Normalization of Spinal Cord Injury Urodynamics after Intrathecal Baclofen Therapy: A Case Report

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ABSTRACT
Setting: Acute Rehabilitation Hospital
Patient: A 47-year-old man fell 7 feet, resulting in acute C4-C5 disc herniation with disruption of the anterior and posterior longitudinal ligaments, with ventral spinal cord compression and cord edema.
Case Description: A 47-year-old man fell 7 feet, resulting in acute C4-C5 disc herniation with disruption of the anterior and posterior longitudinal ligaments, with ventral spinal cord compression and cord edema. He underwent an anterior cervical discectomy at C4-5 and C5-6 with anterior cervical fusion from C4-C7. Postoperatively his examination was consistent with C4 ASIA D tetraplegia in a central cord syndrome pattern. Cytosensormetry (CMG) revealed a hyperactive detrusor with sphincter dyssynergia. This was managed with intermittent self-catheterization. He was impaired by severe lower extremity spasticity, despite oral medication and botulinum toxin injections. He underwent a successful intrathecal baclofen (ITB) trial, and an intrathecal pump was placed two years after initial injury. Eight years after pump placement, CMG revealed relatively normal detrusor and minimal sphincter tone. He voluntarily voids with urgency but improved continence. Although the main indication for ITB therapy is spasticity, improved urodynamics can be an additional benefit.

Key Words: Spinal cord injury, bladder dysfunction, intrathecal baclofen, neurogenic bladder, rehabilitation

INTRODUCTION
Bladder management after spinal cord injury (SCI) is a challenging. Historically, renal failure was the leading cause of death in patients with SCI, therefore close attention is necessary. In neurologically intact individuals, self-controlled micturition is possible, whereas detrusor contractions are accompanied by several well-timed events controlled by the pontine micturition center. These include relaxation of the external urinary sphincter, pelvic floor, bladder neck, and internal sphincter. After incomplete suprasacral SCI, reflexes usually return, and can become exaggerated, and detrusor hyperreflexia and sphincter dyssynergia. The patient then may demonstrate urge incontinence, and incomplete, high pressure voiding with potential vesicoureteral reflux. Filling pressures should be less than 40 cm H2O and voiding pressures less than 60 cm H2O. The goals of treatment include low-pressure urine storage, low-pressure voiding, ideally with continence. This can sometimes be achieved with some combination of oral pharmacologic agents, injections, catheterization, electric stimulation or surgery.

A common problem of suprasacral SCI is lower extremity spasticity. This can cause multiple problems including pain, difficulty with hygiene, incontinence, activity and gait disturbance. Oral baclofen is a first line agent for spasticity in SCI and may affect urodynamics but is often limited by inadequate effect and/or systemic side effects such as drowsiness, insomnia, dizziness, weakness, ataxia and mental confusion. Baclofen works as a γ-aminobutyric acid B (GABA-B) receptor agonist in the central nervous system. Intrathecal baclofen (ITB) pumps deliver localized medication therapy that can be safe and effective for long-term treatment of uncontrolled spasticity in patient with SCI.

Another benefit of ITB therapy is improvement in urodynamic parameters. Nanninga et al. reported 7 cases (6 SCI and 1 transverse myelitis) with neurogenic bladders having decreased sphincter activity during bladder contraction after ITB therapy. Kuns and Delhais reported 9 cases (3 multiple sclerosis (MS) and 6 SCI) with increased bladder capacity, a decrease in residual volume, and diminished pelvic floor spasm after ITB therapy. Three patients became continent. Stiers et al. reported ten patients (SCI, MS, syringomyelia, hereditary spastic paraplegia (HSP)), after ITB therapy had resolution of “irritative” voiding and urge incontinence of the uninhibited bladder. Four of ten patients had abolition of detrusor sphincter dyssynergia (DSD). There was no difference in bladder capacity, compliance, sensation, or voiding pressure. Bushman et al. reported 3 HSP cases with increased bladder compliance and capacity with one patient having resolution of DSD after ITB therapy. Mertens et al. reported 17 patients (SCI, MS, syringomyelia, Friedrich’s Ataxia, cervical myelopathy, dorsal myelopathy) in which 50% had a decrease in detrusor hyperreflexia with reduction of leakage and increase in functional bladder capacity after ITB therapy.

In the physiatric literature, Frost et al. reported three persons with complete paraplegia who benefited from ITB pump placement with either increased bladder capacity or decreased DSD. They reported that ITB caused “simplification of bladder program.” Talalla et al. reported six SCI cases with reduced but incomplete urethral pressure after ITB therapy. We present the case of a man with central cord syndrome who had marked improvement in urodynamics parameters with ITB therapy.

DISCUSSION
ITB pumps are typically placed in patients with upper motor neuron disease for intractable spasticity. Besides the decrease in spasticity, there are marked effects by normalizing urodynamics. This is probably due to the significant inhibitory effect on the micturition reflex, depressing detrusor contraction strength and micturition efficiency, while decreasing bladder capacity. The literature reports vary in the degree of normalization among several upper motor neuron pathologies. Our case shows a marked resolution of neurogenic bladder signs and symptoms with ITB therapy.

Several typical methods of neurogenic bladder treatment have a myriad of complications in themselves including indwelling catheters causing bladder colonization, recurrent bladder infections, kidney and bladder stones, urethral strictures, fistulae, erosions, and bladder cancer. Bladder distention can also lead to lifethreatening autonomic dysreflexia. ITB pump patients have their own set of complications including scar formation, extrusion of the catheter, catheter granuloma and abrupt withdrawal causing anxiety, convulsions, tachyarythmias, hallucinations, autonomic dysreflexia, malignant hyperthermia, and neuroleptic-malignant syndrome. ITB therapy is discontinued, there may be regression of dyssynergic voiding which may trigger autonomic dysreflexia or hyponatremia. Pump overdose is associated with respiratory distress, coma, and seizure threshold reduction. Complications of the conservative management versus more invasive ITB pump placement may be weighty.

There are several questions to be posed about ITB therapy for neurogenic bladder considering the good but inconsistent results in the literature. Talalla et al. speculated whether the urologic affects may be dose related. Is there a normal range that may cause decreased DSD? What is the exact mechanism of action of resolution of neurogenic bladder symptoms with baclofen at the spinal cord? Nanninga et al. proposed that besides changing the bladder innervation, baclofen might also change bladder peristalsis. We present the case of a man with central cord syndrome who had marked improvement in urodynamics after ITB therapy.

CONCLUSION
There have been reports of various levels of normalization of urodynamics in upper motor neuron disease. Although the indication for ITB pump placement is usually for spasticity, normalization of urodynamics is an additional benefit. It may be an alternative method of treatment for neurogenic bladder.

REFERENCES