Report of the Vth International Symposium on Parkinson's Disease—Recent Advances in the Research of Parkinsonism—, Vienna, Austria, September 17—20, 1975*

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275 scientists of 30 countries participated in this symposium. 55 of them held lectures. Prof. Birkmayer was the president of the meeting.

The Ludwig-Boltzmann-Institute for Neurochemistry arranged the Vth International Symposium on Parkinson's Disease—Recent Advances in the Research of Parkinsonism—, in Vienna, September 17—20, 1975. The most famous scientists in this special field of medical research and treatment discussed problems concerning etiology, clinical treatment and medical precaution of Parkinson's disease. It was one of the first diseases of the nervous system for which a biochemical correlation could be proved. Thus we can say that its decoding has to conform with the advances of biochemical and pharmacological research of the nerve cell. The biochemical, pharmacological, neurophysiological and morphological problems of basic research were the most interesting and most thoroughly discussed subjects of this meeting.

Concluding such a congress the question always appears if new knowledges could be achieved. Where do we currently stand in Parkinson research?

The prevailing disturbance in Parkinsonian syndrome is the deficiency of dopamine caused by degeneration of the dopaminergic

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nigrostriatal pathways. Fading of the melanin-containing cells of the substantia nigra can be observed by morphological examination. This dopamine-deficiency which was correlated with the akinetic condition of the patient was proved by O. Hornykiewicz in Vienna in 1960. W. Birkmayer and A. Barbeau started the treatment with L-dopa in 1960/1961. 15 years after these discoveries the basic research work is still occupied especially with the dopaminergic neurons of the striatum.

Peripherally administered L-dopa—especially its combination with a decarboxylase-inhibitor—is transformed presynaptically into dopamine which partly is accumulated and released by impulses. It is released into the synaptic cleft and activates the postsynaptic receptor. A kinetic effect is the result. Dopamine partly is deaminated intra-neuronally by monoamine oxidase (MAO); about 10% of the dopamine released into the synaptic cleft is metabolized by catechol-O-methyltransferase and 90% is reuptaken into the presynaptic neuron. We think to be justified in assuming that the presynaptic neuron is degenerated in Parkinson’s disease. Thus L-dopa can only be effective as long as presynaptic neurons are prevalent. The destruction of dopaminergic neurons cannot be prevented by applying L-dopa. At present experiments are undertaken to make the postsynaptic neurons more amenable to therapy by developing substances which stimulate the postsynaptic receptor. Actually a number of so-called dopaminergic agonists are existing (apomorphine, bromocriptine, lergotril etc.), which have been successfully tested in model experiments. For different reasons (severe side-effects, short-term action, tolerance development) their clinical administration is restricted to single and acute cases. Dopaminergic agonists inhibit the synaptic tyrosine hydroxylase activity and therefore the endogenous L-dopa synthesis. The small effectiveness of treatment of Parkinsonian patients with dopaminergic agonists might also be correlated with the possible degeneration of postsynaptic structures.

Inhibition of intraneuronal MAO-activity is another more efficient possibility to release dopamine for neural transmission. For approximately one year more than 200 patients with Parkinson’s disease were treated at our institute with our MAO-inhibitor combined with Madopar®. The combination of MAO-inhibitors with L-dopa was already described by W. Birkmayer and O. Hornykiewicz in 1962, but the clinical results were not satisfactory because of the various side-effects. The most important result of the research in the field of MAO action is the classification of its 5 isoenzymes into two groups (MAO-A and MAO-B) according to their different substrate and inhibition properties. MAO-A rather deaminates