Treatment of external urethral sphincter hypertonicity by pudendal nerve block using phenol solution in patients with spinal cord injury

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We report our experience utilizing the technique of phenol block of the pudendal nerve in the treatment of voiding dysfunction due to hypertonicity of the external urethral sphincter. We have performed 13 pudendal nerve blocks using a 7% phenol solution in seven patients with spinal cord injury who could not obtain relaxation of the external urethral sphincter with a large postvoid urine residual (150 ml to 600 ml) despite large doses of antispasticity drugs and intermittent catheterisations over three weeks. These drugs were discontinued at least 48 hours before this procedure. The efficacy of the pudendal nerve block could also be tested by the ease of facilitating micturition during or just after the block and measuring the amount of postvoid residual urine and intravesical leak pressure. A pudendal nerve block was produced by injecting a 7% phenol solution medial to the ischial tuberosity having specifically localized the nerve by electrical stimulation. This procedure improved the voiding pattern dramatically, leading to a full stream of urine and a remarkable decrease of postvoid residual volume and intravesical leak pressure. The mean difference of the postvoid residual volume and the intravesical leak pressure before and after pudendal nerve block was 255.7 ml and 57.5 cmH₂O, respectively. We conclude that pudendal nerve block with a phenol solution as a treatment of external urethral sphincter hypertonicity was effective, easy to perform, and had no complication. This treatment should be considered as a possible alternative to more invasive surgical procedures.

Keywords: pudendal nerve block; external urethral sphincter hypertonicity; phenol; spinal cord injury

Introduction

The ideal voiding pattern of the neuropathic bladder is characterized by a low voiding bladder pressure, low postvoid residual volume, no episodes of urinary infection, and continence between catheterisations. External urethral sphincter hypertonicity with or without detrusor hyperreflexia has been known to be a common cause of an unbalanced neuropathic bladder in patients with a spinal cord injury. Voiding dysfunction due to hypertonic detrusor contraction and dyssynergic contraction of the external urethral sphincter presents a frustrating problem in the management of such patients.

Pharmacological treatment of neurogenic bladder dysfunction is aimed at the alteration of the tonicity in various sites in the lower urinary tract. Unfortunately, the use of oral medications for relaxation of the external urethral sphincter is often limited by an inadequate effect and/or systemic side effects. Inade-

quate voiding with persistent high postvoid residual volume and repeated urinary tract infections can be treated by transurethral sphincterotomy, placement of urethral stents or by catheterisation. Surgical management to decrease tonicity of the external urethral sphincter, however, may sacrifice urinary continence and lead to other surgical complications.

We report our experience utilizing the technique of phenol block of the pudendal nerve in the treatment of voiding dysfunction due to hypertonicity of the external urethral sphincter in patients who were refractory to treatment with drugs and bladder retraining with intermittent catheterisations. To control the hypertonicity of the external urethral sphincter, we employed pharmacotherapy first and then performed pudendal nerve block using a 7% phenol solution.

Materials and methods

Seven patients were identified who demonstrated ineffective external urethral sphincter relaxation with large postvoid urine residuals (150 ml to 600 ml)

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despite the use of large doses of antispasticity medications and intermittent catheterisations. All patients had an upper motor neuron spinal cord injury with external sphincter hypertonicity and/or hyperreflexic bladder with no abnormalities of the sacral reflex arc. Three had complete paraplegia, two incomplete, and two had complete quadriplegia. The age of the subjects ranged from 24 to 58 years, with a mean age of 42.3 years. All subjects were male. ASIA (American Spinal Injury Association) Impairment Scale was A in three, B and C in one, and D in two. The mean time since injury was 14.3 months. The urological and neurological states were stable for at least nine months prior to the treatment (Table 1). All patients had failed to respond to bladder retraining with intermittent catheterisation and oral antispasticity medication to relax the external urethral sphincter hypertonicity. The medication used was baclofen 80 mg/day and diazepam 20 mg/day over three weeks. There was no patient with vesicoureteral reflux. The medication was discontinued at least 48 hours before the pudendal nerve block. Patients who exhibited marked straining upon urination, large residual urine volume, and a definite opening of the bladder neck as shown by a voiding cystourethrogram were considered to be suitable for a pudendal nerve block. The presence of contrast medium in the region of the bladder neck during filling was indicative of an open bladder neck. An anatomical outlet obstruction was ruled out before selecting the patients.

The pudendal nerve was approached through the perineum with the patient supine with hips flexed. The nerve was identified at its exit from Alcock's canal. A 10 cm Teflon-coated block needle was introduced medial to the ischial tuberosity and directed medial and cephalad to meet the palpating finger in the rectum at a location just medial to the posterior inferior iliac spine. A 'pop' was felt when the needle penetrated the sacrospinous ligament. With interrupted galvanic electrical stimulation (2–5 mA, 1 msec), contraction of the anal sphincter and external urethral anterior to the anterior wall of the rectum on the finger tip was palpable. When the strongest tonic contraction of the external urethral sphincter or anal sphincter by a given stimulation

intensity was felt, 0.2 ml of a 7% aqueous phenol solution was injected at the point and repeated injections were administered at other adjacent points. By assessing anal sphincter tone and external urethral sphincter tone, we could judge whether the injection was placed in the proper position. Effective urine passage by suprapubic compression was confirmed. If the unilateral pudendal nerve block was ineffective, then a bilateral nerve block was performed. Of the seven patients, six required a bilateral pudendal nerve block. The amount of phenol solution injected at each pudendal nerve ranged from 1.0 ml to 6.0 ml.

The efficacy of the pudendal nerve block could also be tested by the ease of facilitating micturition and measuring the amount of postvoid residual and intravesical leak pressure (leak pressure).^{1–3} The postvoid residual was defined as the volume of fluid remaining in the bladder immediately following the completion of micturition by suprapubic tapping or Credé maneuver after filling the bladder until urethral leakage occurs or until the maximum volume is achieved. The leak pressure was defined as the intravesical pressure when urethral leakage or urine flow occurs. We performed simple water cystometry with a warmed saline (37°C) filling rate of 50 ml/min. The leak pressure could not be calculated in one patient because of no urethral leakage at 700 ml filling.

The outcome was graded as follows: 'Excellent' was recorded when the postvoid residual volume decreased more than 150 ml compared to the before block; 'Good' was recorded when there was a decrease in postvoid residual volume of 101–150 ml; 'Fair' and 'failure' were recorded when the postvoid residual volume decreased 51–100 ml and 0–50 ml, respectively. The mean follow-up period after the block was 10.1 months. After the block, bladder retraining with intermittent catheterisations was continued until the postvoid residual volume decreased below 100 ml. The amount of postvoid residuals before and after the procedure was compared and analysed by *t*-test.

Results

Five patients who underwent a pudendal block had 'excellent' results. Two patients had 'good' and 'fair'

Table 1 Subjects and clinical significances

Age/Sex	ASIA scale	Time after injury	Duration of follow-up after block	Residual urine (ml) (before/after block)	Effect of pudendal block
52-M	T9 ASIA A	9 months	7 months	600/300	Excellent
45-M	C5 ASIA B	13 months	11 months	470/140	Excellent
58-M	T10 ASIA C	10 months	16 months	500/100	Excellent
32-M	C7 ASIA D	21 months	12 months	150/20	Good
49-M	T7 ASIA A	19 months	9 months	250/150	Fair
24-M	T10 ASIA A	12 months	11 months	450/120	Excellent
36-M	T4 ASIA D	16 months	9 months	400/200	Excellent

Effect of decreasing residual urine volume: Excellent > 150 ml; Good 101–150 ml; Fair 51–100 ml; Failure 0–50 ml

results, respectively. The mean difference of the postvoid residual before and after the pudendal nerve block was 255.7 ml (mean 64.3% decrease). The mean leak pressure after the pudendal nerve block was 39.3 cmH₂O (mean 57.6% decrease) with a range of 28 to 55 cmH₂O. The difference of the postvoid residual and the leak pressure before and after the block was highly statistically significant ($P < 0.001$). This procedure improved the voiding pattern by suprapubic tapping or Credé maneuver dramatically, leading to low postvoid residual and low leak pressure. No patients required surgical procedures to decrease outlet resistance during follow-up after the block. There was no impairment of urinary continence.

Discussion

Normal voiding is characterized by complete relaxation of the external urethral sphincter, detrusor contraction and opening of the proximal urethral sphincteric mechanism.⁴ Treatment and retraining of neurogenic bladder dysfunction in patients with spinal cord injury should be aimed at coordination of detrusor contraction or application of external force to the detrusor and resistance of urethral outflow. It is important to maintain low postvoid residual volume and low intravesical voiding pressure in order to limit infection or further pathology in the upper urinary tract system.^{5,6}

Hypertonicity of the external urethral sphincter is a common cause of an unbalanced neuropathic bladder in patients with a spinal cord injury. Such patients may present with a varied symptom complex that cannot simply be defined by the neurological lesion of the spinal cord.⁷ Kaplan *et al*⁷ described that there was a general correlation between the neurological level of injury and the expected vesicourethral function, which was neither absolute nor specific. They suggested that clinical neurological examination alone is not an adequate barometer to predict neurological dysfunction. In our series, five subjects had large vesical capacities despite a neurological level of injury at the cervical or thoracic spinal cord with spastic paralysis of the legs and intact sacral reflex activity.

In most cases, detrusor-external sphincter dyssynergia produces significant outlet obstruction, and the bladder management program must aim to reduce the sphincteric resistance. Hypertonicity of the external urethral sphincter is often refractory to antispasticity drugs. However, α -antagonist medication often reduces the resistance of the bladder neck.

Treatment options such as sphincterotomy, urethral stents, urethral and suprapubic catheters are not without complication or failure.⁸ However, they may be necessary for patients who are unable to perform intermittent catheterisations.⁸ Transurethral sphincterotomy is commonly performed in patients with external sphincter hypertonicity. Juma *et al*⁹ reported long term complications of transurethral sphincterotomy for 63 patients. Nineteen (30%) had significant

upper tract complications. Fifty percent of upper tract complications developed more than 2 years after sphincterotomy. Thirty patients had lower urinary tract complications including recurrent symptomatic urinary tract infection, bladder stone, urethral diverticulum, urethral stricture, bladder neck stenosis, and recurrent epididymitis. Yang and Mayo¹⁰ described a 50% failure rate in their series of external urethral sphincterotomies. Causes for failure included recurrent sphincter dyssynergia, detrusor hypocontractility, bladder neck contracture, and stricture of the external sphincter. In addition, the reoperation rate was 32%. The external urethral sphincterotomy, even in appropriately selected patients, can have a high failure rate over time.

Similarly, Juma *et al*¹¹ reported on the use of a urethral wallstent for the treatment of external urethral sphincter hypertonicity. They found this to be a simple procedure with a short hospital stay and low morbidity, however, its long term safety has not yet been established. Rivas *et al*¹² reported complications of stent insertion in six of 26 patients who received stent insertion. The complications were device migration and secondary bladder neck obstruction.

Pudendal nerve block using a phenol solution in patients with voiding dysfunction due to external urethral sphincter hypertonicity is useful as a minimally invasive measure. Phenol temporarily interferes with nerve function resulting in a decrease in muscle tone and has been widely used in the management of limb spasticity. Methods recommended for control of the spasticity include percutaneous injection of muscle motor points, percutaneous blocks of peripheral nerves, and open injection of phenol to the motor branches of peripheral nerves.

The injection of phenol into a mixed nerve, including the pudendal nerve, can result in the undesirable loss of sensation and painful dysesthesia. A pudendal nerve block in a patient with normal cutaneous sensation in the distribution of the pudendal nerve will result in absent or decreased sensation in the perineal and sacrolabial skin of the injected site.¹³ However, this predictable sensory complication might be negligible in those patients with spinal cord injury who have absent or abnormal sensation in that area.

Various authors have noted an unpredictable duration of the effect of the nerve blocks. Our subjects were followed for a maximum of 16 months (7 months to 16 months) and all subjects maintained their immediate post-nerve block residual volume during the follow-up period.

Urinary incontinence is a frequent complication of surgical procedures to correct neuropathic obstruction of the external urethral sphincter. With the use of pudendal nerve block with phenol solution, however, tissue consistency of the external urethral sphincter and control of urinary continence can be preserved. In our series, no additional disability as a result of the procedure, including incontinence, was found.

In conclusion, pudendal nerve block with a phenol solution as a treatment of external urethral sphincter hypertonicity was effective, easy to perform, and had no complications. This treatment should be considered as a possible alternative to more invasive surgical procedures.

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