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Title: DETRUSOR AND BLOOD PRESSURE RESPONSES TO DORSAL PENILE NERVE STIMULATION DURING HYPER-REFLEXIC CONTRACTION OF THE BLADDER IN PATIENTS WITH CERVICAL CORD INJURY

Bladder hyperreflexia with resultant incontinence is a major problem in suprasacral spinal cord injury (SCI) patients. A management option for incontinence is electrical stimulation to pelvic structure.[1] Group of studies expecting a direct immediate effect of sacral afferent nerve stimulation showed a consistent suppressive effect on provoked reflex contraction.[2] A possible risk of electrical stimulation in SCI patients with high neurological level is the occurrence of autonomic dysreflexia (AD). Although severe hypertension during electroejaculation using a rectal probe has been reported,[3] there is no report about autonomic dysreflexia caused by sacral afferent stimulation to inhibit bladder contraction in SCI patients.

Aims of Study

The aim of study was to investigate the immediate effect of dorsal penile nerve stimulation on detrusor pressure and blood pressure during hyper-reflexic contraction of the bladder in patients with cervical SCI.

Methods

Five male subjects with complete cervical SCI participated. Reflex voiding and/or CIC was the method of bladder emptying in all patients. All the subjects had symptoms of AD when the bladder was full and drainage was delayed.

Standard water-cystometry was performed. Normal saline was infused at a rate of 30mL/min. Blood pressure (BP) was monitored by a percutaneous radial artery catheter. The arterial catheter was connected to a pressure transducer, and BP was recorded simultaneously with the P_{det} . BP was also measured manually using an electronic BP cuff on the contralateral arm.

The dorsal penile nerve (DPN) was stimulated using surface electrodes. Stimulation parameters were biphasic rectangular pulses of 25Hz frequency, 250 μ sec pulse width. Stimulation intensity was twice the threshold of the pudendo-anal reflex.

When hyper-reflexic contraction of the bladder was observed, infusion was stopped and electrical stimulation (ES) applied for one minute. If reflex contraction was suppressed by the electrical stimulation, saline infusion was restarted. This procedure was repeated until the infused volume reached twice the volume at the first reflex contraction. The test protocol was also designed to stop infusion if 1)ES did not suppress reflex contraction, 2) infused volume reached 450ml, or 3) the subject could not tolerate the test.

Results

There were no remarkable changes in BP if DPN stimulation was applied before starting infusion.

There was an initial reflex contraction of the bladder when infused volume reached 75-210mL. The BP rose as detrusor pressure rose in all cases. The mean BP measured manually during the first and the last contraction were 159/101 and 172/109 mmHg (systolic/diastolic) respectively. All the reflex bladder contractions were effectively suppressed by DPN stimulation in all three cases. As the detrusor pressure decreased on DPN stimulation, radial arterial pressure also dropped. The changes of radial arterial

pressure correlated well with detrusor pressure changes in all five cases. The manually measured BP during the first and last contraction was lowered to 129/85 and 141/92 mmHg (systolic/diastolic) respectively by ES.

The suppressed detrusor pressure and BP with DPN stimulation tended to increase as the infused volume increased.

Conclusions

The results of this study indicated that DPN stimulation to inhibit reflex bladder contraction did not produce AD. On the contrary, DPN stimulation lowered the elevated BP of AD, probably by suppression of detrusor contraction.

References

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Title: SELF-CONTROLLED DORSAL PENILE NERVE STIMULATION TO INHIBIT BLADDER HYPER-REFLEXIA IN INCOMPLETE SPINAL CORD INJURY

Electrical stimulation to inhibit hyper-reflexic contraction has been applied to spinal cord injury(SCI) patients. Some reports regarding therapeutic effects showed favorable results in urodynamic study after four to sixteen weeks of stimulation,[1] but the rate of achievement of complete continence was less than 20%. The other group of studies expecting a direct immediate effect of stimulation showed a consistent suppression effect on provoked reflex contraction.[2] However there was no report of clinical trial using this technique.

Aim of Study:

This case report describes for the first time the application of self-controlled dorsal penile nerve (DPN) stimulation for bladder hyperreflexia in a patient with SCI.

Methods:

The patient was a 33-year-old male with C-6 incomplete quadriplegia. He managed his bladder with IC by himself every three to four hours and had been taking Ditropan 25mg per day. He reported occasional leakage between catheterization. He used condom catheter during the night and while he was out. He could feel the sensation of bladder contraction just before urination, but it was so urgent that he could not maintain continence before starting catheterization.

The first urodynamic study was performed to confirm the effect of DPN stimulation on provoked reflex contraction. Standard water-cystometry was performed at a fill-rate of 35mL/min. There was initial reflex contraction when infused volume reached 214mL. Peak pressure was 91.8 cmH₂O. Infusion was stopped and 60mL of saline was removed to abolish the reflex contraction. We repeated removal and infusion of 60mL saline to confirm reproducibility of provocation. The patient reported sensation of contraction every time when reflex contractions occurred. After confirming the reproducibility of provoked reflex contraction, DPN stimulation was applied using portable electrical stimulator. Stimulation parameters were biphasic rectangular pulses of 25Hz frequency, 250μsec pulse width. Stimulation intensity was twice the threshold of the pudendoanal reflex. When a contraction was clearly provoked as identified by the P_{det} pressure exceeding 15cmH₂O, stimulation was started and continued for one minute. Detrusor pressure was suppressed to its initial level and remained stable for 136 seconds after stopping stimulation. As detrusor pressure rose again by provocation or spontaneously, we applied stimulations on three more occasions. The suppressions were consistent, but the duration of suppression after stopping stimulation was shortened compared to the initial trial.

The second urodynamic study was performed to identify the effect of brief intermittent stimulation on reflex contraction during slow filling to simulate physiological conditions. Saline was infused at a rate of 30mL/min until the infused volume reached 100mL, and then at 3mL/min. While the bladder was slowly filled, the patient himself applied stimulation for one minute, whenever he felt a sensation of contraction. The first contraction occurred when 141mL of saline infused, and was effectively suppressed by stimulation. All the subsequent contractions

also were suppressed effectively by stimulations, but the duration of effect became shorter according to the increasing volume infused.

Results of Home Use:

We suggested that he use DPN stimulation in addition to IC to control urgency-incontinence at home. He could start stimulation on sensation of bladder contraction and continued it 60 seconds. He used stimulation only when needed to control voiding during the daytime. He wrote a brief voiding diary for three weeks. He recorded sixty-six catheterizations excluding the first catheterization after awakening. On twelve occasions he started stimulation when he felt the bladder contracting, and was incontinent on only one of those occasions. The catheterized volume was markedly increased from $205.0 \pm 55.3\text{mL}$ to $352.9 \pm 36.7\text{mL}$ when he used DPN stimulation before catheterization. The interval between catheterizations was also prolonged from 242.1 ± 59.0 to 284.2 ± 108.6 minutes with use of stimulation. He feels confident he can maintain continence by the use of self-controlled DPN stimulation. He feels less need to limit his fluid intake.

Conclusion:

Self-controlled DPN electrical stimulation is a simple, non-invasive technique that may improve bladder capacity and continence in selected SCI patients with bladder hyper-reflexia. This technique has the potential to minimize fluid intake restriction, to avoid unnecessary catheterization and to reduce the need for urine collection devices. This technique can be used as an adjuvant method of incontinence management.

REFERENCES

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