tive sleep apnoea and they had also had sleep recordings to confirm the diagnosis. Snoring can be considered as a subclinical stage of obstructive sleep apnoea but not all snorers develop it. It is possible that the muscles in the pharynx are affected in different ways during different stages of the disease.

In the specimens from all patients with obstructive sleep apnoea we found prominent neurogenic changes with signs of both denervation and reinnervation. In the REM-sleep stage the altered polyamine binding site in the NMDA-receptor-gated channel.** It is therefore concluded that: 1) there are other NMDA antagonists clinically available besides ifenprodil and 2) NMDA antagonists have been used successfully for many years in the treatment of PD.

The disappointing results with ifenprodil do not therefore argue against the newly proposed 'functional-mismatch' theory of PD, nor may it be related to: 1) the pharmacokinetic profile of the drug; 2) a rather weak influence of polyamine binding site antagonists compared with the channel blockers on NMDA receptor function or 3) a pathologically altered polyamine binding site in PD. These points have to be examined in future studies.

Correspondence to: Dr Kornhuber.


N-Methyl-D-Aspartate (NMDA) antagonists in Parkinson's disease

We would like to comment on the letter by Montracut et al. in which negative results in Parkinson's disease (PD) with the NMDA antagonist ifenprodil were reported. Ifenprodil acts at the polyamine binding site of the NMDA receptor. The authors state "this drug is, as far as we know, the sole NMDA antagonist currently available on the market", and "our study is the first to investigate the clinical effects of an NMDA antagonist in the treatment of PD."

The adamanantamines amantadine and memantine have been used since 1969 in the treatment of PD.** The weak dopamine agonism in experimental studies seems insufficient to account for their clinical effects.** It has been reported recently that amantadine and memantine act at the PCP binding site of the NMDA receptor coupled ion-channel.** It is therefore concluded that: 1) there are other NMDA antagonists clinically available besides ifenprodil and 2) NMDA antagonists have been used successfully for many years in the treatment of PD.

The disappointing results with ifenprodil do not therefore argue against the newly proposed 'functional-mismatch' theory of PD, nor may it be related to: 1) the pharmacokinetic profile of the drug; 2) a rather weak influence of polyamine binding site antagonists compared with the channel blockers on NMDA receptor function or 3) a pathologically altered polyamine binding site in PD. These points have to be examined in future studies.

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N-methyl-D-aspartate (NMDA) antagonists in Parkinson's disease.

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