

Laser evoked potentials

In contrast to the function of the [visual pathway](#) or [auditory pathways](#) which are electrophysiologically accessible by visual or auditory evoked potentials, the somatosensory pathway cannot be investigated as a whole by conventional [somatosensory evoked potentials](#) (SEP), because these only reflect function of large fibers, dorsal columns, [medial lemniscus](#) and their thalamo-cortical projections mediating sensations like touch and vibration. The other half of the somatosensory system, signaling temperature and pain perception, uses a different set of afferents and different central pathways, the function of which is accessible by laser-evoked potentials (LEPs). LEP can document lesions of the spinothalamic tract and (lateral) brainstem and of thalamo-cortical projections conveying thermo-nociceptive signals. In the peripheral nerve, LEP can help distinguish between large and small fiber neuropathies. The rapid heating of the skin by infrared laser pulses can easily be applied to non-glabrous skin in any dermatome. In recent years, many clinical studies have demonstrated that LEP can supply evidence for establishing clinical diagnoses when deficits of the nociceptive system are present ¹⁾.

Morgalla et al., prospectively enrolled 12 adult patients with unilateral localized [neuropathic pain](#) in the lower [limbs](#) or [inguinal region](#) and followed them up for six months [Laser evoked potentials](#) (LEP) were assessed at baseline, after one month of [Dorsal Root Ganglion Stimulation](#) (DRGS), and after six months of DRGS. Clinical assessment included the [Numerical Rating Scale](#) (NRS), [Brief Pain Inventory](#) (BPI), [SF-36](#), and [Beck Depression Inventory](#) (BDI). For each patient, LEP amplitudes and latencies of the N2 and P2 components on the deafferented side were measured and compared to those of the healthy side and correlated with pain intensity, as measured with the NRS.

At the one- and six-month follow-ups, N2-P2 amplitudes were significantly greater and NRS scores were significantly lower compared with baseline (all p's < 0.01). There was a negative correlation between LEP amplitudes and NRS scores ($r_s = -0.31$, $p < 0.10$).

DRGS is able to restore LEPs to normal values in patients with localized neuropathic pain, and LEP alterations are correlated with clinical response in terms of pain intensity ²⁾.

[Laser-evoked potentials](#) (LEP) were assessed after peripheral [nerve block](#) of the [lateral femoral cutaneous nerve](#) (LFCN) in healthy volunteers from partially anesthetized skin areas to differentially stimulate mechano-insensitive nociceptors.

An [ultrasound guided nerve block](#) of the LFCN was performed in 12 healthy male subjects with Ropivacain 1%. After 30 min, the nerve block induced significantly larger anesthetic areas to mechanical stimuli than to electrical stimuli revealing an area of differential sensitivity. LEPs, reaction times and pain ratings were recorded in response to the laser stimuli of (1) completely anesthetic skin, (2) mechano-insensitive, but electrically excitable skin ('differential sensitivity'), (3) normal skin.

LEP latencies in the area of differential sensitivity were increased compared to unaffected skin (228 ± 8.5 ms, vs. 181 ± 3.6 ms, $p < 0.01$) and LEP amplitudes were reduced (14.8 ± 1.2 μ V vs. 24.6 ± 1.7 μ V, $p < 0.01$). Correspondingly, psychophysically assessed response latencies in the differentially anesthetic skin were increased (649 ms vs. 427 ms, $p < 0.01$) and pain ratings reduced (1.5/10 vs. 5/10 NRS, $p < 0.01$).

The increase in LEP latency suggests that mechano-insensitive heat-sensitive A δ nociceptors (MIA, type II) have a slower conduction velocity or higher utilization time than mechano-sensitive type II A δ nociceptors. Moreover, widely branched, slowly conducting and mechano-insensitive branches of A δ nociceptors can explain our finding. LEPs in the differentially anesthetized skin provide specific information about a mechanically insensitive but heat-sensitive subpopulation of A δ nociceptors. These findings support the concept that A-fibre nociceptors exhibit a similar degree of modality specificity as C-fibre nociceptors³⁾.

References

1)

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