are of rapid evolution. Using a neuropsychological approach, Mildworf et al. find that global deterioration is more marked in AD than in Parkinson’s disease with dementia. In PD there is a correlation between the slowing in the EEG and in motor control and mental deterioration (Korczyn). Stern and Mayeux mention the existence of an early perceptional-motor disturbance in PD, attributed to a dopaminergic deficit. On an epidemiological level, Barclay et al. find familial connections in 35.9% of 259 AD cases, and a high level of other hereditary diseases in the same group. They suggest that genetic factors play a more important role in the etiology of the disease than environmental factors.

The contributions devoted to neuro-imaging clearly demonstrate the superiority of NMR over CT scanning in the distinction between gray and white matter and in the detection of lacunae (Growdon et al.) The periventricular hypodensity frequently observed on the CT scans in AD is of vascular origin, as Leon et al. show by anatomoclinical correlations. Measuring regional cerebral blood flow indicates that it is reduced at an early stage of presenile dementia, aiding in the differential diagnosis from depression (Prohovnik et al.).

In the section on etiological and genetic aspects, one should note in particular the description by Perl et al. of the use of the laser microprobe mass analyzer (LAMMA) in the detection of...
aluminum in cells with neurofibrillar degeneration. Groner et al. review the possibility of a common gene in the etiology of AD and Down’s syndrome.

Blass et al. discuss biological markers. Other authors present the estimation of acetylcholine metabolism in the LCR, of somatostatin-like immunoreactivity, and of biopterin levels (coenzyme of hydroxy-lases). Chapman et al. have found anticholinergic cell antibodies in the serum of AD patients. Adem et al. describe a reduction of cholinergic lymphocytic receptors in the same disease.

The section on experimental models provides excellent information on research on animal systems permitting pharmacological experimentation. Highlights are the review by Gamzu et al. on the techniques for manipulation of memory fixation in animals, London’s report on measuring regional glucose metabolism in the brain, and several works on lesions in the cholinergic system due to the neurotoxin AF64A (ethylcholine aziridinium ion). In the cortex of rats, the implantation of AD tissue fragments leads to the formation of twisted filaments in astrocytes near the implantation site (van den Bosch de Aguilar et al.). These authors also induced necroses in the cortex and hippocampus by intraventricular injection of human brain homogenates, a phenomenon which increases with donor age. These findings seem to support the hypothesis that cytotoxic substances are produced by the brain during aging. Finally, Kopin et al. review parkinsonian syndromes induced in man and animals by MPTP (L-methyl-4-phenyl-2,3,6-tetra-hydropyridine).

In the last section of this volume (development of new drugs, innovation in experimental approach), a considerable number of communications discusses the pharmacological action on the cholinergic system, particularly by acetylcholinesterase inhibitors, such as synthetic carbamates (Weinstock et al.) and sulfoxyl fluorides (Moss et al.). Nerve growth factor has a protective action on experimentally lesioned septal cholinergic neurons (Hefti). Finally, one communication discusses type B monoamine oxidases in human and experimental parkinsonism.

Although this volume has both Alzheimer’s and Parkinson’s diseases in its title, it devotes most of its content to the former disease. Presentation of basic research takes up clearly more space than clinical problems. A large amount of valuable information on recent developments in research is presented and discussed in the perspective of therapeutic biomedical applications. This is a rich source of information for the scientist as well as for clinical investigators eager to enlarge their scientific understanding of very complex diseases. Unfortunately, the ‘camera-ready’ offset print production disturbs the uniformity of the presentation.

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