Guest Editorial

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Continence issues in the patient with neurotrauma

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The bladder has two main functions: storage of urine under low intravesical pressure and periodical release of urine in a controlled coordinated manner during an acceptable time to void. The ability to maintain continence and release urine is under voluntary control mediated by neural inputs to the lower urinary tract from centers located in the brain and spinal cord. A neurogenic bladder dysfunction is result of disease or injury to the neural pathways or neuromuscular junctions controlling these functions, and commonly occurs after spinal cord injury.

SCI incidence is estimated at 15-40 cases per million population. In the past, renal failure was the leading cause of death after SCI[1]. Today mortality from SCI has declined dramatically owing to improved management of urological dysfunction associated with SCI[2].

Normal micturition cycle

Normal bladder function of storage and voiding are controlled by voluntary and reflex mechanism. During the storage phase the bladder expands slowly until an appropriate bladder volume is reached. Sphincter activity must be coordinated with bladder filling to allow adequate storage. Once threshold bladder volume is reached, the sacral reflex centers at S2 through S4 are stimulated with subsequent impulses sent from sacral cord upto the pontine micturition center and frontal cortex. Efferent impulses from the brain stem stimulate bladder contraction, coordinated bladder neck relaxation resulting in sensation of void. If socially appropriate, a conscious decision to void is initiated under the voluntary control of frontal cortex which sends regulatory impulses to the external sphincter, by way of corticospinal tract to the pudendal nerves[3]. Voiding involves detrusor contraction and relaxation of the internal and external sphincters. Conversely if voiding is delayed, voluntary contraction of external sphincter leads to detrusor relaxation, internal sphincter relaxation and additional storage of urine.

During storage phase, a sympathetic reflex activity through a local sacrolumbar spinal reflex pathway is triggered by afferent impulses in the pelvic nerves. This negative feedback mechanism contributes to the storage function of the bladder by increasing bladder capacity, and decreasing the frequency and amplitude of bladder contractions.

Spinal Shock

After a significant SCI, a period of decreased excitability of spinal cord segments at and below the level of the lesion occurs, referred to as "spinal shock". It includes suppression of autonomic activity as well as somatic activity, and the bladder is acontractile and areflexic. The bladder neck is generally closed and competent unless there has been prior surgery or in some cases of thoracolumbar and presumably sympathetic injury. Because sphincter tone exists, urinary incontinence generally does not result unless there gross overdistention with overflow. Urinary retention is the rule, and catheterization is necessary. Clean intermittent catheterization (CIC) is an excellent method of management during this period, but recent reports have shown no difference in outcome when a small-bore Foleys catheter or a suprapubic tube is used at this stage.

Reflex detrusor contractility generally returns if the distal spinal cord is intact and is simply isolated from higher centers. But the strength and duration of such involuntary contraction increases with time. These produce involuntary voiding usually with incomplete bladder emptying. Spinal shock generally lasts for 6 to 12 weeks in complete suprasacral spinal cord lesions however it may last upto 1 to 2 years. In incomplete suprasacral lesions it may last for few days only.

Suprasacral Spinal Cord Injury

Complete lesion above the sacral spinal cord results in

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detrusor overactivity, smooth sphincter synergia (with lesions below the sympathetic outflow) and striated sphincter dyssynergia. There is spasticity of skeletal muscle distal to the lesion, hyperreflexic deep tendon reflexes, and abnormal plantar responses with impairment of deep and superficial sensation. The striated sphincter dyssynergia causes a functional obstruction with poor emptying and high detrusor pressure. In lesions close to the conus medullaris, incomplete bladder emptying may occur due to poorly sustained or absent detrusor contraction. Once reflex voiding is established, it can be reinforced by stimulation of certain dermatomes, as by tapping the suprapubic area.

Sacral Spinal Cord Injury

After recovery from spinal shock, there is generally a depression of deep tendon reflexes below the level of the lesion with varying degree of flaccid paralysis. Sensation is generally absent below the level of lesion. Detrusor areflexia with high or normal compliance is common initially but decreased compliance may develop in some cases. Classic outlet findings are competent but nonrelaxing smooth sphincter that retains some fixed tone but is not under voluntary control. Closure pressures are decreased in both areas. Attempted voiding by straining or Crede's maneuver results in obstruction at the bladder neck or at the distal sphincter area by fixed sphincter tone.

Investigations

Baseline testing includes urinalysis with microscopy and urine culture, serum creatinine and a renal ultrasound to evaluate the upper urinary tract. Other studies can be performed as indicated or during long-term follow-up and include a voiding cystogram which assesses for vesicoureteral reflux, bladder hypertrophy and bladder diverticula, and a dimercaptosuccinic acid renogram which provides functional evaluation of kidney. Cystoscopy in evaluation of hematuria and a CT scan without contrast is most sensitive for evaluation of stones.

Urodynamic studies most definitive modality to assess dysfunction associated with neurogenic bladder and guides in appropriate bladder management. It is conducted once the patient is stable, out of spinal shock, and performing intermittent catheterization[4].

BLADDER MANAGEMENT

Acute management: During spinal shock an indwelling Foley catheter helps to monitor urine output and is usually kept in place until patients are medically stable. CIC can then be initiated, which is performed every 4 hours to maintain volumes at generally less than 500 ml. Clean intermittent self catheterization should be taught as early as possible to patients who have sufficient hand function. Baseline blood urea nitrogen, creatinine, urinalysis and urine culture are obtained. Urodynamic studies are done, generally at 6 weeks or as soon as urinary incontinence occur.

Long term management: The choice of long term bladder management depends on many factors, including the level and completeness of injury, amount of hand function, sex and motivation. Intermittent catheterization is now accepted as the best and safest method of long term management[5]. Chronic indwelling catheters are associated with high rates of chronic urinary infections, urethritis, prostatitis, bladder stones, bladder diverticulae, strictures, abscesses, bladder cancer and upper urinary tract infections[6]. Patients who have incontinence in between catheterizations as a result of hyperreflexic bladder, anticholinergics can be used.

Crede and valsalva maneuvers: Injuries at level of conus or below result in areflexic bladder with impaired detrusor contractions, large bladder capacity, and high residual urine. Bladder contraction and emptying can be facilitated with Valsalva maneuver, which increases intra-abdominal pressure, or the Crede maneuver, which applies direct pressure to the suprapubic area.

Reflex voiding

In supra sacral lesions, the sacral reflex arc is generally intact with a resultant reflexic bladder. Suprapubic tapping leads to bladder contraction and opening of bladder neck. Supra sacral lesions are often associated with DSD, elevated voiding pressures may lead to deleterious effects on the upper urinary tract. Transurethral sphincterotomy is often required to allow bladder drainage at low pressure.

PHARMACOLOGICAL MANAGEMENT

Systemic medication: Pharmacologic agents are used to inhibit involuntary detrusor activity and, as a result, increase bladder capacity and reduce intravesical pressures. Pharmacologic agents currently used are anticholinergics (e.g. oxybutynin, tolterodine), tricyclic antidepressants (e.g. imipramine) and antispasmodic drugs (e.g. baclofen, tizanidine). The use of anticholinergic medication may be associated with

significant undesirable side effects, such as dry mouth and impaired gastrointestinal secretion and motility.

Intrvesical therapy: Botulinum toxin has high affinity for cholinergic peripheral nerve ending and inhibits acetylcholine release at neuromuscular junctions, resulting in prolonged local muscular weakness and paralysis when injected directly into the muscle[7]. For detrusor overactivity, 200 to 300 units of toxin is injected in the detrusor muscle at multiple sites. It supresses bladder overactivity, increases cystometric and maximum bladder capacity, decreases voiding pressure and eliminates urinary incontinence associated with detrusor overactivity[8]. Injection of botulinum toxin type A into the external urethral sphincter is also used to treat DSD and improves voiding[9]. The major drawback is the temporary effect, requiring repeat injections every 6 months.

SURGICAL MANAGEMENT

Electrical stimulation and posterior sacral root rhizotomy: In suprasacral lesions, electrical stimulation of sacral anterior root has been used to produce effective micturition with relatively low residual volumes. Electrodes are surgically implanted on sacral nerves with the stimulator placed under the skin, stimulation of S3 nerve root suppresses hyperreflexic detrusor activity. This combined with division of posterior sacral roots is used to eliminate detrusor and sphincter hyperreflexia, increase bladder capacity and compliance, and decrease incidence of reflex incontinence.

Augmentation cystoplasty, cutaneous conduits and urinary diversions: In patients refractory to conservative management, bladder augmentation is done to increase bladder capacity, increase detrusor compliance and decrease bladder storage pressures to prevent upper tracts deterioration[10]. Some patients who have difficulty with catheterization, have urethral or penile skin changes such as fistulae or strictures, or have incontinence that impairs management of decubitus ulcers. These may require diverting the urine using ileal conduit[11].

Transurethral sphincterotomy: Transurethral incision of the external urinary sphincter can be done in cases of bladder outlet obstruction secondary to DESD with associated high bladder pressures unresponsive to anticholinergics. The decrease in outlet resistance lowers the high voiding pressures associated with DESD.

COMPLICATIONS

The incidence of urologic complications associated with neurogenic bladder has decreased with use of CIC. However, those still exist are chronic urinary tract infections, bladder diverticulae, bladder stones, urethral trauma leading to fistulae or strictures, vesicoureteral reflux, hydronephrosis, pyelonephritis, renal failure and bladder cancer.

Follow-Up

Annual follow-up is recommended for the first 5 to 10 years after injury. If the patient is doing well, then follow-up every other year is advised. Upper and lower tact evaluation with urodynamic evaluation should be done initially and yearly for 5 to 10 years, and then every other year. Cystoscopy is recommended annually in those with an indwelling catheter.

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