W13: Neuro-Urology

Workshop Chairs: Helmut Madersbacher, Austria and Rizwan Hamid, United Kingdom
06 October 2015 14:00 - 18:00

Start	End	Topic	Speakers
14:00	14:05	Introduction	Helmut Madersbacher Rizwan Hamid
14:05	14:25	Neurogenic lower urinary tract (LUT) dysfunction – from Neurophysiology via Pathophysiology to Clinics	John Heesakkers
14:25	14:30	Discussion	All
14:30	14:50	Diagnostic workup: How informative are basic diagnostics, are urodynamics essential?	Marcio Averbeck
14:50	14:55	Discussion	All
14:55	15:15	How to maintain normal renal function, how to achieve continence / how to manage incontinence with overactive detrusor - Therapeutic strategies in 2015	Thomas Kessler
15:15	15:20	Discussion	All
15:20	15:40	How to maintain normal renal function, how to achieve continence / how to manage incontinence with underactive (acontractile) detrusor - Therapeutic strategies in 2015	Helmut Madersbacher
15:40	15:45	Discussion	All
15:45	16:00	Break	None
16:00	16:20	Spinal cord injury-upper motor neuron type-case based approach	Giulio Del Popolo
16:20	16:25	Discussion	All
16:25	16:45	Spinal cord injury- lower motor neuron type-case based approach	Thomas Kessler
16:45	16:50	Discussion	All
16:50	17:10	Multiple sclerosis - case based approach	Rizwan Hamid
17:10	17:15	Discussion	All
17:15	17:35	Spina bifida – case based approach	Karl Sievert
17:35	18:00	Final Discussions	All

Aims of course/workshop

In the first part, information on neurophysiology of the lower urinary tract and on pathophysiology related to cerebral, spinal and peripheral lesions, and how they are reflected in clinical symptoms, are provided. Thereafter a standard diagnostic approach will be presented, and the value of the classic urodynamic techniques discussed. In the second part, an overview on the therapeutic strategies in 2014 in patients (1) with an overactive detrusor and (2) with an underactive detrusor, each combined either with an underactive sphincter, an overactive sphincter or a normal sphincter will be given.

Learning Objectives

1. Understand the underlying pathophysiology of lower urinary tract dysfunction according to the level and extent of the lesion within the CNS and the peripheral nervous system.

2. Do the basic diagnostic workup and to know when urodynamics are necessary and urodynamics essential.

3. Choose amongst the available therapeutic options for different patterns of neurogenic lower urinary tract dysfunction.

4. Investigate and formulate a urological management plan for specific neurological conditions

5. Identify and manage urological complications

6. Manage the urological symptoms in context of the neurological disease

Neurogenic Lower Urinary Tract Dysfunction
– from Neurophysiology via Pathophysiology to Clinics

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INTRODUCTION

The lower urinary tract (LUT), the urinary bladder and urethra, serves two reciprocal functions: storage of urine without leakage and periodic evacuation of urine. These two functions depend on central as well as peripheral autonomic and somatic neural pathways,[1, 2] but also on voluntary control which requires the participation of higher centers in the brain. Because of the complex neural regulations, the central and peripheral nervous control of the LUT is prone to a variety of neurological disorders. In this lecture the principals of neuronal control of the LUT function will be summarized as well as the pathophysiology of neurogenic LUT dysfunction in specific lesions.

NEUROPHYSIOLOGY OF THE LOWER URINARY TRACT

Normal LUT function

The LUT function can be divided in filling and voiding functions. Normal filling function includes 1) sensation bladder fullness, 2) postpone urination, 3) maintaining continence and 4) low bladder pressure. Normal voiding function means to void voluntarily and smoothly without straining or post-void residual.

Reflex pathways controlling urine filling and voiding

Coordinated activities of the peripheral nervous system innervating the LUT during urine storage and voiding depend on multiple reflex pathways organized in the brain and spinal cord. The central pathways regulating LUT function are organized as on-off switching circuits that maintain a reciprocal relationship between bladder and its outlet.[1-3]

The filling phase

The accommodation of the bladder to increasing volumes of urine is primarily a passive phenomenon dependent on the intrinsic properties of the detrusor.[1-4] In addition to this passive accommodation mechanism, the afferent activity induced by bladder distention can trigger reflex activation of the sympathetic outflow to the

LUT. It contributes to the storage mechanism that inhibits the parasympathetic efferent outflow to the bladder and promotes closure of the urethra through activation of ⍺1A-adrenoceptors. Furthermore it facilitates relaxation of the detrusor via activation of ⍺⍺-adrenoceptors[4] (**Figure 1A**). This reflex is organized in the lumbosacral spinal cord. During bladder filling, the bladder afferent input also activates the pudendal motoneurons innervating the external urethral sphincter (EUS) [3, 5] and increases EUS activity. The EUS motor neurons are also activated by urethral/perineal afferents in the pudendal nerve[6]. These excitatory sphincter reflexes are organized in the spinal cord. Moreover, a supraspinal urine storage center located in the dorsolateral pons sends descending excitatory inputs to the EUS motoneurons to increase urethral resistance[7,8] (**Figure 1A**).

The voiding phase

The filling phase can be switched to the voiding phase either involuntarily or voluntarily. When bladder volume reaches the micturition threshold, bladder afferents in the pelvic nerves trigger the micturition by acting on neurons in the sacral spinal cord, which then send their axons rostrally to the periaqueductal gray (PAG), which in turn communicates with the pontine micturition center (PMC)[9, 10]. Activation of the PMC reverses the pattern of efferent outflow to the LUT, inhibiting sympathetic and somatic pathways and activating parasympathetic pathways. The voiding phase consists of an initial relaxation of the urethral sphincter followed by a contraction of the bladder. Thus voiding reflexes depend on a spinobulbospinal pathway (**Figure 1B**). Secondary reflexes elicited by urine flow into the urethra also facilitate bladder emptying.[1, 3] Inhibition of EUS activity during voiding depends, at least in part, on supraspinal mechanisms.

Peripheral and spinal pathways

The peripheral nervous mechanisms for bladder emptying and urine storage involve efferent and afferent signaling in pelvic (parasympathetic), hypogastric (sympathetic) and pudendal (somatic) nerves.[1, 2] (**Figure 2**).

Efferent pathways of the LUT

1. Parasympathetic pathways

Pelvic parasympathetic nerves, which arise at the sacral spinal cord (S_2-S_4), provide an excitatory input to the bladder and an inhibitory input to the bladder neck and urethral smooth muscle to eliminate urine [1, 2, 11] (**Figure 2**). Parasympathetic preganglionic neurons send axons to peripheral ganglia. Parasympathetic postganglionic neurons in humans are located in the bladder wall.[3] Parasympathetic postganglionic nerve terminals release Acetylcholine (ACh), which can excite muscarinic receptors in the detrusor smooth muscle cells, leading to bladder contractions.[12,13] The postganglionic parasympathetic input to the urethra elicits inhibitory effects mediated at least in part through the release of nitric oxide (NO), which directly relaxes the urethral smooth muscle.[3, 14, 15] Therefore, the excitation of

sacral parasympathetic efferent pathways induces a bladder contraction and urethral relaxation to promote bladder emptying during micturition.

2. Sympathetic pathways

Hypogastric sympathetic nerves, which arise at the thoraco-lumbar level of the spinal cord (Th_{11}-L_2), provide a noradrenergic inhibitory input to the bladder and excitatory input to the urethra to facilitate urine storage[3] (**Figure 2**). The sympathetic activation causes inhibition of the parasympathetic pathways at spinal and ganglionic levels, and releases noradrenaline (NA) from its postganglionic terminals, which elicit relaxation the bladder body and contractions of the bladder neck and urethral smooth muscle[3].

3. Somatic pathways

Somatic efferent motoneurons which activate the external striated urethral sphincter muscle and the pelvic floor muscle are located along the lateral border of the ventral horn in the sacral spinal cord (S_2-S_4), commonly referred to as the Onuf's nucleus [16] (**Figure 2**). The somatic motoneurons send axons into the pudendal nerves. Combined activation of sympathetic and somatic pathways increases bladder outlet resistance and realises urinary continence.

Afferent pathways of the LUT

The pelvic, hypogastric, and pudendal nerves also contain afferent axons that transmit information from the LUT to the spinal cord (**Figure 2**). [1, 2, 17, 18] The pelvic afferent nerves, which monitor the volume of the bladder and intravesical pressure, consist of small myelinated Aδ and unmyelinated C fibers. Normal micturition reflex is mediated by myelinated Aδ fibers, which respond to bladder distention and active contraction[3, 18, 19] (**Figure 3**). The activation threshold for Aδ fibers is 5-15 cmH_2O[1]. C-fiber afferents have a high mechanical threshold and are usually unresponsive to mechanical stimuli such as bladder distention[19] and therefore have been termed as "silent C fibers". However, these fibers respond primarily to chemical, noxious, or cold stimuli. During inflammation or neuropathic conditions, there is recruitment of C-fiber bladder afferents, which form a new afferent pathway that can cause detrusor overactivity and bladder pain[20] (**Figure 3**).

Brainstem (Pons)

The dorsal pontine tegmentum has been established as an essential control center for micturition and called as the "pontine micturition center (PMC)" or the "M region" due to its median location.[10, 21, 22] Brain imaging studies have revealed increase in blood flow in this region of the pons during micturition.[23] Neurons in the PMC provide direct synaptic inputs to sacral PGN, as well as to GABA-nergic neurons in the sacral dorsal commissure (DCM).[21] The former neurons carry the excitatory outflow to the bladder, whereas the latter neurons are thought to be important in mediating an inhibitory outflow on EUS motoneurons during micturition[24]. As a result of these reciprocal connections, the PMC can promote coordination between

the bladder and urethral sphincter. In the cat, another area, located in the ventrolateral pontine tegmentum and is called the "L-region", which controls the motoneurons of the pelvic floor, including the external urethral sphincter.[25] This region might be considered as the pontine storage center **(Figure 1A)**. In humans the laterally located L-region is especially active in volunteers who tried but did not succeed to micturate.[25, 26]

Suprapontine pathways

Studies in humans indicate that voluntary control of urine storage and voiding depends on connections between the frontal cortex and the septal/preoptic region of the hypothalamus as well as connections the paracentral lobule and the brainstem.[1, 3] Lesions to these areas of cortex exhibit detrusor overactivity (DO) because of lack of cortical inhibitory control. Brain imaging studies in right-handed both male and female volunteers have demonstrated decreased blood flow in the right anterior cingulate gyrus during urine withholding prior micturition and increased blood flow in the right dorsomedial pontine tegmentum, in the PAG, in the hypothalamus and in the right inferior frontal gyrus during voiding.[23, 26, 27] It has been implicated that the midbrain PAG receives bladder filling information, and the hypothalamus has a role in the beginning of micturition.[28] Furthermore, the human cingulate and prefrontal cortices are activated during both micturition and continence, indicating that these areas are important for the onset of micturition, but not for the reflex itself.

PATHOPHYSIOLOGY OF THE LOWER URINARY TRACT DYSFUNCTION

When a neurological lesion is present, the type of LUT dysfunction roughly depends on the site and the extent of the lesion[29].

Neurological pathology can be divided in 1) suprapontine, 2) brainstem (pontine), 3) suprasacral spinal cord, and 4) sacral and subsacral (cauda equine and peripheral nerve) lesions (**Figure 4**). Madersbacher et al described the common patterns of neurogenic detrusor-sphincter dysfunction in a diagram associated with these lesions (**Figure 5**).[30]

Suprapontine lesions

Patients with lesions above the pons commonly demonstrate DO caused by lack of cortical inhibitory control, but they preserve coordinated synergic sphincter function (**Figure 5-A**). However, these patients sometimes may purposely increase sphincter activity during an overactive detrusor contraction to avoid urgency incontinence[31]. Typical suprapontine lesions include cerebro-vascular accident (CVA), dementia, brain tumors, and cerebral palsy.

Cerebro-vascular accident (CVA)

The most common LUT symptoms after stroke are nocturia (36%), urge incontinence (29%) and difficulty in voiding (25%) [32]. There is a positive correlation between the occurrence of LUT dysfunction and hemiparesis[32]. Urodynamic studies revealed DO

in 68%, uninhibited sphincter relaxation in 36%[32]. In patients with brainstem strokes, voiding symptoms are more predominant[33].

Brainstem (pontine) lesions

As the pontine micturition center (PMC) and the lateral storage centre are located in the brain stem, lesions of this area demonstrate a variety of LUT dysfunction. Both filling and voiding dysfunctions often occur simultaneously. The common diseases of this region include Parkinson's disease, multiple system atrophy (MSA), and multiple sclerosis.

Parkinson's disease

LUT dysfunction occurs in 37-71% of patients[39, 40]. Storage symptoms were observed in 28%, voiding symptoms in 11%, and both symptoms in 21% [40]. The frequency of LUT symptoms correlated with the severity of the disease, but not with the duration of the illness or sex[40]. DO was observed in 81%, external sphincter relaxation failure in 33%, and underactive detrusor in 66% of women and 40% of men[41].

Multiple system atrophy (MSA)

MSA involves various syndromes resulted from degeneration of neurons and associated fibers of motor and extrapyramidal systems including the cerebellum and brain stem. LUT symptoms often precede orthostatic hypotension and other autonomic nervous system symptoms in MSA patients[42]. Almost 100% of MSA patients have some kind of LUT symptoms[43, 44]. The most common urodynamic abnormalities are abnormal sphincter EMG (in 82-91%)[42, 44, 45], DO (in 56%)[44], DSD (in 45-47%)[44, 46, 47], uninhibited external sphincter relaxation (in 33%)[44], and underactive detrusor (in 71% of women and 63% of men)[44]. Significant PVR is observed in about half of MSA patients[48].

Suprasacral spinal cord lesions

Spinal lesions mostly cause simultaneous dysfunction of the detrusor and the sphincter. In suprasacral spinal cord lesions a typical pattern of the LUT dysfunction is DO associated with DSD (**Figure 5-B**). If the lesion is complete, sensation of bladder filling disappears. Basically spontaneous reflex voiding is possible, however, it is uncontrolled, causing reflex incontinence, and unphysiological. DSD leads to unbalanced voiding with the possibility of a dangerous high pressure situation. While most traumatic spinal cord lesions give LUT dysfunction which can be predicted fairly well from the level and completeness of injury, the LUT dysfunction in many other neurological disorders such as myelomeningocele are more difficult to categorise[29] (**Figure 5-B, C , E &F**).

Spinal cord injury (SCI)

DO was demonstrated in 95%[49], and DSD in 68%[50] of the patients with suprasacral lesions. In patients with sacral lesion, an acontractile detrusor was found in 86%, and

low compliance in 79%[49]. Upper urinary tract changes were observed in 30-42% of SCI patients[51, 52].

Multiple sclerosis (MS)
The LUT dysfunction is mainly due to spinal lesions, although brain lesions may contribute[30]. Impairment of neurological function results from demyelinating plaques of the white matter of the brain and spinal cord, especially the posterior and lateral columns of the cervical cord. The prevalence of LUT dysfunction in MS patients is 33-52%, and its incidence is related to the disability status[53]. Urodynamic studies revealed DO in 44-99%, DSD in 6-66%, and detrusor underactivity in 31-38% [54-56]. The upper urinary tract is rarely involved[57].

Sacral and subsacral (cauda equina and peripheral nerves) lesions
Lesions of conus causing dysfunction of the sacral parasympathetic neurons and the EUS motoneurons are categorized as the same group as subsacral (cauda equina and peripheral nerves) lesions. For complete sacral or subsacral lesions, classically named as lower motor neuron lesions, loss of bladder sensation and acontractile detrusor with incompetent urethra are characteristic (**Figure 5-E**). However, acontractile detrusor combined with normal or overactive urethra may occur in lesions, comprising the conus and nearby area above (**Figure 5-D & F)**. In lumbo-sacral lesions, espetially in myelomeningocele, overative detrusor combined with incompetent urethra sometimes occur (**Figure 5-C)**. Subsacral (cauda equina and peripheral nerves) lesions are often imcomplete and associated with a variety of combination patterns of the detrusor and urethral dysfunctions. For example, in case of pelvic nerve plexus injury after radical pelvic organ surgery, if the pudendal nerves remain intact, impaired bladder sensation and underactive/acontractile detrosor may be combined normally functioning urethra (**Figure 5-D)**. On the other hand, the pudendal nerves are selectively disrupted, for instance, in traumatic pelvic fracture, incompetent urethra may occur with normal detrusor function (**Figure 5-H)**

Myelodysplasia (Myelomeningocele MMC) and spina bifida
The incidence of LUT dysfunction is very high (>90%) in MMC patients[29]. Most have LUT dysfunction leading to incontinence and/or upper tract deterioration.

Diabetes Mellitus
Ther are no proper epidemiological studies on the incidence of diabetes-related LUT dysfunction. Neurogenic LUT dysfunction occurs in 43-87% of insulin-dependent DM patients with no sex or age differences[58]. LUT dysfunction is characterized as reduced bladder sensation and impaired empting function due to detrusor underactivity[59-61].

Peripheral neuropathy after pelvic surgery

No good epidemiological studies on the incidence of LUT dysfunction related with peripheral nerve injury in pelvic surgery were performed. Neurogenic LUT dysfunction occurs in 30-50% of patients after extensive pelvic surgery. LUT dysfunction is characterized as reduced bladder sensation, low compliant detrusor and impaired empting function due to detrusor underactivity[29].

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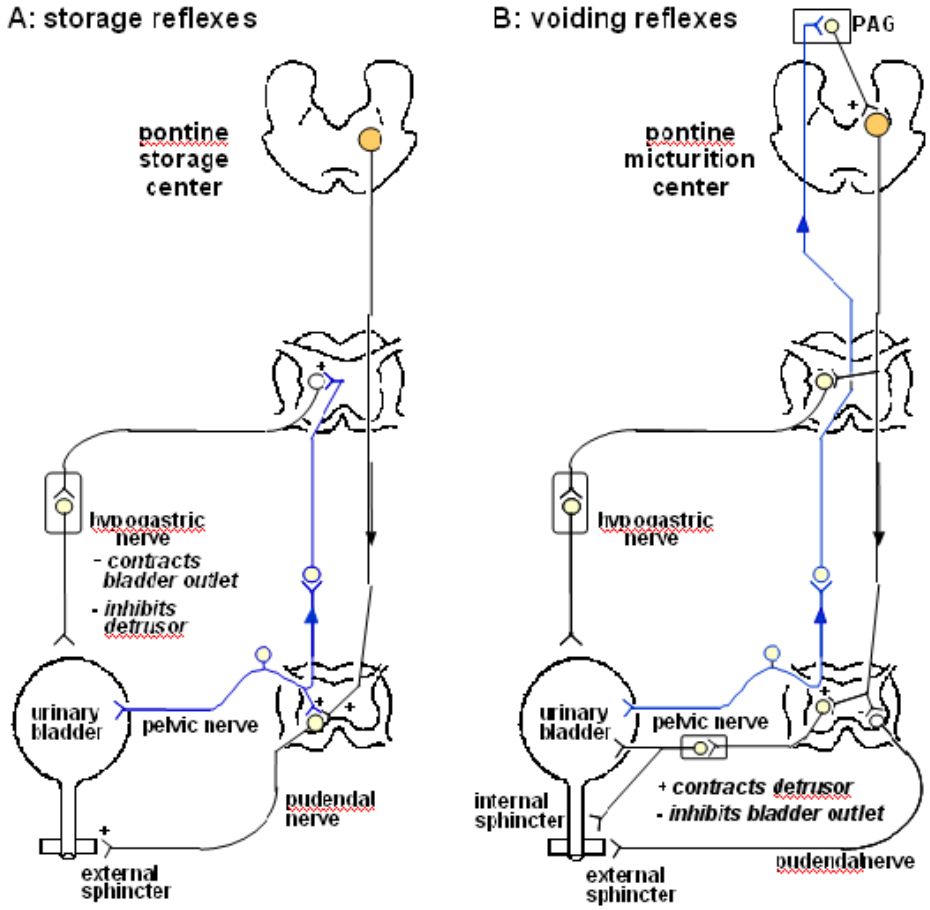


Figure 1. Neural circuits controlling continence and micturition. A: Storage reflexes, B: Voiding reflexes (Modified from Yoshimura & de Groat[2])

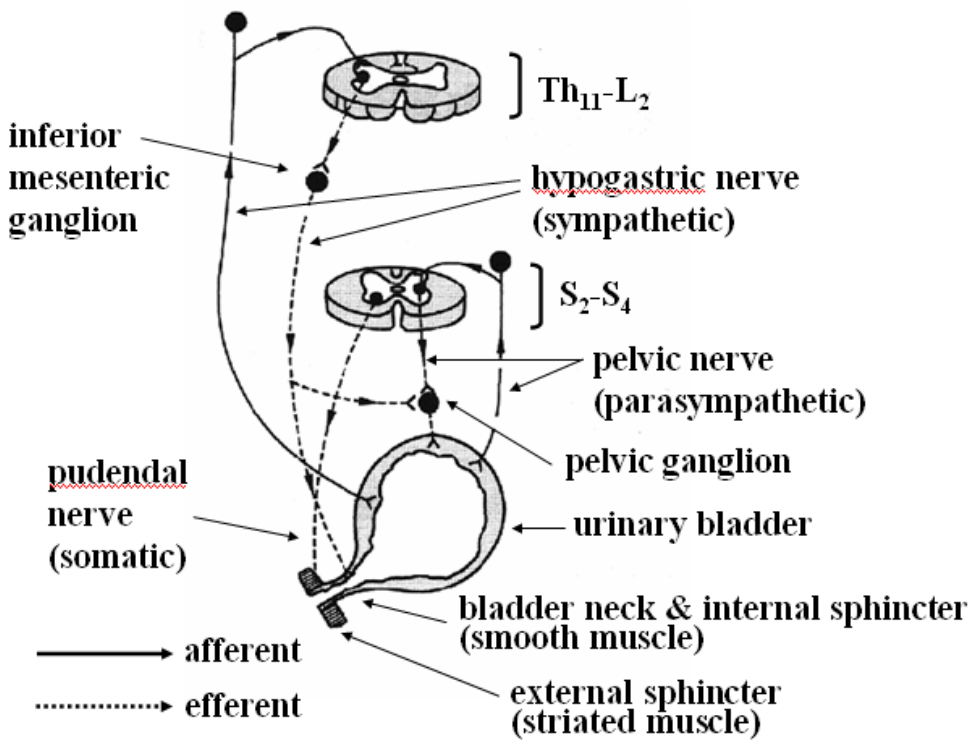


Figure 2. Sympathetic, parasympathetic, and somatic innervation of the lower urinary tract. (Reproduced from Yoshimura & de Groat[2])

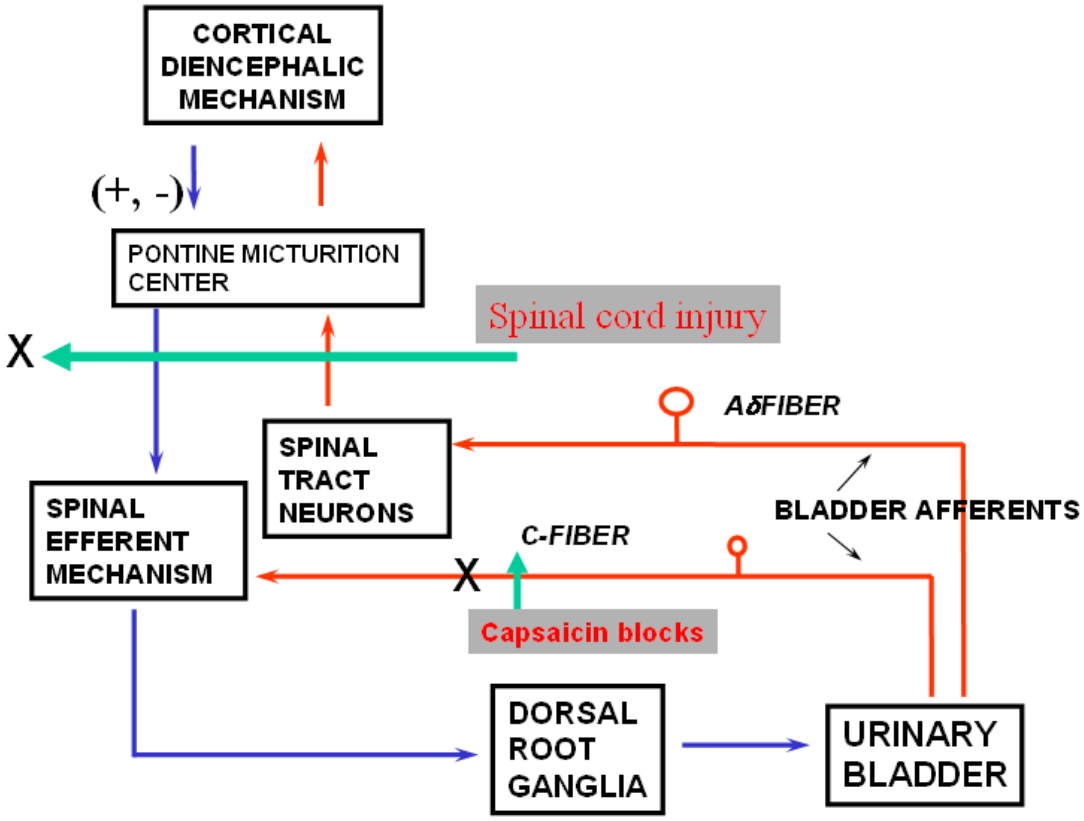


Figure 3. The central reflex pathways that regulate micturition in normal and spinal-cord-injured cats (Modified from Yoshimura et al[3])

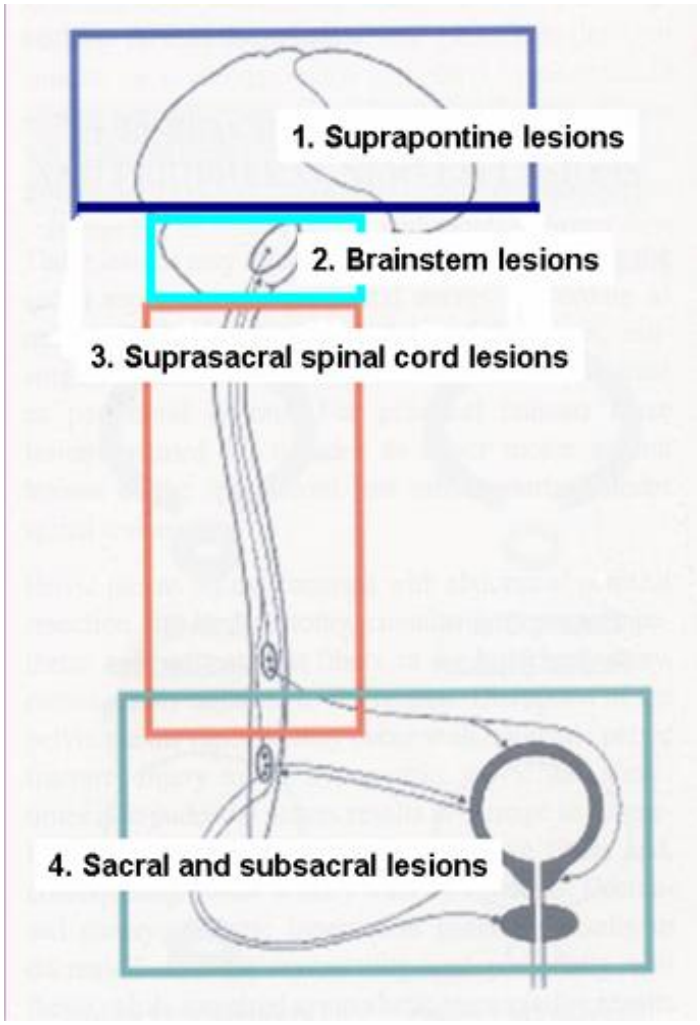


Figure 4. Classification of neurological lesions
(Modified from Madersbacher et al[30])

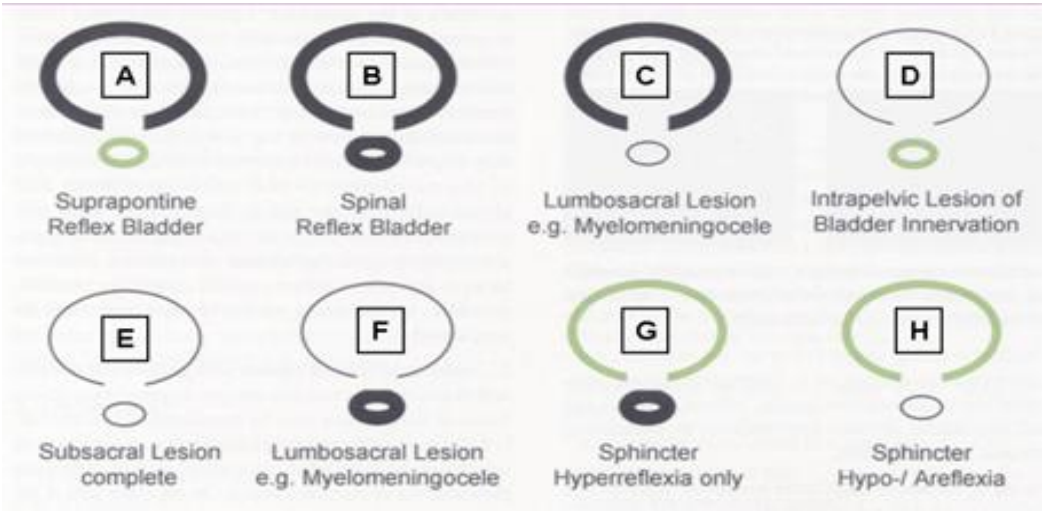


Figure 5. Madersbacher classification system with typical neurogenic lesions (Modified from Madersbacher et al[30])
Heavy lines symbolize overactivity, thin lines underactive or acontractile and green lines normal function of the relevant structure

ICS ANNUAL MEETING – MONTREAL 2015

NEURO-UROLOGY WORKSHOP

Lecture:

Diagnostic workup - How informative are basic diagnostics, are urodynamics essential?

Speaker:
Márcio Augusto Averbeck <marcioaverbeck@gmail.com>, Brazil
Prof. MD, Head of Dept. of Female Urology of the Brazilian Society of Urology (SBU)

ICS Neurourology Promotion Committee Member

Department of Urology – Mãe de Deus Center Hospital, Porto Alegre/Brazil

VideoUrodynamics Unit – Moinhos de Vento Hospital, Porto Alegre/Brazil

Prof. M.A. Averbeck (Brazil) will inform you about the diagnostic workup:

Introduction

The lower urinary tract (LUT) has its function related to the storage of urine at low pressure and the normal voiding process, which depends on the effective contraction of the detrusor and synergic relaxation of the urethral sphincter. This activity is regulated by a neural control system in the brain and spinal cord that coordinates the urinary bladder and bladder outlet (1). Neurological diseases, e.g. spinal cord lesions, multiple sclerosis, Parkinson disease, etc, can lead to lower urinary tract dysfunction and its consequences (urinary tract infections, urinary incontinence, stone formation, renal failure, etc).

One interesting point to observe is that lower urinary tract symptoms (LUTS) and long-term complications often do not correlate (2). One of the most fearful complications is renal damage secondary to elevated storage pressure in the bladder (3). In this setting, **the diagnostic workup is important to establish whether the patient with neurological disease has a low or high risk of subsequent complications** (1-4). Suprasacral infrapontine spinal lesions can often cause sustained elevated storage pressure in the bladder, due to a combination of detrusor overactivity and detrusor-sphincter dyssynergia. Fortunately, in all other patients with neurogenic lower urinary tract dysfunction (NLUTD), the risk of renal damage is significantly lower (1). Nowadays, with improved diagnostic workup and development of new treatment strategies, e.g. antimuscarinics, botulinum toxin A, bladder

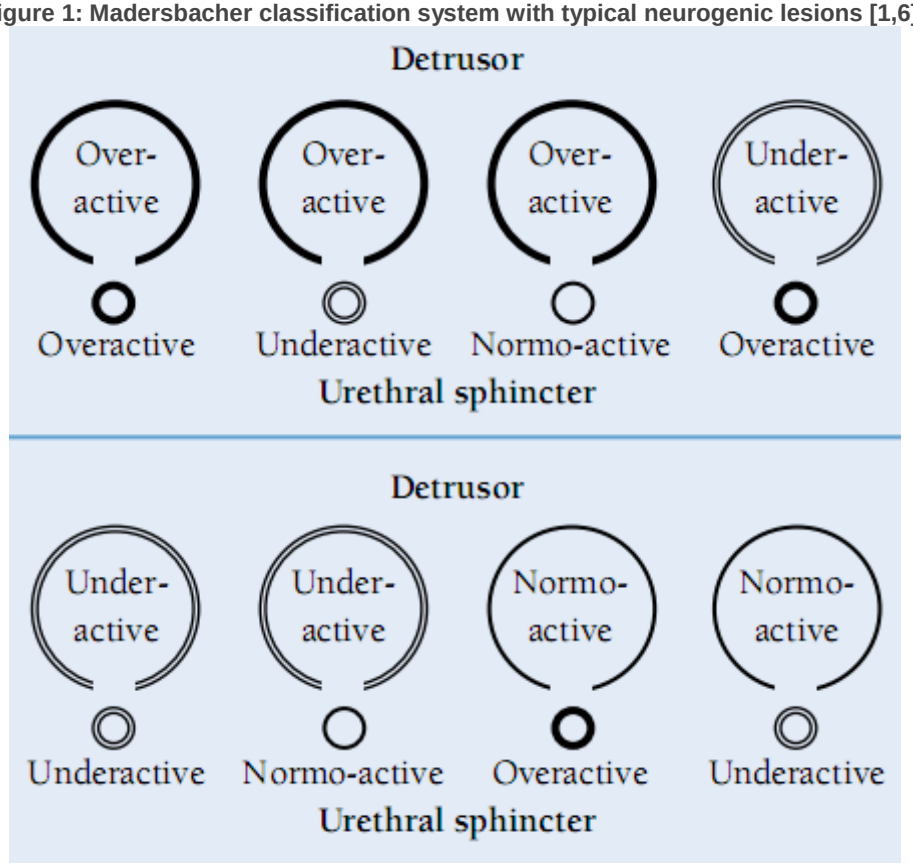
augmentation techniques and intermittent self-catheterization, respiratory diseases became the most frequent (21%) cause of death in patients with spinal cord injury (SCI) (5).

As urodynamics and clinical symptoms often do not correlate, asymptomatic patients can present with abnormal urodynamic findings being so far unrecognised risk factors (6). Changes in urodynamics and subsequent damage to the LUT may antedate clinical symptoms. Therefore, comprehensive urological assessment is essential in patients with NLUTD. Treatment and intensity of follow-up examinations are based on the type of NLUTD (1).

A thorough medical history, including voiding diary, and targeted physical examination is mandatory, before any additional diagnostic investigations are planned. Early diagnosis and treatment are important to avoid irreversible changes within the LUT, both in congenital and acquired neurological diseases (1).

The diagnostic workup must be aimed to accurately evaluate the status of the detrusor and the sphincter. In these regards, Madersbacher (6) proposed a simple classification focused on therapeutic consequences. This classification describes several NLUTD on the basis of the activity/contractility of the detrusor muscle and external urethral sphincter during voiding and filling phase (Figure 1).

Figure 1: Madersbacher classification system with typical neurogenic lesions [1,6]*



*Adapted from the Guidelines on Neurogenic Lower Urinary Tract Dysfunction, European Association of Urology 2015

Patient History

History talking will guide the physical examination and helps to understand the findings of further investigations, such as urodynamics. A comprehensive history must be focused on both storage and voiding phases.

Present history includes medication currently taken and specific urinary history.
Specific urinary history must address bladder sensation (sensation of bladder fullness, sensation for the urge to void, urgency, urgency incontinence), initiation of micturition (normal, precipitate, reflex, strain, Credé), interruption of micturition (normal, paradoxical, passive), enuresis, mode and type of bladder emptying (catheterization). A voiding diary must always be done, as it provides objective information about day- and night-time voiding frequency, volume voided, incontinence and urge episodes. It is also useful in patients performing intermittent catheterization (1).

Bowel habits must also be addressed since urinary bladder and rectum share a common embryological origin. Besides, their autonomic and somatic innervations have close similarities and the close neighborhood of these two organ systems let assume that dysfunction in one may influence, also mechanically, the function of the other (7).

Other aspect that should be remembered is the sexual function, as neurogenic diseases may cause impairment of both LUT function and sexuality (1).

There are some symptoms and signs that cause great concern: pain, haematuria, dysuria and fever can mean urinary tract infection and must be promptly addressed.

In regards to the past history, it is important to look for hereditary or familial risk factors, obstetric history, accidents and operations, especially those involving the central nervous system incl. spinal cord and spine as well as surgery on organs in the small pelvis (1). Diseases such as diabetes, syphilis, Parkinsonism, multiple sclerosis and encephalitis must always be investigated.

Validated questionnaires should be used to evaluate quality of life (QoL) and the effects of LUTS on daily activities.

Physical Examination

The motor nerve supply of the LUT is provided by the parasympathetic outflow arising from S2–S4. The external (striated) urethral sphincter is innervated by the pudendal nerve, arising from the Onuf's nucleous in S2-S4.

The physical examination is essential for the reliable interpretation of subsequent urodynamics and must be described comprehensively. All sensations and reflexes in the urogenital area, along with careful testing of the anal sphincter and pelvic floor muscles must be carried out.

Sensations on the sacral dermatomes (S2-S5) must be recorded and specified according to the presence (increased/normal/reduced/absent) and type (sharp/blunt) (8). The study of reflexes

(increased/normal/reduced/absent) such as Bulbocavernous reflex, Perianal reflex, Cremasteric reflex and Plantar responses (Babinski) should always be carried out.

Digital rectal examination in male patients can not only provide reliable information on prostate volume and presence of nodules and hardening (one must remember that bladder outlet obstruction and LUTS due to prostate enlargement may occur concomitantly with neurological diseases), but also information on the anal sphincter tone and the ability to contract the anal sphincter voluntarily, both in men and in women.

Careful inspection of genitalia also plays an important role in women, as pelvic organ prolapse might be associated with LUTS incl. post-voiding residual urine.

Urodynamics

Urodynamic study is intended to assess the function (or dysfunction) of the lower urinary tract. It has a great importance in the evaluation of patients with neurogenic lower urinary tract dysfunction, especially to detect risk factors for the upper urinary tract in time, before they cause irreversible changes.

In patients with spinal cord injuries above Th 5 – Th 6, it is important to measure blood pressure during the bladder filling, due to the risk of autonomic dysreflexia (AD). It is a sudden and exaggerated autonomic response, which can cause life-threatening blood hypertension if not properly managed (9).

Preparation

It is recommended that the patient empties the rectum in the usually way the evening before the investigation (avoid enemas shortly before the investigation).

Drugs that influence the LUT function should be stopped at least 48 hours before (if feasible) or otherwise be considered when interpreting the data obtained.

Technique

All urodynamic findings must be reported in detail and performed according to the ICS "Good urodynamic practices" (10).

A 2-day-bladder diary is recommended before the urodynamics (8).

Urodynamics comprise several steps, as free uroflowmetry (with assessment of residual urine), filling cystometry, flow pressure study, and electromyography (EMG). Each of the listed procedures has some interesting points for discussion (see below). Video-urodynamics can add benefits to the evaluation of neurogenic patients.

- **Free uroflowmetry and assessment of residual urine**

This is an initial step, which gives a first impression of the voiding function. Free uroflowmetry objectively demonstrates the pattern of urine flow (low, intermittent, normal) and should be repeated 2-3 times before invasive urodynamics (bladder catheterization). Unfortunately, in some circumstances (detrusor underactivity, detrusor-sphincter dyssynergia, etc) the free

uroflowmetry cannot be done in the setting of neurogenic lower urinary tract dysfunction, as the patient is not able to void. Whenever it is feasible, the free uroflowmetry should be done. Voiding volumes superior to 100-150 ml are often taken as representative for evaluation.

- **Filling cystometry**

This step is either a single investigation (e.g. to control bladder compliance) or, more often, combined with flow pressure studies and video-urodynamics. Normally, the filling cystometry documents the status of the bladder during the filling phase and is performed with body-warm saline in a temperature-controlled room (1)

Through this phase it is possible to identify pathological signs, as detrusor overactivity, low detrusor compliance, abnormal bladder sensation and urinary incontinence.

Other important parameter that can be obtained from filling cystometry is the "detrusor leak point pressure" (DLPP). DLPP is defined as the lowest detrusor pressure at which urine leakage occurs in the absence of either a detrusor contraction or increased abdominal pressure, and may estimate the risk for renal failure.

- **Pressure flow study**

This step is performed after the filling cystometry and documents the LUT function during the voiding phase. Some pathological findings include detrusor underactivity, detrusor-sphincter dyssynergia, and residual urine.

- **Perineal electromyography (EMG)**

This is an additional tool to evaluate the activity of the pelvic floor muscles during the filling cystometry and pressure flow study, mostly superficial electrodes are used to register a random EMG of the pelvic floor musculature. The correct interpretation may be difficult due to artefacts introduced by other equipment used (1). Nevertheless, the EMG can bring valuable information for the diagnosis of detrusor-sphincter dyssynergia.

- **Video-urodynamics**

Video-urodynamics is the gold standard for urodynamic investigation in patients with neurogenic lower urinary tract dysfunction (1). This is a combination of filling cystometry and pressure flow study with imaging. Pathologic findings which can be shown by video-urodynamics include detrusor-sphincter dyssinergia (internal vs external sphincter), neurogenic configuration of the bladder (pseudodiverticulae, trabeculations), vesico-ureteral reflux (VUR), influx of contrast medium into the male adnexa, among others.

Provocative tests during urodynamics

Coughing, triggered voiding, or anal stretch are **provocative tests** to be used during the urodynamics in order to trigger detrusor contraction in the setting of NLUTD.

The **"ice water test"** consists of fast-filling cystometry with cooled saline to distinguish between upper motor neuron lesion and lower motor neuron lesion. Patients with upper motor neuron lesion will develop most probably a reflex detrusor contraction, even when not yet present with normal cystometry (11).

Specific uro-neurophysiological tests

Below are listed some specific tests, performed as part of the neurological work-up (1) in specialised centers:

• EMG (in a neurophysiological setting) of pelvic floor muscles, urethral sphincter and/or anal sphincter;

• nerve conduction studies of pudendal nerve;

• reflex latency measurements of bulbocavernosus and anal reflex arcs;

• evoked responses from clitoris or glans penis;

• sensory testing on bladder and urethra.

SUMMARY

There are typical urodynamic manifestations of neurogenic lower urinary tract dysfunction (NLUTD). They can not be substituted by history, clinical examination or echography.

Non invasive urodynamics (bladder diary, free flow and PVR) have to be done before, together with history and clinical examination, as they are the basis for interpretation of the urodynamic curves.

Typical urodynamic findings in patients with NLUTD include:

- Changes in bladder sensitivity (hyposensitivity, hypersensitivity, vegetative sensation/dysaesthesia)
- Low compliance
- Detrusor overactivity (spontaneous or provoked)
- Detrusor underactivity or acontractility
- Detrusor-sphincter dyssynergia
- Sphincter underactivity (can be suspected through EMG findings and incontinence during filling cystometry)

These signs warrant further neurological evaluation, as LUTD may be the presenting symptom of NLUTD (1).

Urodynamics are definitely an important diagnostic tool for patients with neurogenic LUT dysfunction. However, in contrast to spinal cord lesions, urodynamics may not be essential in some patients with cerebral diseases, especially when the upper urinary tract is normal and no significant post-void residual urine is present.

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THE MANAGEMENT OF THE OVERACTIVE DETRUSOR
+ normal sphincter
+ overactive sphincter
+ underactive sphincter

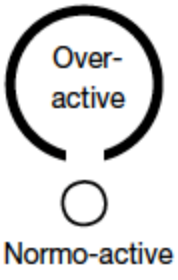
Thomas M. Kessler (Zürich, Switzerland)

According to the ICS terminology 2002, detrusor overactivity is a urodynamic observation characterized by involuntary detrusor contractions during the filling phase which may be spontaneous or provoked. There are 2 patterns of detrusor overactivity, i.e. (1) phasic detrusor overactivity defined by a characteristic wave form, and it may or may not lead to urinary incontinence, and (2) terminal detrusor overactivity defined as a single involuntary detrusor contraction occurring at cystometric capacity, which cannot be suppressed, and leads to incontinence usually resulting in bladder emptying (voiding).
There is no lower limit for the amplitude of an involuntary detrusor contraction but confident interpretation of low-pressure waves (amplitude smaller than 5 cmH_2O) depends oh high-quality urodynamic technique.

There are several reasons for an overactive detrusor: In the context of a neurological disorder, detrusor overactivity is usually observed in patients with a cerebral and/or suprasacral spinal lesion. The management of the overactive detrusor depends also on the function/dysfunction of the sphincter and whether the lesion is complete or incomplete.

1. MANAGEMENT OF THE OVERACTIVE DETRUSOR COMBINED WITH A NORMAL SPHINCTER

Patients with suprapontine cerebral lesions usually present with detrusor overactivity combined with a normal sphincter. Typically, there is a synergic function of the detrusor and sphincter, i.e. there is generally no relevant post void residual. Thus, the therapeutic concept is treat detrusor overactivity (see below). Only with pontine lesions detrusor and sphincter may either be overactive or underactive deserving special management.



2. MANAGEMENT OF THE OVERACTIVE DETRUSOR COMBINED WITH AN OVERACTIVE SPHINCTER

In patients with suprasacral spinal lesions, the bladder and the striated sphincter as well as the pelvic floor musculature are overactive. Detrusor sphincter dyssynergia is the main problem and puts at risk the upper urinary tract due to low compliance and/or high intravesical pressures during the storage and voiding phase. However, the overactive sphincter provides urinary continence once detrusor overactivity is under control and regular bladder emptying is achieved. Thus, the therapeutic concept is to treat detrusor overactivity and to assist or accomplish bladder emptying.

How to manage detrusor overactivity

- Behavioral treatment: includes lifestyle modifications, prompted voiding, timed voiding (bladder training), biofeedback for supporting the voiding pattern modification.

- Antimuscarinics: are the pharmacological first line treatment of detrusor overactivity. Eight antimuscarinics (darifenacin, fesoterodine, imidafenacin, oxybutynin, propiverine, solifenacin, tolterodine, trospium chloride) with different dosages, formulations, and routes of administration are currently used for treating detrusor overactivity. Although there is a huge number of randomized trials for patients without an underlying neurological disorder, high-level evidence is limited in neurogenic detrusor overactivity. There are differences in efficacy and especially adverse event profiles of the different antimuscarinics and an individualized pharmacological approach seems warranted but there are no clear-cut findings regarding the first choice antimuscarinic for treating neurogenic detrusor overactivity.

- Non invasive neuromodulation: Percutaneous tibial nerve stimulation, transcutaneous tibial nerve stimulation, transcutaneous electrical nerve stimulation of the pudendal nerve (clitoral/penile or intravaginal electrical stimulation) may be considered, especially in patients with MS or incomplete SCI but further high-evidence level studies are necessary.

- Intradetrusor injections of botulinum neurotoxin type A: are a generally accepted and widely used second-line treatment for neurogenic detrusor overactivity. Although several types of botulinum toxin are available, only onabotulinumtoxinA (Botox®) is currently FDA approved for treating neurogenic detrusor overactivity incontinence and a dose of 200 units Botox® is generally recommended.

- Sacral neuromodulation: is a well-established second-line therapy for non-neurogenic lower urinary tract dysfunction, i.e. non-obstructive chronic urinary retention, urgency-frequency syndrome, and urgency incontinence. There is evidence indicating that sacral neuromodulation may also be effective and safe for patients with neurogenic lower urinary tract dysfunction (including detrusor overactivity and detrusor sphincter dyssynergia) but the number of

investigated patients is low and there is a lack of randomized, controlled trials. In neurological patients, this treatment should only be offered within well-designed studies in specialized centers.

- Bladder augmentation: due to the afore-mentioned treatment options bladder augmentation is rarely necessary nowadays. Nevertheless, it is still an excellent option in some highly selected patients.

- Urinary diversion: In the case that all other treatments fail, i.e. as an “ultima ratio”, urinary diversion (usually combined with cystectomy) have to be considered (for instance, a patient with refractory urinary incontinence and anatomical low-compliance bladder). The type of urinary diversion (orthotopic bladder substitute (usually not indicated due to the overactive sphincter with consecutive post void residual), heterotopic bladder substitute (continent catheterizable reservoir), ileal conduit) depends on the course of the underlying neurological disorder and the patient’s manual dexterity.

How to empty the bladder

- Alpha-blockers: may be successful for decreasing bladder outlet resistance, post void residual, and autonomic dysreflexia. Alpha-blockers are worth trying out.

- Intermittent (self-)catheterization: see handout Prof. H. Madersbacher.

- Indwelling transurethral / suprapubic catheter: see handout Prof. H. Madersbacher.

- Sacral neuromodulation: see above.

- Intra-sphincter injections of botulinum neurotoxin type A: Although intra-detrusor injections for treating neurogenic detrusor overactivity is a highly recommended treatment, intra-sphincter injection is not due to limited effectiveness.

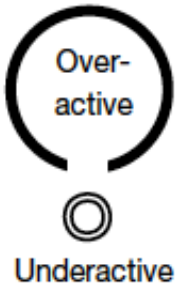
- Sphincterotomy: decreases the bladder outlet resistance but also increases the risk for urinary incontinence requiring management by external devices (condom catheters, pads, diapers). In addition, sphincterotomy needs to be repeated at regular intervals in many patients. Considering the other treatment options available, sphincterotomy becomes rarely necessary. However, it might be considered for men with cervical spinal cord injury and unbalanced reflex bladder.

- Intraurethral stents: Although the results are comparable with sphincterotomy, the costs, possible complications and re-interventions are limiting factors.

- Urinary diversion: see above.

3. MANAGEMENT OF THE OVERACTIVE DETRUSOR COMBINED WITH AN UNDERACTIVE SPHINCTER

Detrusor overactivity combined with an underactive sphincter results in detrusor overactivity incontinence combined with stress incontinence due to the de creased outlet resistance. The therapeutic concept is to treat detrusor overactivity (see above) and to increase the outlet resistance.



How to mange stress incontinence

- Pelvic floor muscle exercise: may be helpful in patients with incomplete lesions and can be combined with other treatment options (electrostimulation, duloxetine etc.).

- Electrostimulation: High evidence-level studies for neurological patients are lacking. Nevertheless, considering the favorable adverse event profile, electrostimulation may be tried out in patients with incomplete lesions.

- Duloxetine: Although there are no high-evidence level studies in neurological patients, duloxetine may be successful in mild stress incontinence.

- Bulking agents: are not recommended outside of well-designed clinical trials due to the lack of high evidence-level studies in neurological patients.

- Suburethral slings: are established in women, for men the artificial urinary sphincter is the first choice.

- Artificial urinary sphincter: gold standard in severe stress incontinence.

References related to the topic and worthwhile to read

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HOW TO MAINTAIN NORMAL RENAL FUNCTION, HOW TO ACHIEVE CONTINENCE, HOW TO MANAGE INCONTINENCE

THERAPEUTIC STRATEGIES in 2015

H. Madersbacher (Innsbruck, Austria)

The two aims in the management of patients with neurogenic lower urinary tract dysfunction (LUT-dysfunction) are (1) to protect the upper urinary tract to guarantee adequate (normal) life expectancy, and (2) achieve continence or to manage incontinence for improving their quality of life.

The most important objective is to achieve a safe situation urodynamically, which requires a bladder of sufficient capacity, filling at low pressure and emptying fully without hyperpressure or obstruction of the outlet. The indication for a therapeutic option must be based on the underlying pathophysiology, on risk-factors involved, however, also disease specific considerations as well as the abilities and the needs of the individual patient must be taken into account.

Depending on the localization and the extent of the neurological lesion, the detrusor and sphincter may react either with overactivity or underactivity (acontractility), mostly both, detrusor and sphincter, are affected.

THE MANAGEMENT OF THE UNDERACTIVE / ACONTRACTILE DETRUSOR

+ underactive sphincter
+ overactive sphincter
+ normal sphincter

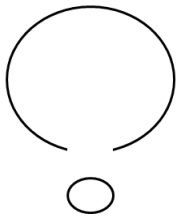
Helmut Madersbacher (Innsbruck, Austria)

According to ICS terminology (2002) detrusor underactivity is defined as a contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or a failure to achieve complete emptying within a normal timespan. Acontractile detrusor is one that can not be demonstrated to contract during urodynamic studies. Although not defined by the ICS as a detrusor with a maximum detrusor pressure (pdetr. max.) below 30 cm H2O in men and 15 cm H2O in women can be considered to be underactive.

The reasons for an underactive detrusor are several. In the context of neuropathology detrusor acontractility or detrusor underactivity are usually observed with damage of (1) the lumbosacral spinal cord, (2) the bladder peripheral efferent pathways, (3) the bladder peripheral afferent pathways and (4) maybe due to a secondary myogenic failure.
There are various options for the management of the underactive /acontractile detrusor, depending also on the function/dysfunction of the sphincter and whether the lesion is complete or incomplete.

1. MANAGEMENT OF THE ACONTRACTILE / UNDERACTIVE DETRUSOR COMBINED WITH AN UNDERACTIVE (INCOMPETENT) SPHINCTER

In complete lesions, the bladder and the striated sphincter as well as the pelvic floor musculature are acontractile. As the bladder capacity is high and the intravesical pressure is low, the kidneys are not at risk as long as regular low pressure emptying is provided. However, neurogenic stress incontinence due to the incompetent sphincter is a problem.



Sacral / Subsacral Spinal Lesion

How to empty the bladder

- **Bladder expression**
  Bladder expression has been recommended since a long time for these patients. With bladder expression (Valsalva or Credé manoeuvre) often unphysiologic high intravesical pressures are created. The reasons for difficulties in expressing the urine from the bladder are the difficulties to open the bladder neck and a functional obstruction at the level of the pelvic floor despite complete flaccid paralysis, resulting in deformation and narrowing of the membranous urethra (Madersbacher, 1975).

  Therefore, bladder expression is potentially hazardous for the urinary tract. Before recommending bladder expression it must be proved that the situation in the LUT is urodynamically safe. Contraindications, such as vesico-uretero-renal reflux, prostatic reflux, urethral pathology, hernias, have to be excluded. ***In general, bladder expression should be replaced by CIC in most patients.*** Alpha-blockers may facilitate voiding, however, urinary stress incontinence may be induced or increased. Therefore Valsalva and Credé only guarantee a good quality of life and are cost effective in long term when the indication is proper and when the situation remains stabile throughout the years, best controlled by (video-)urodynamics.

- **Pharmacotherapy**
  So far there is no randomized controlled study which proves the efficacy of cholinergics. They are not able to induce/increase detrusor contractions, however they seem to increase the muscle tone of the detrusor. The patient may feel the bladder at a lower filling volume. Lowering of outflow resistance is another option to facilitate bladder expression. However, there is no controlled trial, which really proves the efficacy of alpha-blockers. Dosages of spasmolytics needed for the relaxation of striated sphincter are often not tolerated because of side effects, especially general muscle weakness.

- **Intermittent catheterisation**
  In complete lesions with detrusor acontractility therefore for many patients the method of choice to empty the bladder is intermittent catheterisation, either using the sterile or the hygienic technique.

- In **incomplete lesions** with preserved afferent fibres, **intravesical electrostimulation (IVES)** is useful to induce or to improve sensitivity and contractility of the detrusor (s. below).

How to manage neurogenic stress incontinence

The other problem with this type of neurogenic LUT dysfunction is the underactive, incompetent sphincter causing neurogenic urinary stress incontinence.

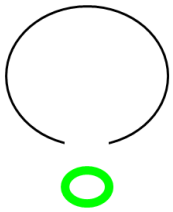
- **Behavioural therapy**
  Neurogenic urinary stress incontinence can be improved to a certain degree by regular bladder emptying, controlled fluid intake and the avoidance of urinary tract infections.

- **Pharmacotherapy**
  There is no controlled trial which really proves the efficacy of α-adrenergics in neurogenic urinary stress incontinence. Whether the 5-HT and NA-Reuptake inhibitor Duloxetin is able to improve neurogenic stress incontinence in incomplete lesions, still needs to be proved through studies.

- **Surgery**
  Especially in “walkers” continence can only be restored through operative treatment. Although the design of the artificial sphincter has improved considerably over the years, there is still a complication rate of about 30 % in these patients on the long term. In women the risk of arosion at the bladder neck is high, overall long-term continence is satisfactory with about 90 % (Venn et al. 2000). Prerequisites for the implantation of an artificial urinary sphincter is neurogenic stress incontinence, an acontractile detrusor (can be achieved by pharmacotherapy or other procedures), a motivated patient and adequate manual dexterity. Moreover, the system has to be replaced between 10 and 15 years after implantation. For women bladder neck slings (mostly from the rectus fascia) have been applied with success. Whether and under which conditions also mid-urethral slings are successful is still under debate as the reports are controversial (Pannek et al. 2012; Abdul-Rahman et al. 2010).

- **Condom catheters and external appliances**
  Despite all efforts, some patients with neurogenic bladder and urinary incontinence need some kind of assistance to gain social continence. In males, a condom catheter (CC) has been one of the choices. Until now, a wide variety of condom catheters has been

available, such as a simple thin latex, plastic or silicon condom catheters or a condom catheter with a double rows of convulsions near the catheter tip to prevent kinking, with an inner flap to prevent the backflow of the urine to the shaft of the penis and an inner wall coated with a self-adhesive. Even a special condom with a passage for catheterization without removal of the condom has been developed. Condom catheters still play a role in the control of urinary incontinence in male patients. Long-term use may cause bacteriuria, but does not increase the risk of UTI compared to other methods of bladder management. Complications may occur less often if the condom catheter is applied properly, and if all hygienic measures necessary are taken, i.e. frequent (daily) change of the condom catheter and maintenance of a low bladder pressure. To prevent a latex allergy, which is becoming more and more common, a silicone condom catheter should be used. To prevent compressive effects the proper size of the condom catheter with self-adhesive is recommended. Moreover, in order to prevent bladder and upper urinary tract damages regular bladder emptying with low bladder pressure and low PVU should be pursued also when a condom catheter is used. In incomplete lesions pelvic floor reeducation may help in the individual patient.



Intrapelvic Lesion

2. MANAGEMENT OF THE ACONTRACTILE / UNDERACTIVE DETRUSOR COMBINED WITH AN OVERACTIVE ("SPASTIC", "HYPERREFLEXIC") SPHINCTER

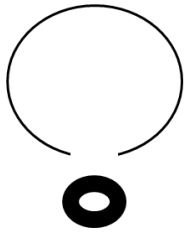
This pattern can be seen with lesions at the conal and epiconal level of the spinal cord, e.g. in myelodysplastic children. The main problem is bladder emptying, the overactive sphincter secures continence, provided that the bladder is emptied regularly.

- **Intermittent (Self-)catheterisation**

This dysfunctional pattern is a clear indication for intermittent (self-) catheterisation which provides regular bladder emptying with low pressure and the spastic sphincter secures continence in between.

In **incomplete lesions** the underactive and hyposensitive detrusor is a good indication for intravesical electrostimulation (see below) to achieve bladder sensitivity, however, balanced voiding is mostly not achieved due to sphincter spasticity. Lowering of outflow resistance caused by the spastic striated sphincter is difficult to achieve (see above). Sacral nerve stimulation (SN5) should be considered to improve voiding (referral to a specialized center).

3. MANAGEMENT OF THE ACONTRACTILE / UNDERACTIVE DETRUSOR COMBINED WITH A NORMALLY FUNCTIONING SPHINCTER



Epiconal Lesion

This pattern is seen with peripheral lesions of bladder innervation especially after pelvic surgery. As the pelvic plexus contains both parasympathetic and sympathetic fibres, decreased parasympathetic innervation may result in decreased detrusor contractility or acontractility, while impaired sympathetic transmission results in incomplete bladder neck closure with weakness of the smooth muscle sphincter. After pelvic surgery urinary retention may occur and **intermittent catheterisation** is the method of choice , however reeducation of the bladder should be aimed as the **lesions are mostly incomplete**. Again **intravesical electrostimulation** is the method of choice to achieve or to speed

up bladder rehabilitation. Also cholinergics in combination with alphablockers and with regular bladder emptying may be successful to achieve balanced voiding. Also sacral nerve stimulation should be considered to improve detrusor function (referral to a specialized center).

4. **MANAGEMENT OF DETRUSORACONTRACTILITY IN THE SPINAL CORD INJURED PATIENT DURING THE SPINAL SHOCK PHASE**

The acute phase of the spinal cord lesion is characterized by the "spinal shock phase" with absent spinal reflexes below the lesion. The bladder is able to store urine, but the patient unable to void; unless appropriate measures are taken, overflow incontinence with bladder-overdistention , urinary tract infections and damage to the upper urinary tract will occur. Prevention of early complications, such as bladder overdistention, urinary tract infections, stone formations and urethral injury is a prerequisite for successful rehabilitation of lower urinary tract dysfunction.

Securing of bladder emptying is of paramount importance. This can be done by intermittent catheterisation (IC), by a suprapubic catheter (SC) or by a transurethral indwelling catheter (ID), rarely adequate spontaneous voiding is possible in incomplete lesions.
Intermittent catheterisation has proved to have the lowest complication rate. The alternative of first choice is a **suprapubic catheter,** connected to a urine drainage bag. Only if a suprapubic catheter is contraindicated (injuries in the small pelvis, acute abdomen, pregnancy) **transurethral indwelling catheterisation** is indicated.
However, despite daily catheter care, changing of the catheter each week, continuous drainage into a closed urine collecting device and fixation of the penis at the abdomen near the groin to avoid a decubital ulcer in the urethra at the peno-scrotal angle, the rate of complications is high. Any catheter should be removed as soon as possible based on a daily decision.

5. **LONG TERM-INDWELLING TRANSURETHRAL AND SUPRAPUBIC CATHETERS**

The long-term use of indwelling catheters for the management of neuropathic bladder is not favorable due to unavoidable complications. In chronic patients, acceptable indications include impossibility to perform IC, uncontrollable urinary incontinence with problems in wearing continence devices (condom catheters) and in patients with an acute situation in the upper urinary tract including urosepsis. For long-term use, in general, a suprapubic catheter is preferable.

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PATHOPHYSIOLOGY of Neuropathic Bladder Dysfunction

The coordinated control of bladder and urethral function is a complex process dependent on neural pathways in the brain and spinal cord. This control ensures storage of urine at a low bladder pressure with continence and complete bladder emptying when socially acceptable.

A host of different disease processes can lead to damage to the various different regions that have a role in controlling bladder filling and emptying. Conditions include head injury, stroke,

multiple sclerosis, dementia and Parkinson's disease. In general, the severity of the urinary tract dysfunction is reflected in the extent of the general disease process; it is unusual to see a patient with severe urological problems and little evidence of other neurological effects of their brain disorder.

Suprapontine Lesions

Suprapontine lesions can lead to problems with either neurogenic detrusor overactivity (NDO) or urinary retention although the latter is less commonly seen as the presenting problem.

It is important to appreciate that NDO seen with a suprapontine lesion is not associated with detrusor sphincter dyssynergia (DSD). The brain lesion is leading to an alteration in the "set point" at which the micturition reflex is activated but the reflex itself remains coordinated as the spinal cord and brainstem centres are all still intact and able to deliver sphincter relaxation as the bladder contraction occurs. This results in low pressure bladder emptying and allows voiding to completion to occur so that suprapontine lesions are very rarely associated with renal damage as a result of high pressure urine storage and voiding.

Suprapontine lesions may present with a variety of different symptoms. These include urinary retention, a lack of awareness of bladder filling and emptying, impaired appreciation of the social context of micturition, frequency with urgency and incontinence.

Supraconal Spinal Cord Injury

Injury to the spinal cord can occur through different disease processes. These include trauma, cervical spondylosis, prolapsed intervertebral discs, multiple sclerosis, transverse myelitis, tumours, vascular disease and abscesses. The majority of lesions will be incomplete, that is there will be some

preservation of sensation and voluntary movement in the dermatomes and myotomes that are distal to the site of the cord damage. However, the pathophysiological effects of cord injury are best appreciated by considering the patient with a complete spinal cord injury.

We will consider a patient with a cord transaction at the T4 level. The immediate effect of such an injury is a loss of reflex activity in the cord below T4; this is termed spinal shock. They will have a flaccid paralysis and loss of sensation below the injury level. Loss of sympathetic nervous system activity will result in reduced blood pressure due to vasodilatation which will also lead to penile engorgement. Parasympathetic paralysis leads to urinary retention (with eventual overflow incontinence) and an ileus with constipation. Spinal shock resolves over days and weeks as reflex activity returns.

The T4 lesion will, of course, preserve normal spinal cord function above T4 but below the injury level the spinal cord will be viable and functional but disconnected from the higher centres in the brain. The segment below the injury level is called the distal autonomous cord (DAC). Somatic reflex activity in the DAC will lead to increased striated muscle tone and reflexes with associated spasm as a result of stretch reflexes which lack descending inhibitory influences. There will be complete sensory loss from the injury level down.

Reflex activity in the sympathetic system is also uninhibited and this leads to the important syndrome of autonomic dysreflexia. The phenomenon is seen when a noxious stimulus such as bladder distension, constipation or trauma sends a sensory input into the DAC. The response is a mass sympathetic outflow which will result in vasoconstriction in particular. This will decrease the intravascular space and increase venous return to the heart and lead to an increase in cardiac output (Starling’s law). The brain and spinal cord above the injury level will respond by increased vagal activity (bradycardia and negative inotropic effects) and vasodilatation above the injury level. For patients with an injury

level below T6, the compensatory mechanisms are sufficient to prevent significant blood pressure changes. However, injuries above T6 have a large part of the body under the influence of the DAC and a rather limited area supplied by the normal brain/spinal cord. The distal vasoconstriction cannot be compensated for and there is a rise in blood pressure, which can be severe. The classical picture is of a patient with a severe headache, facial flushing, a cool trunk and lower limbs and a bradycardia. Immediate treatment is required; removal of the noxious stimulus will lead to a rapid resolution of the crisis. If the stimulus cannot be removed then antihypertensive medication must be administered in a way which produces an immediate effect.

Urinary tract reflex activity will also return as spinal shock resolves. Initially bladder filling occurs with normal bladder compliance but then the bladder will contract spontaneously in response to filling – neurogenic detrusor overactivity. This reflex is probably mediated (at least in part) by afferent C-fibres which are not generally involved in normal voluntary detrusor contractions. The response of the distal striated sphincter to the bladder contraction is active contraction rather than relaxation – detrusor sphincter dyssynergia (DSD). This results in a prolonged bladder contraction with obstructed voiding; in men, the bladder neck and prostatic urethra is seen to be widely opened on X-ray screening, with hold up at the distal sphincter. When the sphincter eventually relaxes some voiding will occur but bladder emptying is often incomplete as the detrusor contraction fades prematurely. Increased intravesical pressures are associated with vesico-ureteric reflux in a minority of patients. It should be noted that the degree of DSD that is seen varies from patient to patient; some patients will have complete reflex bladder emptying with little evidence of any distal sphincter hold up while others will not void at all during episodes of neurogenic detrusor overactivity.

Bowel pathophysiology follows a similar pattern with reflex bowel evacuation occurring but with obstructed defaecation in some

patients due to sphincter dyssynergia. Penile erection will occur in response to direct genital stimulation (and psychological stimuli in some cases with injuries below the T11-L2 sympathetic outflow). Reflex ejaculation can be preserved if the injury is above T11 but is usually inhibited so that relatively strong genital stimulation is needed to fire the reflex.

Conus and Cauda Equina Injury

Once again it is apparent that most distal spinal cord and peripheral injuries are incomplete. However, it is helpful to consider the effects of a complete injury in order to understand the pathophysiological effects of damage at this level of the neuroaxis.

There will be a complete sensory loss in the affected dermatomes;; the “saddle area” is supplied by S2 downwards. Somatic motor nerves will have been destroyed (cell bodies are in the anterior horn of the spinal cord) and this will result in a flaccid paralysis. Anal tone will be reduced as a result.

The detrusor will be areflexic but compliance will be reduced in some patients so that the bladder is not necessarily “safe” as resting pressures may be high with a risk of upper tract dilatation in some cases. The distal striated sphincter will be flaccid but the smooth muscle of the distal sphincter will retain tone; in some cases, tone will be low and stress incontinence will be prominent while in other cases, tone will be relatively high with the potential to allow a poorly compliant bladder to retain urine at high pressures. If smooth muscle tone is high, the condition is described as “non-relaxing urethral sphincter obstruction”.

Reflex rectal activity is absent so that constipation is present. Anal sphincter weakness will lead to faecal incontinence. Impotence is usually present although some residual psychogenic erectile activity may be preserved. There is no reflex ejaculation.

CASE STUDY 1:

SPINAL CORD INJURY – Upper motor neuron type

A 31 years old man sustained a T2 spinal cord injury (SCI). He is wheelchair bound and has been emptying his bladder spontaneously. He complains of recurrent UTI's and urinary leakage. He is not on any medications.

Q: What is the likely type of injury he has sustained?

A: He has sustained an upper motor neuron (UMN) type injury, with reflex bladder voiding, detrusor sphincter dyssynergia and poorly sustained bladder contractions.

Q: What are the main investigations in this patient that will help you in management?

A: Video-urodynamic studies and ultrasound scan of kidneys.

Q: What are the findings on this VCMG trace? (will be shown during presentation)

A: There appears to be neurogenic detrusor activity. It appears to be a classical saw tooth pattern. The detrusor pressures are very high and sustained.

Q: What is shown on this picture?

A: This demonstrates bilateral reflux and a trabeculated bladder.

Q: What is the abnormality on the voiding study?

A: This demonstrates a blown up prostatic fossa and a contracted sphincter suggestive of detrusor sphincter dyssynergia.

Q: What is detrusor sphincter dyssynergia?

A: It is defined as involuntary contraction of the urethral and/or periurethral striated muscle simultaneously with a detrusor contraction. This is specific to a supraconal neurological disorder.

Q: How will you treat this patient?

A: The aim is to achieve low pressure storage and complete bladder emptying without incontinence.

One will be guided by the urodynamics result but assuming that this patient with T2 SCI has neurogenic detrusor overactivity and DSD, he will be started on anticholinergic medications and institute a program of clean self intermittent catheterization (CSIC). He will be closely monitored and will undergo ultrasound scan of the kidneys and repeat urodynamics to ensure the bladder pressures have come down. Assuming that there are no problems i.e. UTI;s or problems with CSIC he will be reviewed yearly.

Q: He comes back and although he was bothered by UTI's he doesn't like CSIC and wants to know other options?

A: Apart from CSIC the other options include:

- Behavioural & timed void – not suitable for him
- Indwelling catheter – Suprapubic or urethral – not acceptable to him as he is very young and mobile
- Urethral stents / sphincterotomy with sheath - again not acceptable to him
- Augmentation cystoplasty with or without Mitrofanoff but will need to perform CSIC (almost 100% certain)
- Sacral anterior root stimulator (SARS) – not suitable for him as he has an incomplete SCI. This is an option for wheelchair bound patients who have complete injury as it requires a posterior rhizotomy to abolish NDO. This will

also lead to a loss of reflex erections.

Q; What are the pros and cons of SARS?

A: The benefits include:

- Abolition of reflex bladder
- Increased bladder capacity
- Abolition of autonomic dysreflexia
- Bowel management

However the downsides are:

- Stress incontinence
- Loss of reflex erections
- Loss of reflex ejaculation
- Loss of reflex defecation

Q: What are the problems with indwelling catheters?

A: They include

- rUTI’s
- Blockages
- Need for regular changes
- Stones
- Risk of cancer

Q: What are the complications of stents in his case?

A: He will be incontinent and will have to wear sheath with risk of detachment. The stents can dislodge, block and there is a risk of encrustation. External sphincterotomy is the standard alternative to the use of a stent for patients with problematic DSD who wish to use penile sheath collection, but is irreversible.

Q: He can perform CSIC if urinary leakage can be controlled but feels anticholinergics are not helpful in the absence of infections?

A; I think he has uncontrolled NDO and I will suggest intravesical botulinum toxin.

Q: What type of botulinum toxin is normally used?

A: Botulinum toxin A is used in urology.

Q: What's the efficacy and side effects of botulinum toxin?

A; The efficacy ranges from 36-89% (mean 70%). The effects last from 4-10 months (mean 6 months). The side effects include pain, UTI (<5%), bleeding (<5%), no benefit, need for further injections, need for self-catheterization (very variable 4-45%). In NDO it is more than 50%. The general effects include flu like symptoms, dry mouth and malaise. It is an unlicensed indication.

Q: How does it work and how you give the injections?

A: It is thought to work by blocking the presynaptic release of acetylcholine at the neuromuscular junction. It achieves this by blocking the SNAP 25 protein. This is the basis of decrease in detrusor pressures and phasic contractions in both idiopathic and neuropathic bladders. However, the patients also report a significant decrease in urgency and hence, it is suggested that botulinum toxin also modulates the sensory pathways. This is thought to work by acting on P2X receptors.

It can be administered under general or local anaesthesia. In NDO 300IU (now 200IU) mixed in 30 mls of normal saline are injected at 30 sites sparing trigone. The injections can be

submucosal or intra-detrusor.

Q: Are you aware of any long term effects of repeated injections or Botox loses its efficacy after repeated injections?

A: No significant bladder fibrosis has been reported on histological examination after repeated injections. Also up to 9 repeat injections have not demonstrated any decrease in efficacy of botulinum toxin.

Q: is there a role of sacral neuro-modulation (SNS) for treatment of NDO with incontinence in this patient.

A: Recently, it has been shown that SNS might be beneficial in controlling NDO in SCI patients but this is still in investigational stage.

Q: What is the basis of SNS

A: The basis is continuous use of mild electrical activity to stimulate the sacral nerves to the bladder and pelvic floor which in turn activate or inhibit neural reflexes.

Q: How is the SNS delivered?

A: Generally a 2 stage technique is used as it has been shown that the 2 stage method improves the rate of positive tests from 50% to 80%. This is a minimally invasive procedure and can be performed under general or local anaesthesia. Initially a test implant is inserted into the S3 foramina. The patient keeps a symptoms diary for 2 weeks and this is compared with the pre-operative evaluation. A greater than 50% benefit in symptoms entitles the patient to have the second stage (permanent implant) fitted.

Q: What is the efficacy / side effects and complications of SNS?

A: It is thought to be effective in 60-70% of cases. The effects last for 3-5 years. Occasionally, the patient complains of pain at the site of neurotransmitter implant. They can also develop leg pain. The explantation rate is 10%. This is mainly due to infection or lack of sustained efficacy.

Q: What is a clam cystoplasty (see picture) and how it works?

A: The principle is to bivalve the bladder like a clam and patch the defect with a piece of bowel, generally ileum. This increases the capacity of bladder and lowers the intra detrusor pressures. Additionally, it decreases the amplitude of contractions by preventing sustained detrusor contractions.

Q; What are the contra-indications to a clam?

A: Severe inflammatory bowel disease i.e. Crohn's disease, post surgery or radiotherapy critically short or abnormal bowel. Patients' unwillingness or inability to perform self-catheterization.

Also, significant renal or hepatic impairment as it may result in an inability to cope with metabolic consequences of bowel in urinary tract.

Q: What are the potential complications of clam cystoplasty?

A: This includes:

- Mucus: the average daily production from the incorporated bowel segment is 35-40 grams. This does not decrease over time. This can lead to infections, stone formation and blockages. Bladder washouts might be required with acetylcysteine to dissolve excess mucus.

- Bacteriuria & UTI: Almost 100% of patients will have asymptomatic bacteriuria. The incidence of clinically significant UTI is around 4-43%.

· Biochemical abnormalities: The presence of absorptive bowel in the urinary tract leads to reabsorption of ammonium chloride and excretion of bicarbonate resulting in acid base imbalance. This is clinically important in very few cases. These patients can develop hyper-chloraemic acidosis. The treatment is administration of bicarbonate in about 15% of cases.

· Intermittent catheterisation rate: The rate is around 50% in idiopathic patients but near to 100% in the neurogenic patient.

· Stones: The reported rates are highly variable between 0-53%. It is generally thought to be around 15%. They are more common with an associated Mitrofanoff procedure.

· Perforation: Spontaneous perforation is a rare complication (<1%) but carries a mortality of 25% mostly due to delay in diagnosis.

· Cancer: There is an increased incidence of cancer in augmented bladders. However there is a long latent period (>10 years). This is associated with chronic inflammation, urinary stasis and recurrent UTI's. The tumors are generally adenocarcinomas and in the region of anastomosis. The mechanism seems to be related to bacteriuria. This leads to reduction of urinary nitrates to nitrites by colonic bacteria. This reacts with urinary amines to form N-nitrosamine, implicated in carcinogenesis.

· Bowel changes: This usually results in diarrhea. The symptom is troublesome in up to 30% of cases. Also there can be a decrease in absorption of B12 and folic acid leading to neurological complications.

Q: How do you follow up a patient with clam cystoplasty?

A: Once stable the patients are seen on a yearly basis with ultrasound scan of kidney and an x-ray KUB. The biochemical analysis includes evaluation of kidney and liver function and estimation of serum chloride, bicarbonate, B12 and folic acid. They are advised to contact the department urgently if they develop recurrent UTI's, haematuria significant weight loss or severe lethargy. They will undergo yearly surveillance cystoscopies 10 years after operation. However it seems that the rate of pick up by this method is questionable.

Q: What are the main complications with UMN type SCI?

A: This is NDO with DSD leading to rUTI's, low bladder capacity with urinary incontinence, reflux with scarring and deterioration of upper tract function. However, the most dangerous problem at present is autonomic dysreflexia (AD).

Q: What is AD?

A: It is sudden disordered sympathetic activity due to a specific stimulus below the level of SCI in patients with an injury level above T6.

Q: What are the causes of AD?

A: It can be nociceptive stimulation in the region of the distal autonomous cord. In the majority of cases the trigger is an overfilled bladder, constipation or distal skin infection.

Q: What is the mechanism of AD?

A: There is sympathetic over activity of the distal autonomous cord leading to vasoconstriction in the territory supplied by the sympathetic nerves originating from the distal autonomous cord with compensatory vasodilatation of the normally innervated sympathetic territory

Q: What happen below the level of SCI A: These include

- Increasing vasoconstriction
- Decreasing compensatory vasodilatation
- Splanchnic venoconstriction
- Increased venous return to heart
- Increased cardiac output with increased systemic resistance
- Severe hypertension
- Compensatory bradycardia

Q: What does patient experience?

A: The patient complains of the following symptoms

- Headache (usually)
- Hypertension
- Bradycardia

• Sweating and flushing

Q: What is the treatment?

A: this is a life threatening condition. The management includes:

- Remove cause (drain bladder, evacuate bowels, examine toes)
- Sit up the patient
- GTN spray
- S/L Captopril or IV Labetolol, Phentolamine

CASE STUDY 2:

SPINAL CORD INJURY – Lower motor neuron type

A 38 years old man sustains a L3 SCI.

Q: What is the likely clinical picture of this type of injury?

A: Assuming that there has been damage to the conus, this will be a lower motor neuron (LMN) type SCI. The features include:

- Flaccid paralysis and sensory loss
- Absent conus reflexes.

- Detrusor areflexia or reduced compliance.
- Sphincter weakness or non-relaxing urethral sphincter obstruction.

Q: What do you know about detrusor leak point pressure (DLPP) & abdominal leak point pressure (ALPP)

A: DLPP is the lowest detrusor pressure at which urine leakage occurs in the absence of either a detrusor contraction or increased abdominal pressure. McGuire observed in spina bifida patients that if this is greater than 40 cmH2O then there is a higher risk of damage to upper tracts.

The ALPP is the intravesical pressure at which urine leakage occurs due to increased abdominal pressure in the absence of a detrusor contraction. If this is greater than 150 cm H2O then the urethra is unlikely to be the cause of urinary incontinence.

Q: How will you then manage this patient?

A: If compliance is normal he will have a generally safe bladder and the options include:

- Behavioural & timed voiding in incomplete lesions
- Emptying by straining if sphincter weakness allows the bladder to empty effectively in this way
- CSIC if incomplete voiding or has UTI's If he complains of urinary incontinence then the treatment options include:
  - Sheaths
  - Bulking agents
  - Slings

- Artificial urinary sphincter (AUS)

Q: How many components are there in an AUS?

A: Three. Cuff, reservoir and control pump

Q: What is the success rate of AUS?

A: It is around 80% at 10 years. However it is less in SCI patients especially if they are wheelchair bound.

Q: What are the complications of AUS?

A: They include

- Infection & Erosion
- Persistent leakage
- Mechanical failure

CASE STUDY 3:

MULTIPLE SCLEROSIS

A 48 years old lady has been diagnosed with Multiple Sclerosis about 3 years ago. She presents with worsening lower urinary tact symptoms. Her main complaints are frequency, urgency,

nocturia with occasional urinary incontinence. She also suffers from urinary tract infections from time to time.

Introduction

MS affects women three times as often as men. The clinical picture depends on the distribution of demyelination in the nervous system with spinal cord disease being particularly associated with urinary tract symptoms. Progression is very variable and follows different patterns (e.g. relapsing/remitting and primary or secondary progressive disease). Acute deterioration can occur in response to urinary tract infections.

The commonest pattern of lower urinary tract dysfunction is a combination of neurogenic detrusor overactivity and impaired bladder emptying due to detrusor sphincter dyssynergia and poorly sustained detrusor contraction. The severity of the urological symptoms is broadly correlated with the overall level of disability.

Clinical Assessment and Management

This must include a general assessment (cognitive ability, hand function, mobility etc.) and enquiry into urinary, bowel and sexual symptoms. If the patient is not catheterised, a urine sample should be tested in order to look for evidence of infection.

The ambulant patient most commonly presents with urgency, frequency and incontinence. Problems with bladder emptying are also commonly reported. Specific investigation can be limited to a measurement of residual urine volume. A classic algorithm is in use which suggests that intermittent self catheterisation (ISC) be introduced if the residual is over 100ml. Anticholinergic drugs are used if there is urgency present either in the patient with good bladder emptying or despite bladder emptying issues being addressed by ISC. The success of this basic approach is

dependent on the expert assistance of specialist nurses.

In patients with persistent symptoms, it is advisable to undertake urodynamic studies in order clearly to define the underlying pathophysiology.

Intravesical injections of botulinum toxin have established a clear role in managing persistent detrusor overactivity, although virtually all MS patients will need to self-catheterise after treatment.

Desmopressin has been shown to be useful in reducing nocturia although the usual precautions need to be taken in order to detect hyponatraemia which will affect a minority of patients who are on long-term therapy.

Surgical treatment has a very limited role in treating persistent detrusor overactivity in patients with MS. Neuromodulation (sacral nerve stimulation) has been reported to be effective in some patients but the evidence base is extremely weak. Cystoplasty is rarely indicated as future neurological deterioration may interfere with the ability of the patient to self-catheterise.

Containment of incontinence is an important aspect of care in the patient with more advanced disease. Pads in women and the use of penile sheath collection systems should be introduced with the support of continence nurse specialists.

Suprapubic catheterisation is needed in many patients either because bladder emptying is not adequately addressed with ISC or because incontinence cannot be contained. It is important that patients and carers do not fall into the trap of expending excessive amounts of time and energy in trying to avoid catheterisation, as effective and convenient bladder drainage can frequently be achieved by insertion of a suprapubic catheter. A catheter valve should be offered as many patients prefer to

use a valve rather than a leg-bag during the day; urine storage problems can be addressed by using anticholinergics, botulinum toxin or urethral closure.

For the catheter-intolerant patient with repeated blockages, frequent infections or pain which does not respond to standard measures, an ileal conduit diversion may be needed. In such patients, a decision has to be made as to whether to undertake a simple cystectomy at the same time as the diversion by way of prophylaxis against the development of pyocystis.

Bowel management is also important. Patients with MS usually have intact sacral reflexes; this allows a regime that involves stimulating a reflex bowel evacuation at a convenient time (typically alternate days). Firstly stool consistency and bowel motility are addressed with oral laxatives and, secondly, bowel evacuation is triggered by rectal stimulation, suppositories or enemas. An alternative is to use rectal irrigation.

Sexual dysfunction should also be addressed. Management of erectile dysfunction follows conventional lines.

CASE STUDY 4:

CAUDA EQUINA SYNDROME

A 33 years old fit builder develops sudden onset of lower back

ache after a period of heavy lifting. He starts to develop numbness in lower limbs spreading to perineal area. He takes pain killers but the symptoms do not settle and over the next few days he starts to develop urinary and bowel dysfunction. An emergency MRI reveals slipped disc at L5/S1 level. He undergoes emergency decompression. The recovery is ok for limb numbness but he cannot empty the bladder and is dependent on self catheterization.

A classical cauda equina syndrome (CES) arises from compression of the nerve roots that are passing along the spinal canal in the lumbar region. Nerves that will be affected include:

- Nerves to skeletal muscle - Compression of these nerves will result in muscle weakness, typically affecting the lower legs, toes and anal canal regions. Absent reflexes may also be detected.

- Somatic sensory nerves - These nerves provide sensations over the skin of the saddle area in particular. This includes the perianal region, the perineum, the back of the legs and the feet.

- The innervation of the lower urinary tract - This will classically result in retention of urine with damage to the nerve supply to the external urethral sphincter resulting in stress incontinence. There will also be a loss of sensation of bladder filling and voiding.

- The innervation to the lower gastrointestinal tract - Damage to these nerves will result in constipation. Weakness of the anal sphincter may result in faecal incontinence. There will be an absence of bowel related sensation.

- Damage to the innervation of the genital tract - This will result in an absence of penile erection along with absent ejaculation and a loss of sense of orgasm and other sexual

sensation.

The commonest presentation of CES is an acute central disc prolapse in the lumbar region. Severe pain will be accompanied by neurological symptoms and signs and by urinary retention (and/or incontinence) with constipation. This is a neurosurgical emergency that mandates investigation with an immediate MRI scan of the spine and surgical decompression.

Diagnostic difficulty can arise if the onset of symptoms is insidious and the syndrome incomplete.

Clinical Assessment and Management

Patients with CES are often discharged from spinal surgery units soon after undergoing spinal decompression. Too often, there is an underestimation of the degree of disturbance to pelvic organ function and the patient will be left with life-changing symptoms that have not been adequately addressed. It often falls to the urologist to pick up on these issues. Admission to the regional spinal injuries unit is worth considering as specialist nursing and medical input is important.

The patient will usually either be in a degree of chronic retention or be emptying by straining. Stress incontinence is very likely to be present. ISC should be discussed and trialled. Urodynamic studies should be performed. If the patient remains wet due to stress incontinence while on ISC, surgical treatment should be offered with an artificial urinary sphincter in men and either an artificial sphincter or pubo-vaginal sling in women. It is important to appreciate that a patient who is reliably dry will inevitably need to self- catheterise; if bladder emptying is achieved by straining, some degree of stress incontinence will always be present and the trade off that the patient faces is that treatment of incontinence will necessitate a switch to ISC.

Urine storage at low pressure cannot be guaranteed in patients with CES as poor compliance is a feature in a minority of cases and patients with incomplete lesions may have neurogenic detrusor overactivity. Poor compliance is most reliably dealt with by augmentation cystoplasty; there is limited data available on the effect of botulinum toxin injections on bladder compliance and long-term, reliable restoration of safe urine storage probably cannot be relied on. An alternative to cystoplasty in the patient with a poorly compliant bladder is suprapubic catheterisation, with the catheter maintained on free drainage in order to keep bladder pressures low.

Patients who have problems with urethral ISC due to physical or psychological difficulties with the technique should be offered a continent, catheterisable abdominal (Mitrofanoff) conduit.

If bladder compliance is normal but a patient elects to use a long-term suprapubic catheter to empty the bladder, a catheter valve should be offered. Stress incontinence may need to be treated in order for the patient to be dry while the valve is in use.

Bowel problems are typically of constipation and stress faecal incontinence (embarrassing loss of flatus and anal seepage are particularly problematic). Patients may find an anal plug helps to contain seepage. Manual evacuation of faeces by the patient is the mainstay of management in this group of patients; dietary adjustments and the use of oral agents may be helpful as well. Rectal irrigation is finding an increasing role. A minority of patients with CES will find their bowel problems so difficult to manage that a colostomy is requested; a transverse colostomy may be preferred to a sigmoid stoma as the left colon will be denervated and a sigmoid colostomy may be constipated as a result.

Impotence and lack of orgasm are additional problems that need to be considered. Once again, management of erectile dysfunction follows traditional lines.

PRINCIPLES OF MANAGEMENT OF A NEUROPATHIC BLADDER

The main principle of management of a neuropathic bladder is to assure that the detrusor pressure remains within safe limits during both the filling phase & the voiding phase with the aim of:

1. Protecting of the upper urinary tract
2. Improving of urinary continence
3. Improving the patient's quality of life
4. Restoring normal (parts of) lower urinary tract (LUT) function

When treating patients with neurogenic lower urinary tract dysfunction (NLUTD) it is important to consider any patient's disability, cost-effectiveness of the treatment, technical intricacy of administrating the treatment and any possible complications.

The treatment of NLUTD falls into the following main categories:

- Behavioral approaches
- Lifestyle interventions
- Pads, portable urinals

- LUT rehabilitation
- External appliances:

  clean intermittent catheterization

  condom or Foley catheterization for patients with incomplete bladder emptying (e.g. elevated PVR)
- Pharmacotherapy Antimuscarinic anticholinergic agents are the standard therapy
- Surgery

  Intradetrusor Botulinum toxin injections

  External Sphincterotomy

  Bladder augmentation

  Bladder reconstruction

  Urinary Diversion

The choice of treatment depends on which LUTS is being treated i.e. storage, voiding and/or post-micturition symptoms.

SUMMARY AND TAKE HOME MASSAGES

The management of a neuropathic bladder secondary to various neurological conditions has to be individualized not only according to the disease process but also keeping in mind the patient's desires and limitations of the healthcare system. The overall principles of managing a neuropathic bladder are fairly

standardized as per EAU & ICI guidelines.

Recommended Reading & References

1 - A UK consensus on the management of the bladder in multiple sclerosis. CJ Fowler, JN Panicker, M Drake, C Harris, SCW Harrison, M Kirby, M Lucas, N Macleod, J Mangnall, A North, B Porter, S Reid, N Russell, K Watkiss and M Wells. Journal of Neurology, Neurosurgery and Psychiatry 2009, 80, 470-7.

2 - British Association of Urological Surgeon's suprapubic catheter practice guidelines. SCW Harrison, WT Lawrence, R Morley, I Pearce and J Taylor. BJU International 2011, 107, 77-85.

3 - International Consultation on Incontinence – 2012

4 - European Association of Urology Guidelines -2013

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Notes