

SPINAL INHIBITION OF THE NOCICEPTIVE INPUT BY HIGHER FREQUENCY NON-PAINFUL STIMULI

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Introduzione

•There are some evidences that the non-nociceptive Abeta fiber input may inhibit the electro-cortical activity related to the A-delta fiber input processing.

•We have previously shown that phasic non-nociceptive electric inputs may reduce the amplitude of the laser evoked potentials (LEPs) and that this inhibition takes palce at brain level.

 However, phasic non-nocieptive stimulation represents an experimental model different from cutaneous stimulation commonly used to reduce pain in daily life.

AIM of the present study was to assess the pain reduction due to non-painful cutanous stimulation. In particular, we investigated the scalp LEP modifications during either homotopic or heterotopic tonic activation of non-nociceptive somatosensory afferents.

Metodi

Patients

• We recruited ten healthy volunteers (6 men and 4 women, mean age: 25.83±3.06 years). LEP recording technique

- Electrical stimuli (duration: 0.2 ms, rate: 2 Hz) were delivered on the right radial nerve at the wrist. The stimulus intensity was fixed at two times the sensory threshold, which was referred as non-painful by all our subjects.
- Cutaneous heat stimuli were delivered by a CO₂ laser (10.6 µm wave length, 2 mm beam diameter, 10 ms pulse duration - ELEN, Florence, Italy) on skin. The radial and ulnar territories of both hand dorsum were stimulated.
- Laser stimuli intensity: 18 mJ/mm², which was reported as a painful pinprick by all our subjects.
- Thirty trials, with an interstimulus interval of 10 seconds, were recorded for each stimulation site and for each condition.
- Multi-electrode recording with 31 scalp electrodes placed following positions of the 10-20 International System. Bandpass 0.3 - 70 Hz. Analysis time: 1000 ms with a bin width of 2 ms (500 Hz sampling rate)

Experimental procedure

LEPs were recorded after stimulation of the radial and ulnar territories of both the right and left hand dorsum in 2 conditions: 1) no conditioning stimulation (baseline condition), and 2) electrical non-painful stimulation of the radial nerve at the right wrist (gating condition). All Recordings were separated by a 5 minute time interval

LEP analysis

- The peak latencies and the peak-to-peak amplitudes of the LEP components were measured.
- To calculate the amplitude of the N2/P2 complex we considered the peak-to-peak amplitude at the Cz recording electrode
- For the analysis of LEP amplitude modification, the LEP amplitudes in the gating condition of each stimulation site were expressed as percentages of the amplitudes of the corresponding LEP components recorded in the baseline condition, which were assumed as 100%.
- T-test was used to compare the LEP latencies in 2 condition.
- One-way ANOVA was used to compare the N2/P2 amplitudes among all subject in 2 condition, by considering the stimulation site as sources of variability. P<0.05 was considered as significant.

Risultati



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- 1)As compared to the baseline, in the gating condition the N2/P2 amplitude was reduced in amplitude after stimulation of the radial territory of both hands (p<0.001 and p<0.001 for right and left hand, respectively) and of the right ulnar region (p=0.008), while no inhibition was found to left ulnar region stimulation (p=0.06).
- 2)The largest N2/P2 inhibition was obtained after stimulation of the right radial territory. A lower inhibition (around 35%) was obtained after stimulation of the right ulnar and left radial territories.
- 3) A significant increase of the N2 latency was observed to stimulation of the right radial territory during the gating condition, as compared to the baseline (P=0.02).

Conclusioni

- High-frequency non-painful stimulation of the right radial nerve inhibits the nociceptive input coming from both the ipsilateral and contralateral homotopic regions, and from a close ipsilateral heterotopic territory.
- On the contrary, it does not have any effect on the nociceptive input due to stimulation of a contralateral heterotopic area.
- These results suggest that spinal segmental inhibitory mechanisms are mainly involved.

