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Effects of sacral neuromodulation on afferent signal processing in patients with neurogenic lower urinary tract dysfunction – preliminary results

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Background: Sacral neuromodulation (SNM) is a well-established therapy for non-neurogenic lower urinary tract dysfunction (LUTD) with increasing evidence in patients with neurogenic LUTD (NLUTD). While SNM seems to involve modulation of spinal cord reflexes and supraspinal networks, the exact mechanism of action remains unclear. Neuromodulation has previously been shown to affect afferent signal processing, specifically long-latency tibial sensory evoked potentials (SEPs) (higher amplitudes after neuromodulation) and increased current perception thresholds (CPTs) after SNM. However, the relationship between clinical outcomes and afferent changes needs further investigation. The aim here is to investigate the effects of SNM on afferent signal processing and in relation to clinical success in patients with NLUTD.

Methods: Afferent nerve function was investigated in 40 patients with refractory NLUTD before and after SNM testing. Tibial, pudendal and lower urinary tract electrical sensory assessments (LUTESA) including CPTs and SEPs were performed. 3Hz electrical stimulation was used for tibial and pudendal assessments, and 0.5Hz for LUTESA stimulating at bladder dome, trigone, proximal and distal urethra, one after the other. Vertex (versus Fz) recordings were filtered (notch, 0.5 – 70Hz bandpass), segmented and averaged per visit, stimulation location (task) and subject. Mean SEP trajectories and the presence of components (tibial and pudendal SEPs: P40, N50, P65, N85; LUTSEPs: P1, N1, P2) were analysed over both visits. In patients with all components present, peak markers were individually set for latency and peak-to-peak amplitude analysis.

Results: CPTs did not change after SNM testing on group level (n=40) in all tasks. For tibial and pudendal SEPs, the P40, N50, P65, N85 components were visible on group level, and marker analyses (n = 18) revealed no changes in latencies and amplitudes between visits. Consequently, tibial and pudendal SEPs group mean trajectories were compared using fixed time points. This pre-post SNM analysis showed amplitude changes rather in late (> 85 ms) than in early components. Regarding LUTESA outcomes, P1, N1 and P2 SEP components were present on group average for all stimulation locations. Group averages of LUTSEPs showed some trends with consistent changes over time (p-value around 0.1), in particular increased amplitudes after SNM in the transition between N1 and P2, but no significant difference in P1, N1 & P2 components pre-post SNM testing. Considering clinical success, analyses revealed differential effects between SNM responders and nonresponders in CPTs for tibial stimulation only (decreased in responders, no change in nonresponders). LUTESA indicated significant group effects (higher CPTs in non-responders) for both visits. Regarding SEP components, differential effects were present after SNM for tibial nerve stimulation (smaller amplitudes after SNM in components ~250 ms). In LUTSEPs differential effects were mainly found in early components.

Conclusions: This is one of the first SNM studies combining tibial, pudendal and lower urinary tract SEPs with CPT assessments in patients with NLUTD. Our results revealed no SNM effects regarding the predefined SEP components, however over the whole SEP trajectories some changes over time were observed pointing towards SNM effects. Considering the heterogenous urological and neurological patient population, these are promising results and further investigations are warranted