**RESULTS:** Spinalized rats developed urinary bladder hyperreflexia after 3 weeks of spinalization. This was associated with a significant increase in the neuropeptide content of the DRG of L6. S1 electrostimulation lead to the decrease of the neuropeptide content of L6 significantly. L6 DRG content of SP was 0.050±0.003, 0.063±0.003 and 0.041±0.005 pmole for controls, spinalized group and spinalized stimulated group respectively, while that of NKA was 0.036±0.003, 0.051±0.005 and 0.040±0.005 pmole and that of CGRP was 0.371±0.131, 0.426±0.095 and 0.195±0.019 pmole for the same groups respectively. This was associated with a reduction in the amplitude of the longer latency evoked responses (probably mediated by the C-afferent fibers) without marked affection of the latency. In contrast, spinalization and S1 neurostimulation did not affect the neuropeptide content of L5 DRG except for the CGRP, which increased with spinalization and decreased with neurostimulation.

**CONCLUSION:** Sacral root neurostimulation abolished the hyperreflexia with a simultaneous drop of the elevated neuropeptide content of L6 root DRG in spinalized animals. This may indicate that blocking of the hyperactive C-afferent fibers is one of the mechanisms of action of sacral root neuromodulation.

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## CONTRACTION OR SUPPRESSION OF THE BLADDER BY MAGNETIC STIMULATION OF THE SACRAL ROOTS? RESOLVING THE PARADOX.

Aims of Study Electrical stimulation of the sacral anterior roots through implanted electrodes gives consistently good bladder pressure rises and voiding in patients with a spinal cord injury (SCI) [1] Bladder emptying [2] and detrusor contractions [3,4] are said also to be produced by non-invasive functional magnetic stimulation (FMS) of the sacral nerves in spinal injury but this type of stimulation appears to give equivocal and inconsistent bladder contractions. Paradoxically, FMS of the sacral nerves profoundly suppresses both detrusor hyper-reflexia in SCI [5] and normal voiding contractions in healthy volunteers [6]. The aim of this study was to help resolve this paradox by re-examining the effects of non-invasive FMS of the sacral nerves at different bladder volumes in patients with SCI and comparing the findings with those in healthy volunteers. Local ethics committee approval and informed consent was obtained.

Methods The bladder was filled slowly by catheter to three different levels in 3 patients with SCI and 3 healthy volunteers. Full capacity was indicated by detrusor hyper-reflexia in the patients and a strong desire to void in the volunteers. In both groups, FMS was applied over the sacrum to optimally stimulate the S3 nerve roots (by observing toes and anal sphincter contractions). When the bladder was filled to about half capacity, FMS at 15–25 pulses per second and between 70-100% maximum output of the stimulator for 5 seconds of continuous stimulation was given to attempt evocation of a detrusor contraction. Stimulation was then repeated again after further bladder filling to near capacity. To conclude the experiment, stimulation was given during the beginning of a hyper-reflexic contraction in the SCI patients and a voiding contraction in the normal volunteers to test for detrusor suppression.

Results When the bladder was at *half capacity,* FMS elicited small rapidly occurring artifact in detrusor pressure, probably the result of skeletal movement associated with stimulation, but in neither patient nor healthy volunteer could unequivocal detrusor contractions be seen time-locked to the stimulation (see Figure 1). However, at *near capacity* in the patients only, a hyper-reflexic contraction was occasionally seen but at variable latency ranging from a few seconds to several tens of seconds but always following cessation of the 5s stimulation. In marked contrast, at *full capacity* when hyper-reflexia or voiding took place, a highly consistent time-locked suppression of these detrusor contractions was observed with latencies less than 5s and taking between 10-20s to reach greater than 80% suppression.

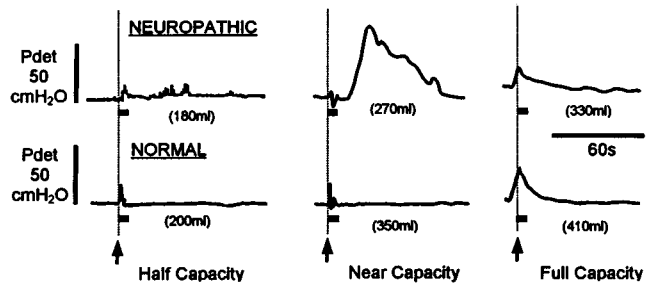


Figure 1 shows characteristic results from one each of the patients and volunteers. The arrows mark the beginning of magnetic stimulation in each trace and the black bars the duration of stimulation. The values in brackets were the approximate volumes in the bladder at each test.

Conclusions Non-invasive magnetic stimulation of the sacral nerves, unlike direct electrical stimulation of the sacral anterior roots, does not appear to stimulate pre-ganglionic parasympathetic pathways sufficient to cause significant bladder contractions. In spinally-injured patients, detrusor hyper-reflexia can sometimes be provoked when the bladder is near to full capacity, perhaps as a result of rebound excitation following reflex inhibition caused by magnetic stimulation of the larger sacral sensory pathways. We confirm previous studies showing that FMS of the sacral roots causes immediate and profound suppression of both detrusor hyper-reflexia in SCI and normal voiding contractions.

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## COMPUTER MODELS AS A RESEARCH TOOL FOR THE INVESTIGATION OF LOWER URINARY TRACT PHYSIOLOGY

Aims of Study: The current knowledge about the lower urinary tract function is not complete and sometimes ambiguous Therefore, to describe the neural control, assumptions have to be made concerning the mechanisms that are responsible for normal and pathological behaviour. So far experimentally verified quantitative descriptions of the complete lower urinary tract function in humans are not available. Computer models allow functional simulations in which the effect of various assumptions on lower urinary tract behaviour can be visualised. Our goal is to simulate signals that resemble detrusor and urethral pressure and flow as measured during a urodynamic investigation with a transparent model that includes afferents related to bladder wall tension and to urethral stretch.