tive sleep apnoea and they had also had sleep recordings to confirm the diagnosis. Snoring can be considered as a subocclusive 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 such cases we found analysis of the fibre type spectra to study fibre type adaption less relevant. Subtypes of fibre type II (a, b and c) were identified but showed a great variation and a further analysis was not considered necessary in the limited number of subjects.

Our morphometry did not show "a great variability of muscle fibre size" or atrophy as interpreted by Iannaccone *et al.* The fibre size distribution showed similar abnormalities in all patients, mostly with a two peak dispersion which is typical for a neurogenic lesion.

The muscle specimens were obtained during surgery from the same part of the cranial portion of the palatopharyngeal muscle in all patients and controls. The muscle was exposed after the tonsil had been removed. The same surgeon (HL) collected or supervised the collection of all specimens.

We consider our finding of a neurogenic lesion in the palatopharyngeal muscle in patients with obstructive sleep apnoea to be correct. The same type of lesions have been described by Woodson *et al*[5] in the uvular muscle of these patients. Recently we have published a study showing affected sensory pharyngeal nerves in obstructive sleep apnoea patients.[6]

We believe the different findings in the study by Iannaccone *et al*[1] are due to a different biopsy site being examined and that the patients were not suffering from obstructive sleep apnoea. Whether the type of abnormalities described in the MPC muscle by Iannaccone *et al* are present also in the palatopharyngeal or uvular muscle of snorers remains to be studied.

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

We would like to comment on the letter by Montastruc *et al*[1] 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 adamantanamines amantadine and memantine have been used since 1969 in the treatment of PD.[2-4] The weak dopamine agonism in experimental studies seems insufficient to account for their clinical effects.[4,5] It has been shown recently that amantadine and memantine act at the PCP binding site of the NMDA receptor-coupled ion-channel.[4-6] 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 "glutamate hypothesis" of PD,[7,8] but may be related to: 1) the pharmacokinetic profile of the drug[1]; 2) a rather weak influence of polyamine binding site antagonists compared with ion-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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Montastruc et al reply:

We acknowledge Drs Kornhuber and Riederer's comments concerning our trial on ifenprodil in Parkinson's disease (PD).[1] We are well aware that the adamantanamines amantadine and memantine have anti-NMDA properties but amantadine is also known to release dopamine from striatal neurons, to inhibit the reuptake of dopamine and to have anticholinergic effects.[2] Despite some experimental data, the respective role of these different pharmacological mechanisms remains unknown to explain the clinical anti-Parkinsonian properties of these drugs. Amantadine has only a weak and transient clinical anti-Parkinsonian efficacy which may be compatible with a weak dopamine effect. There are mainly speculative links between the anti-NMDA and the anti-Parkinsonian effects of amantadine. In our opinion, it is therefore still premature to support Kornhuber and Riederer, because of the amantadine and memantine data only, that "NMDA antagonists are used successfully for many years in the treatment of PD". This is why we were interested in investigating the clinical effects of another drug, such as, ifenprodil, which does not have dopamine effects.

It is perfectly clear, as we had already stated in our first letter,[1] that "our work does not exclude a definite role for NMDA antagonists in PD" because of the negative ifenprodil data. We had also already written that other NMDA antagonists with better pharmacodynamic or pharmacokinetic profile may be effective.[1] Since our results have been published, we know that there is another NMDA antagonist which is available in clinical practice. A recent open study has suggested that dextromethorphan, an antitussive drug that is also a non competitive antagonist of the NMDA receptor, might have some efficacy in PD.[3]

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Risk of stroke in TIAs with a cerebral infarct on CT

I have read the article by Koudstaal *et al*[1] in which the authors find a relevant ischaemic lesion on CT in 13% of TIAs, 35% of RINDs and 49% of minor stroke. In their wide bibliographic review they only mention one comparative study on infarction characteristics between patients with transient and persisting signs.[2] We reported four years ago similar results in 219 patients with reversible ischaemic attacks demonstrating that the frequency of brain infarction was related to the duration of the neurological deficit.[3] Ischaemic lesions on CT closely correlated with abnormalities on supra-aortic trunk angiography or Doppler ultrasonography. A higher percentage of recurrence was found in those patients with infarctions, but the difference was not significant.

Koudstaal *et al* mentioned the possibility of an increased risk of major stroke in TIAs with cerebral infarct on CT.[1] Using our pre-

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