Abstract

The possible risk of electrical stimulation to high spinal cord injury patients is occurrence of autonomic dysreflexia. The aim of this report was to investigate the immediate effect of dorsal penile nerve stimulation to the detrusor and blood pressure during hyper-reflexic contraction of bladder in patients with cervical SCI. Three male subjects with complete cervical SCI were tested using water-cystometry. Detrusor and radial arterial pressure were recorded simultaneously. When the hyper-reflexic contraction was observed, electrical stimulation for one minute was conducted. The blood pressure rose remarkably as detrusor pressure rise by contraction in all cases. All the reflex contractions were effectively suppressed by dorsal penile nerve stimulation. As the detrusor pressure decreased by stimulation, radial arterial pressure also dropped immediately. This result represented that dorsal penile nerve stimulation could lower the elevated blood pressure in autonomic dysreflexia by suppression of detrusor contraction.

Introduction

Bladder hyperreflexia with resultant incontinence is a major problem in suprasacral spinal cord injury (SCI) patients. Pharmacological treatment with anticholinergic agents helps to prevent incontinence, but some patients do not tolerate the side effects of the drugs.1

Another management option for incontinence is electrical stimulation. Electrical stimulation to inhibit hyper-reflexic contraction has been applied to SCI patients. There have been two types of application for SCI patients. One expects a therapeutic effect to continue after some treatment period, and the other expects an immediate, direct suppressive effect on hyper-reflexic contraction of the bladder. Some reports regarding therapeutic effects showed favorable results in urodynamic study after four to sixteen weeks of stimulation,2,3 but the rate of achievement of complete continence was less than 20%. The other group of studies expecting a direct immediate effect of sacral afferent nerve stimulation showed a consistent suppression effect on provoked reflex contraction.4 This stimulation expecting a direct immediate response effectively inhibited unwanted detrusor contractions and increased cystometric capacity in spinal cord injury patients.5

The possible risk of electrical stimulation to SCI patients with high neurological level is occurrence of autonomic dysreflexia (AD). Recent evidence suggests that functional electrical stimulation (FES) assisted training or therapeutic interventions may induce AD in high level SCI patients.6 Although severe hypertension during electroejaculation using a rectal probe was reported,7 there was no report about autonomic dysreflexia caused by sacral afferent stimulation to inhibit the bladder contraction in SCI patient.

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

Methods

Three male subjects with complete cervical SCI, age 33 - 36 years, duration of injury 4 – 10 years, were participated. Reflex voiding was the method of bladder emptying in all patients, combined with CIC in one. None of the three patients had any significant urological abnormality or symptomatic urinary tract infection at the time of the study. Any medication for bladder dysfunction was stopped at least 72 hour before testing, and none of them use antihypertensive medication routinely. All the subjects had symptoms of AD, when if the bladder was full and drainage was delayed.

Test settings

Standard water-cystometry which involved simultaneous measurements of intra-vesical pressure, intra-rectal pressure and the subtracted detrusor
pressure ($P_{det}$), was performed with the patient supine at a fill-rate of 30mL/min through a urethral catheter with normal saline at room temperature. Blood pressure (BP) was monitored by an intra-arterial catheter introduced percutaneously into the radial artery. The arterial catheter was connected to one of pressure measuring units in cystometry, and BP was traced simultaneously with the $P_{det}$. BP was also measured manually using electronic BP cuff at contralateral upper arm during the important events of the test.

Stimulation to suppress the reflex contraction was applied using a portable neuromuscular stimulator. The dorsal penile nerve (DPN) was stimulated using round surface electrodes of 1cm diameter. The cathode was placed proximally and anode distally on the dorsum of the penile shaft 2 cm apart. Stimulation parameters were biphasic rectangular pulses of 25Hz frequency, 250μsec pulse width. Stimulation intensity was twice the threshold of the pudendoanal reflex.

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

Rapid-acting antihypertensive agents were prepared for uncontrolled AD. The approval of the local ethical committee and the informed consent of the patients were obtained.

**Results**

Initial BP before starting infusion were 112 - 131mmHg(systolic) and 69 - 89mmHg(diastolic). There were no remarkable changes in BP by DPN stimulation before starting infusion.

There was initial reflex contraction when infused volume reached 75 - 210mL. There were 2 to 3 more reflex contractions until infused volume reached twice of volume at the first contraction.

The BP rose as detrusor pressure rise remarkably by contraction in all cases (figure 1). 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 contractions were effectively suppressed by DPN stimulation in all three cases. As the detrusor pressure decreased by DPN stimulation, radial arterial pressure also dropped. The changes of radial arterial pressure correlated well with detrusor pressure changes in all three cases (figure 1). The manually measured BP during the first an 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 (figure 2).

![Figure 1. Detrusor pressure ($P_{det}$) and radial arterial pressure (BP) Responses on dorsal penile nerve stimulation during hyper-reflexic contraction of bladder. Data from patient number 1. Electrical stimulation suppressed both $P_{det}$ and BP.](image1)

![Figure 2. Changes of blood pressure with electrical stimulation during the first reflex contraction and the last contraction.](image2)

**Discussion/Conclusions**

The present study demonstrated a favorable response of BP to DPN stimulation in cervical SCI patients. It has been known for many years that detrusor instability can be improved by electrical stimulation of pudendal afferent fibers. Stimulation...
of large sacral afferents has been shown to produce acute inhibition of detrusor hyper-reflexia by pudendal-pelvic spinal reflex pathways.\textsuperscript{10}

Limited experience exists in its use in the treatment of neurogenic bladder of SCI. Hyper-reflexic contractions in suprasacral SCI were suppressed effectively by either electrical stimulation of the pudendal nerve.\textsuperscript{4} Wheeler et al\textsuperscript{11} reported that the cystometric bladder volume during DPN stimulation also increased.

The possible risk of electrical stimulation to SCI patients with high neurological level is occurrence of autonomic dysreflexia. There was a report, which described severe hypertension in patients with high SCI undergoing electro-ejaculation.\textsuperscript{7} On the contrary, BP was not elevated remarkably by electrical stimulation alone before starting infusion in our study. This finding could be explained by different stimulation site and intensity.

Recently, increases in BP and concomitant bradycardia, suggestive of AD, have been documented during FES in individuals with a high SCI.\textsuperscript{6} These responses were unaffected by the use of topical anesthetic cream on the skin at the stimulation site. This suggests that other mechanisms than skin nociception are operative in FES-induced AD.\textsuperscript{12}

FES nociception could come from stimulation of one or a combination of the following: electrical activation of pain fibers (A delta and C fibers) of the skin, or muscle: isometric muscle contraction and its resultant ischemia, metabolites and/or musculotenous stress.\textsuperscript{13}

The results of this study represented that DPN stimulation to inhibit the reflex contraction did not produce AD. In addition, DPN stimulation could lower the elevated BP in AD by suppression of detrusor contraction.

References