

BLADDER NECK RESPONSES IN THE ISOLATED SPINAL CORD ELICITED BY PUDENDAL NERVE STIMULATION

Aims of Study

Electrical pudendal nerve stimulation is known to have a potential neuromodulative effect on neurogenic and non-neurogenic bladder dysfunction. The efficiency of this procedure has been established and various bladder problems has been successfully treated. The neuronal effects of pudendal nerve stimulation has been studied in normal and spinal animals. Important aspects of the spinal mechanism of action has been identified (1). However in human, the underlying neuronal mechanism and the involved pathways remain unknown. In this prospective study we focused on the effects of pudendal nerve stimulation in patients with complete spinal cord injury in order to identify neuromodulative processes that occur exclusively on spinal level.

Methods

20 complete spinal male presenting with upper motor neuron lesion and neurogenic incontinence underwent detailed neurological examination according to the ASIA protocol to define the segmental level of spinal cord injury. Patients with incomplete lesions defined as any sensory or motor function preserved in the lowest sacral segment were excluded. Somatosensory evoked potentials (SSEP) evoked by pudendal and tibial nerve stimulation confirmed complete lesions of the ascending somatosensory pathways of both these nerves. To assess conus medullaris and cauda equina function electrophysiological recordings of bulbocavernosus (BCR) and anal reflexes as well as electromyogram and nerve conduction studies of the lower limbs were performed and showed unaffected sacral segments.

In 20 patients 56 stimulations using biphasic rectangular impulses (0.2 ms, 10 Hz) with intensities up to 100 mA were applied to the dorsal penil nerve. In six patients 18 stimulations were repeated after intravenous application of 7 mg phentolamine. Bladder, bladder neck, and external urethral sphincter pressure were continuously recorded by a microtip transducer. Correct placement of the pressure sensors within the bladder, bladder neck and external urethral sphincter was ensured radiologically. Data were statistically evaluated by analysis of variance for repeated measurements (ANOVA, level of significance $p < 0.05$).

Results

Electrical pudendal nerve stimulation was well tolerated in all cases. No patient reported any direct sensation from the external genitalia associated with stimulation. Mean initial bladder pressure increased during stimulation from 11 to 15 cm H₂O (non significant). Mean initial bladder neck pressure increased during stimulation from 33 to 75 cm H₂O ($p < 0.001$). Mean initial external urethral sphincter pressure increased during stimulation from 59 to 135 cm H₂O. The latencies to the external urethral sphincter responses range between 27 and 41 ms. The latencies to the bladder neck responses range between 188 and 412 ms. Phentolamine decreased initial bladder neck pressure from 33 to 24 cm H₂O ($p < 0.05$) and reduced the pressure rise during stimulation significantly ($p < 0.05$).

Conclusions

Pudendal nerve stimulation evoke somatic responses in the external urethral sphincter and autonomic responses in the smooth muscle sphincter controlling the bladder neck. Longer latencies of the bladder neck responses and the sensitivity to the α -blocking agent phentolamine indicate that sympathetic α -adrenergic fibres are involved in this responses. Therefore, somatic afferent fibres of the pudendal nerve project on thoracolumbar sympathetic neurons to the bladder neck and modulate their function. This spinal interaction of somatic and autonomic pathways provide a acute neuromodulative effect which support bladder neck competence and promote continence. The underlying neuronal mechanism occur below a complete spinal cord lesion exclusively on spinal level and represent a neuromodulative process in the isolated spinal cord of spinal human.

This work was partially funded by the International Institute for Research in Paraplegia (P17/97-2000) and the Swiss National Science Foundation (32-52562.97)

1. The neurophysiological basis of bladder inhibition in response to intravaginal electrical stimulation. J Urol 1983;129(2):405-10.

