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Source generators of the early somatosensory evoked potentials to tibial nerve stimulation: an intracerebral and scalp recording study

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Abstract

Objective: To investigate the location of the cerebral generators of the early scalp somatosensory evoked potentials (SEPs) after tibial nerve stimulation.

Methods: Tibial nerve SEPs were recorded in 15 patients, suffering from Parkinson's disease, who underwent implantation of intracerebral (IC) electrodes in the subthalamic nucleus, in the globus pallidum or in the thalamic ventralis intermediate nucleus. SEPs were recorded both from the scalp surface and from the IC leads.

Results: The lemniscal P30 response was recorded by all the electrodes. The IC waveforms included a negative N40IC response, followed by a positive (P50IC) and a negative (N60IC) potential. The N40IC, the P50IC and the N60IC potentials did not differ in latency from the P40, the N50 and the P60 responses recorded by the Cz electrode. In 6 patients, in which SEPs were recorded also during the voluntary movement of the stimulated foot (active gating), an amplitude reduction of the SEP components following the P30 potential was observed during movement at the vertex and in the IC traces. Instead, in the contralateral temporal traces the SEP components (N40temp and P50temp) were not modified by active gating, and in the ipsilateral parietal traces only the positive potentials at about 60 ms of latency was decreased.

Conclusions: Two differently oriented generators are active in the contralateral hemisphere at both 40 and 50 ms of latency after tibial nerve stimulation. One source is oriented perpendicularly to the mesial hemispheric surface and generates the potentials recorded by the contralateral temporal and the ipsilateral parietal leads; the other dipolar source is radial to the hemispheric convexity, and generates the potentials at the vertex and those recorded by the IC electrodes.

Keywords: Somatosensory evoked potentials; Source; Tibial nerve; Human brain

1. Introduction

Controversies still exist about the number and the cerebral location of the source generators of the somatosensory evoked potentials (SEPs) after tibial nerve stimulation. In particular, the topography of the negative potential in the fronto-temporal region contralateral to the stimulation and the scalp distribution of the P40 response spreading to the ipsilateral hemisphere have suggested that both these SEP components might be generated by the same source, oriented perpendicularly to the mesial hemispheric surface (Tsumoto et al., 1972; Cruse et al., 1982; Kakigi and Jones, 1986; Kakigi and Shibasaki, 1992). On the other hand, other studies have hypothesized two different cerebral generators to explain the topography of the early tibial nerve SEPs (Beric and Prevec, 1981; Vas et al., 1981; Desmedt and Bourguet, 1985; Pelosi et al., 1988; Yamada et al., 1996). Moreover, the differing effect of the voluntary movement of the stimulated foot on the P40 potential, which is significantly reduced, and on the fronto-temporal negative response, which remains almost unaffected, suggests that these SEP components are generated by different neuronal clusters (Tinazzi et al., 1997a). More recently, we could separate two subcomponents of the P40 potential, by using the phenomenon of gating. In our studies, the amplitude of the parietal subcomponent of the P40 response was scarcely reduced by both the active movement of the stimulated foot and the isometric contraction of the calf muscles

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ipsilateral to the stimulation, which, instead, entailed a strong reduction of the P40 amplitude measured at the Cz vertex (Valeriani et al., 1998, 2000). This result suggests that both the fronto-temporal negative potential and the parietal P40 subcomponent (P40par) origin from the same source perpendicular to the mesial hemispheric surface, while the vertex P40 subcomponent (P40ver) is generated by a radial dipolar source. This hypothesis was reinforced by studies on dipolar source modelling of tibial nerve SEPs, which showed the activity of two differently oriented dipoles in the 40 ms latency range (Valeriani et al., 1997a, 1999; Baumgärtner et al., 1998). These data, however, cannot represent a definitive demonstration, since any dipolar model built by source modelling strategies does not exclude other solutions. In order to strongly confirm the hypothesis of two sources contributing to the early scalp SEPs, one of which oriented tangentially and the other one radially, we should demonstrate the negative counterpart of the radial dipole that generates the P40ver potential. This could be obtained by means of recording electrodes located deeply under the cerebral cortex. Therefore, we collected tibial nerve SEPs from intracerebral (IC) electrodes placed in the subthalamic nucleus (STN), in the globus pallidum (GP) or in the thalamic ventralis intermediate nucleus (Vim) in 15 patients suffering from Parkinson's disease (PD). STN, GP and Vim stimulations represent recently introduced techniques for the treatment of PD motor symptoms, which are resistant to pharmacological treatment (Benabid et al., 1991, 1996; Limousin-Dowsey et al., 1999; Koller et al., 1999). In these techniques, a stimulating electrode is inserted by a stereotaxic procedure in the STN, in the GP or in the Vim, and high-frequency stimuli are adjusted at appropriate frequency-intensity levels, in order to reduce the PD motor symptoms without major side effects. During the days immediately following the implant, the stimulator is switched off but the IC lead is still accessible, so that the 4 contacts can be connected to the neurophysiological equipment and used as IC recording sites.

2. Materials and methods

2.1. Patients

We recorded SEPs to tibial nerve stimulation in 15 subjects (11 men, 4 women, 53.2 ± 9.8 mean age) suffering from PD, in which the motor symptoms (tremor, dystonia, rigidity) were not controlled by the pharmacological treatment. All patients gave their informed consent to participate in the study and approval was obtained by the local ethical committee. IC electrodes were placed in the right and the left STN in 5 and 3 patients, respectively; right and left GP were implanted in two patients and in one patient, respectively; in one patient GP was implanted bilaterally; lastly, right Vim was implanted in two patients and IC electrodes were placed in the left Vim of the remaining patients. In all patients the therapeutic stimulation began 6 days after the implant.

2.2. Surgical procedure

A multipolar stimulating electrode (DBS 3387, Medtronic) was inserted in the selected target (STN, GP or Vim) via a stereotaxic technique (Leksell frame, intraoperative ventriculography, Guiot's scheme), without general anaesthesia. Different types of bipolar or monopolar montages were attempted and different frequency/intensity combinations were used as well, in order to achieve the best therapeutic effect. The clinical outcome was excellent in all patients. In particular, when the stimulator was switched on the motor impairment, evaluated by the Unified Parkinson's Disease Rating Scale, was lesser (mean value 22.44) than the one obtained by the strongest pharmacological treatment before the implantation (mean value 27.12). Moreover, the implanted patients did not have any dyskinesia due to L-3,4-dihydroxyphenylalanine (L-DOPA). Magnetic resonance imaging (MRI) confirmed the appropriate electrode positioning (Fig. 1). A chronic stimulating system was then implanted (Itrel II; Medtronic, Neurological Division, MN, USA), delivering bipolar stimulation with 2 V, 210 μs P.W. and 130–185 Hz in frequency for the Vim, with 1.5–2.5 V, 60–90 μs P.W. and 180–185 Hz in frequency for the STN, with 2–3 V, 210 μs P.W. and 185 Hz in frequency for the GP.

2.3. SEP recording technique

SEPs were recorded in awake patients no sooner than 5 days after surgery. For SEP recording, patients lay on a couch in a warm and semidarkened room. The tibial nerve contralateral to the implanted IC electrodes was stimulated; stimuli (0.2 ms duration) were delivered by skin electrodes at the ankle and had an intensity slightly above the motor threshold. The stimulation rate was 1.5 Hz. SEPs were recorded at rest in all our patients, while in 6 patients SEPs were collected also during a rapid flexo-extension movement of the stimulated foot (active gating). In order to enable the patients to perform the foot movement, some of them were given soluble L-DOPA (125 mg) 20 min before the task. The absence of any modification in amplitude of the lemniscal P30 response in the gating conditions (see Section 3) ensured that the stimulating electrode contact was not affected by the foot movement. Disk recording electrodes (impedance below 5 kΩ) were placed at 4 locations of the 10–20 system: anterior temporal contralateral to the stimulation (T3 or T4), parietal ispsilateral to the stimulation (P3 or P4), Cz and Fz. In 4 patients the contralateral temporal electrode and the ipsilateral parietal lead were not used; in two patients the contralateral temporal electrode was not used; lastly, only one electrode at the Cz vertex recorded surface SEPs in another patient. In all patients, SEPs were recorded also from the 4 contacts of the