Sailer and colleagues (2003) evaluated short- and long-latency afferent inhibition in patients with Parkinson’s disease. They reported that short-latency afferent inhibition (SAI) is normal in Parkinson’s disease off dopaminergic medication, but that it is reduced in the more affected side in Parkinson’s disease on medication. Figure 2 of the paper mentioned above shows a tendency to more pronounced SAI in the more affected side of Parkinson’s disease patients than in control subjects and that this tendency is reversed by L-dopa administration. Therefore, a different possible explanation of their findings is that SAI is pathologically increased in Parkinson’s disease patients and that L-dopa administration restores it.

To evaluate whether there is pathologically increased SAI in Parkinson’s disease, we studied three patients with a pure hemiparkinsonian syndrome [mean age 67.3 ± 9.1 (SD) years; mean Unified Parkinson’s Disease Rating Scale III motor score of the affected side 8.7 ± 4.6]. Hemiparkinsonian patients are an extremely informative group in which to investigate SAI, since each subject can serve as his or her own control. We compared the two sides of the patients and compared the patients with 12 age-matched controls (mean age 73.1 ± 5.4 years). All the patients were newly diagnosed and were not taking any form of anti-parkinsonian medication.

Short-latency inhibition was studied using the technique that we have recently described (Tokimura et al., 2000). Conditioning stimuli were single pulses of electrical stimulation applied to the median nerve at the wrist. The intensity of the conditioning stimulus was set at just over motor threshold for evoking a visible twitch of the thenar muscles. The intensity of the test cortical magnetic shock was adjusted to evoke a muscle response in the relaxed first dorsal interosseus muscle with an amplitude of ~1 mV peak-to-peak.

The conditioning stimulus to the peripheral nerve preceded the magnetic test stimulus. Interstimulus intervals (ISIs) were determined relative to the latency of the N20 component of the somatosensory evoked potential evoked by stimulation of the median nerve. ISIs from the latency of the N20 plus 2 ms to the latency of the N20 plus 8 ms were investigated in steps of 1 ms. Five stimuli were delivered at each ISI. The subject was given audiovisual feedback at high gain to assist in maintaining complete relaxation. The amplitude of the conditioned motor evoked potential (MEP) was expressed as the percentage of the amplitude of the test MEP. The percentage inhibition of the conditioned responses at the seven different ISIs was averaged to obtain a grand mean.

Results are summarized in Fig. 1. The amount of SAI was significantly increased in the affected side in hemiparkinsonian patients (responses reduced to 22.1 ± 10.6% of test size) compared with normal controls (responses reduced to 45.3 ± 16.2% of test size; \( P < 0.05 \), unpaired t test), with a highly significant difference between the pathological and the unaffected side of the hemiparkinsonian patients (responses reduced to 54.1 ± 12.9% of test size; \( P < 0.05 \), paired t test). There was no significant difference between the SAI of the unaffected side of the patients and that of the controls (\( P > 0.05 \)).

Therefore, these findings favour the hypothesis of enhanced SAI in Parkinson’s disease. SAI is reduced or abolished by intravenous injection of the muscarinic antagonist scopolamine (Di Lazzaro et al., 2000), and evaluation of SAI gives the opportunity to test non-invasively the excitability of some cholinergic circuits in the human cerebral motor cortex. The enhancement of SAI in the most affected side of our hemiparkinsonian patients suggests an increase in cholinergic muscarinic activity in the contralateral cerebral cortex.

Altered muscarinic cortical activity in Parkinson’s disease is also supported by several post-mortem studies in patients with Parkinson’s disease that have shown an increase in the total number of muscarinic cholinergic receptors in the
Evaluation of SAI in Parkinson’s disease patients may provide an *in vivo* demonstration of functional changes in central activity in Parkinson’s disease patients.

**References**


