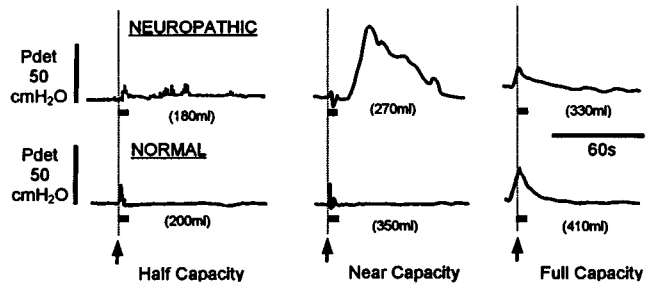

Figure 1 shows characteristic results from one each of the patients and volunteers. The arrows mark the beginning of magnetic stimulation in each trace and the black bars the duration of stimulation. The values in brackets were the approximate volumes in the bladder at each test.

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.

Methods: To simulate lower urinary tract behaviour, mechanical properties of the lower urinary tract and its neural control are modelled (figure 1). The mechanical part describes the properties of the detrusor and the urethra with its striated sphincter. The neural control is described by a number of blocks and connecting lines. The blocks represent anatomical structures where neural interaction is assumed to take place. The lines between these blocks represent connections that are assumed to be involved in lower urinary tract control. The blocks and connections that are used in our model are as much as possible based on anatomical structures that were shown to be important in lower urinary tract control.

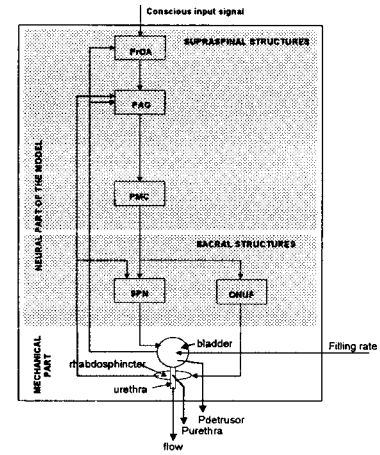

Figure 1: Schematic representation of the model. The model has two input signals and three output signals. The output signals are the detrusor pressure (Pdetrusor), the urethral pressure (Purethra) and the flow. The dashed lines represent the afferent signals. Other neural connections are characterised with solid lines.

Specific anatomical structures, both sacral and supraspinal structures, are described to be involved in the neural control of the lower urinary tract. The supraspinal control in our model consists of three blocks: the pontine micturition centre (PMC), the periaquaductal gray (PAG), and the preoptic area (PrOA). These structures are assumed to be involved in the 'unconscious' control of storage and micturition. We assume that the other centres known to be involved in lower urinary control affect the 'conscious' control of the lower urinary tract. Although the difference between conscious and unconscious control is not very distinct in vivo, we assume that under normal circumstances the conscious part only initiates micturition. The conscious control is represented by an input signal to the PrOA-block.

The sacral part of the neural control comprises two blocks that represent the sacral parasympathetic nucleus (SPN) and the nucleus of Onuf (ONUF). The SPN-block is assumed to have two input signals. The first input is connected to the PMC-block and the other input signal depends on the urethral stretch. The ONUF-block, like the SPN-block, is assumed to be connected to the PMC-block. The output of the ONUF-block represents the somatic nerves to the rhabdosphicter.

Results: With this rather simple model, which includes afferents related to bladder wall tension and urethral stretch, behaviour that resembles normal lower urinary tract behaviour was simulated. Every 100 sec a pressure of 10 cmH_2O was added to the detrusor pressure. These disturbances in the detrusor pressure did not cause micturition or leakage. The effect of these disturbances depends on the volume in the bladder. Due to a larger volume in the bladder, urethral pressure decreases more severely. At 550 sec the applied disturbances cause a decrease in urethral pressure as well as a detrusor contraction. Due to the assumption that urethral afferents have an excitatory effect on the SPN and an inhibitory effect on the PAG, at the start of micturition the input from the PrOA to the PAG is very important. If the inhibition of the PAG by the PrOA is still large enough, due to the increase in afferent signal from the urethra, micturition is inhibited although already some detrusor contraction has occurred.

Conclusion: One of the most important assumptions included in the present model is that afferent signals from the urethra are involved in the neural control on both sacral and supraspinal level. By including this assumption, behaviour that resembles normal lower urinary tract behaviour can be simulated. Besides normal behaviour also behaviour that resembled detrusor overactivity was simulated after disturbances were added to the model.

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PLASMA ELASTASE REGULATION IN STRESS URINARY INCONTINENCE

Aims of Study: Stress urinary incontinence (SUI) is the involuntary leakage of urine due to weakened support of the urnary bladder and urethra. Although the etiology of SUI is unknown, factors such as aging and