BOOK REVIEW


These volumes' stated aim is to update clinical neurologists on progress made in basic science in the field of Parkinson's disease. The volume on symptomatic versus preventative therapy has more immediate appeal to those in clinical practice. Many neurologists would immediately be drawn to the chapter on monoamine oxidase inhibitors in an attempt to discover whether they should still be prescribing deprenyl (Selegeline) to their patients at the onset of symptoms. With the manufacturers now extending the possible indications for deprenyl into other neurodegenerative disorders such as Alzheimer's disease, a rigorous analysis of published data on the safety and efficacy of this compound is long overdue. Unfortunately you will not receive clear direction in your clinical practice here, though Dr Olanow does provide a concise and readable account of the controversy surrounding use of the drug. I suppose most readers will come away with their own beliefs more clearly entrenched; I for instance, find the evidence for a neuroprotective effect unconvincing and do not therefore currently prescribe a drug with no clear indication and a potential to produce adverse effects.

The remaining chapters include brief reviews of the aetiology and treatment of Parkinson's disease. From a practical point of view, one of the most important controversies at the moment is the role of direct-acting dopaminergic agonists in the early management of the disease. Calne's chapter on combination therapy of dopamine precursors and dopaminergic receptor agonists was enlightening, especially when read with the consensus statement at the end of the volume. There seems to be increasing evidence of a prognostic advantage of starting pergolide or bromocriptine early, especially in younger patients.

The volume on dopamine D1 receptors contains relatively little of direct relevance to a clinician, though the general introduction provides an excellent brief review of the brain's dopaminergic circuitry. We have come a considerable way since 6-hydroxydopamine lesions of the rat nigrostriatal tract, and now have at least five dopamine receptor subtypes and considerable inter-species variation to contend with. The "classical" D1 receptor agonist SKF 38393, the basis of much rodent research, has turned out to have relatively little D1 activity in primates. D1 and D2 receptors are likely to be present on the same striatal neurone, but most of the motor response to dopaminergic agonists seems to occur through the D1 receptor, although it is possible that simultaneous D2 receptor stimulation is required for the full response repertoire. Other neurotransmitters interact at various sites in the CNS, including the cholinergic, excitatory amino acid and opioid peptide systems. As a result it seems likely that only studies on humans or human-derived neurotransmitter receptor systems are likely to be able to enable prediction of the clinical efficacy of a new drug treatment for Parkinson's disease. As regards aetiology and prevention of the disease, we are still entirely in the dark.

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