



Voiding Dysfunction due to Underactive Detrusor: Can one restore function?

W10, 15 October 2012 09:00 - 12:00

Start	End	Topic	Speakers
09:00	09:05	Welcome and introduction	• Jean-Jacques Wyndaele
09:05	09:20	Pathogenesis and clinical picture	• Jean-Jacques Wyndaele
09:20	09:40	Diagnosis	• Stefan de Wachter
09:40	09:55	Special tests	• Tom van Meel
09:55	10:15	Behavioural training and drugs	• Jean-Jacques Wyndaele
10:15	10:30	Topics from first part	All
10:30	11:00	Break	
11:00	11:15	Physiotherapy	• Alexandra Vermandel
11:15	11:30	Neuromodulation	• Stefan de Wachter
11:30	11:45	Catheterization	• Stefan de Wachter
11:45	11:55	Specific surgery	• Jean-Jacques Wyndaele
11:55	12:00	Conclusions	All

Aims of course/workshop

Voiding dysfunction due to detrusor underactivity is underdiagnosed. We will discuss possible pathogenesis, clinical picture in men and women, diagnostics and special tests. We will suggest when behavioural treatment, physiotherapy, neuromodulation, catheterization and special surgery should be applied and what the outcome is. Interactive case discussions will illustrate the presentations and may challenge them.

Educational Objectives

There is definite need to better understand underactive detrusor and the clinical consequences. In this workshop all available data from basic and clinical literature will be put together, compared, and valued in order to understand what we still do not know and to define the best practical treatments available today.

Underactive Detrusor, an Overview

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Introduction

This course aims at discussing a clinical item of Functional urology which has been receiving very little attention from researchers and clinicians. And yet it is clinically relevant and often present while if demonstrated during work out in cases with LUT problems it receives often little attention. The consequence of this attitude is not really understood and the role it can play in outcome of some treatments remains to be further studied. Without any doubt it deserves more attention.

Definition: Underactivity has been defined as a contraction of reduced strength and /or duration resulting in prolonged or incomplete emptying of the bladder. It might be necessary to broaden his description as some patients will use Valsalva /straining and succeed in emptying completely in a timely manner. Some will show during straining a detrusor contraction, others will show no such contraction during a urodynamic test.

Pathogenesis, Differentiation and Clinical Picture

The pathophysiological mechanisms can differ. Probably the pathogenesis is multifactorial.

Detrusor contraction can be weak but continue for a long time. Detrusor contraction can be weak or stronger but exist only very shortly, too short to permit proper emptying. Detrusor contraction can also not be used, completely or partly, as in cases of voiding with abdominal straining. Detrusor contraction can also be present but in an insufficient way, in patients who perform intermittent catheterization without trying to void spontaneously.

Detrusor underactivity is a urodynamic diagnosis and thus not a disease, as it can be present in many different conditions and can have many different causes.

One cause can be neurologic and this would then be called “neurogenic detrusor underactivity”. There are several neurologic diseases in which NDU has been demonstrated alone or in combination with overactivity: Parkinson disease, multiple sclerosis, diabetes, spinal cord lesion and more.

Probably in many a direct cause can not be found, which would then be called “idiopathic detrusor underactivity”.

One can add “functional detrusor overactivity” in cases where detrusor activity is potentially present but is not used, as in cases with stress urinary incontinence or dysfunctional voiding.

Another way to differ DU would be histological, neurologic or combined causes.

The detrusor muscle is a smooth muscle and its anatomo-physiological properties are important to understand mechanisms behind its activity or activity disturbances.

Detrusor muscle is one of the tissues present in a bladder wall and too much collagen f.e. can prevent muscle contractions to result in sufficient and long enough pressure development in the

bladder. Histologically other disturbances have been demonstrated as widening of spaces between muscle cells.

Detrusor activity relates to innervation and in normal voiding depends on proper sympathetic/parasympathetic interaction, central afferent signaling, cerebral reaction and integration and proper peripheral nerves and spinal cord transmission. The urethral/bladder interaction is important. Also the bowel/ bladder interaction, still often overlooked can play a role.

Clinical Picture

DU can cause symptoms directly related to voiding as hesitancy, weak stream, prolonged bladder emptying. But it can also cause postvoid dribbling. Incomplete emptying can cause feeling of pressure in lower abdomen. Complications as recurrent urinary tract infection, stone development, hydronephrosis can be seen. Other symptoms can be related to alternative ways of bladder emptying: incontinence, prolaps et al.

Epidemiology

DU has been found in elderly people (> 60 years old). It was found very prevalent in incontinent institutionalized elderly. Menopause has been mentioned as cause due to axonal degeneration and loss of muscle cells.

Neurological causes have been mentioned before and should also include iatrogenic causes as after major pelvic surgery with nerve damage. Being under anesthesia without proper bladder drainage is a known cause of retention and overdistension as described after delivery and different surgery.

Bladder outflow obstruction in men with development of residual is a special interesting clinical item. Mostly attributed to bladder outflow obstruction, it has been shown that DU is not always the consequence of exhaustion of obstructed voiding but can also be present without BOO.

Surgery for incontinence in women is evenly challenging as often DU has not been diagnosed previous to surgery.

DU can be caused by drugs with bladder relaxing effect. These are not only antimuscarinics but also neuroleptics, calcium channel antagonists.

Though an accurate estimate of prevalence is lacking one can assume that DU is prevalent and that there is a clear clinical need to include it in diagnosis of different LUT conditions.

References

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Handouts Workshop on “Voiding dysfunction due to underactive detrusor: can one restore function?”

The following is an excerpt from an article by MJ Semins and MB Chancellor, published in Nature Clinical Practice Urology 2004, Vol 1, PP 78-84. It gives a nice summary of the available literature. It will be expanded upon and updated during the presentations at the workshop, but already provides the reader with available literature and references.

Diagnosis

Incomplete bladder emptying indicative of detrusor underactivity is an entity that is not well known. It can be caused by abnormal bladder contractility, bladder outlet dysfunction or both. Complete bladder emptying is dependent on the CNS, detrusor smooth muscle activity, coordinated bladder and urethral sphincter function, and voluntary initiation of voiding. Should any of these components go awry, incomplete bladder emptying could result(1).

Underactive detrusor is often accompanied by sensory dysfunction of the bladder, which is mainly caused by diabetes. This is characterized by impaired sensation of fullness, increased bladder capacity, reduced detrusor voiding pressure and increased postmicturition residual volume (2). Besides neurogenic failure, myogenic failure can also cause detrusor underactivity.

Evaluation always begins with a careful history and physical examination. General symptoms of impaired bladder emptying are a feeling of fullness in the bladder area, straining to void, hesitancy, interrupted or diminished stream, double voiding, sensation of incomplete emptying, lower abdominal discomfort, constant dribbling, and recurrent urinary tract infections (3). Physical examination might reveal a distended bladder, but the most characteristic features are elicited by a careful neurourologic examination. Evaluating the sacral dermatomes includes assessing perianal sensation, anal sphincter tone, and the bulbocavernosus reflex. Deep tendon reflexes in the lower extremities, clonus, and plantar responses should also be routinely evaluated (3).

Non-invasive evaluation consists of urinalysis to rule out urinary tract infection, ultrasound to check for postvoid residual urine and uroflowmetry to screen for reduced flow. The findings of reduced flow rate on uroflowmetry and postvoid residual urine does not discriminate between outflow obstruction or underactive detrusor. This can be evaluated by urodynamics which also evaluates bladder sensation. Interpretation problems arise when a patient with impaired

bladder emptying is not able to void during the urodynamic tests. In these patients ambulatory urodynamics may be helpful (4).

Treatment

One approach to treating detrusor underactivity is to drain the bladder by indwelling urethral or suprapubic catheters or clean intermittent catheterization. There is a consensus that intermittent self-catheterization is the preferred treatment. Indwelling catheters are used when patients are either unable to or refuse to intermittently catheterize themselves. Generally, suprapubic tubes are more comfortable than urethral catheters, are easier to change and allow the patient to remain sexually active. In female patients, long-term urethral catheterization can cause erosion damage and fistula formation to the vagina and should be avoided (3). Those patients with sensory uropathy, for example from diabetic cystopathy, should void at routine times to prevent chronic overdistention of the bladder (5).

Neuromodulation is another method to treat the underactive detrusor. Intravesical electrical stimulation has been proposed by some but is not generally accepted due to poor reproducibility of the results and the intensive treatment schedule. Sacral nerve stimulation can promote voiding in patients with idiopathic detrusor sphincter dyssynergia and incomplete bladder emptying (6). The role of other forms of neuromodulation eg urethral afferent stimulation, pudendal stimulation and tibial nerve stimulation is currently under investigation.

The complete manuscript can be found at

<http://www.nature.com/nrurol/journal/v1/n2/full/ncpuro0054.html>

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Workshop 10

Voiding Dysfunction due to Underactive Detrusor: Can one restore function?

Special tests by Tom David Van Meel

Following a cauda equina or a conus medullaris lesion, neurogenic lower urinary tract dysfunction (NLUTD) may underpin on the afferent side dysfunction of bladder filling sensations and on an efferent side reduced bladder activity.

There is no doubt that neurophysiological testing has been, is and will be the most important tool in developing a better understanding of lower urinary tract function (LUT). It was thought that a positive bethanechol test revealed detrusor denervation hypersensitivity and the muscular integrity of an acontractile detrusor (1). The method to quantify the filling function and micturition, using an urodynamic investigation, is the gold standard for investigating NLUTD(2). During urodynamics, there is a possibility to combine bladder pressure measurement and bladder filling sensations. Determination of electrical perception threshold (EPT) in the bladder allows for the investigation of afferent LUT pathways and has been proposing to detect and differentiate neurologic bladder dysfunction(3). These tests may influence diagnosis or help in patient management.

We aim to shed light on the clinical impact of sensory evaluation in the LUT using cystometric sensory evaluation and determination of EPT in the bladder in cauda equina syndrome(CES) patients.

Patients with a CES injury were included in this study. All patients attended the urodynamic clinic as part of their diagnostic work-up, and were considered fully cooperative. A 7.4 Fr 3 lumen catheter with two pressure lines was inserted transurethraally for urodynamic investigation.



The bladder was emptied before filling the bladder. Abdominal pressure was measured using a single lumen balloon catheter introduced in the rectum. All pressure lines were connected to a 6-channel urodynamic unit. The vesical, urethral and rectal pressures were measured simultaneously. All patients were evaluated using a standardized protocol, including cystometry and determination of EPTs in the bladder. After these initial tests, a subcutaneous injection of a muscarinic agonist (1.5ml of bethanechol chloride 5mg/ml) was given. After 15 minutes, urodynamic investigation and EPT determination were again assessed as described during initial investigations. The bladder was again emptied before the start of the second bladder filling. Detrusor pressure was monitored until 250ml bladder filling or an increase of detrusor pressure > 80 cmH₂O. The bethanechol test was considered positive if the detrusor pressure increased above 20 cmH₂O.

Bladder fullness sensations are conveyed to the spinal cord by the pelvic and hypogastric nerves. As the bladder fills, increasingly strong bladder afferents travel via synapses in the sacral cord to the brainstem and midbrain. These afferents arising from the bladder are mechanoreceptive and nociceptive. Clinical evaluation of the afferent system using sensations of bladder filling seems not to be very accurate in cauda equina injury patients. Moreover, this information could not distinguish complete or incomplete injuries. EPT determination in the bladder showed reproducible results. On the basis of these data, we concluded that bladder EPT can be used to confirm the results of the bethanechol test and can be used to evaluate changes in the bladder sensory innervation.

In patients with a cauda equina injury, the functional state of the afferent nervous system can be clinically evaluated by the combination of the results of the bethanechol test and bladder EPT. These tests should be included in the neurological work-up. Bladder filling sensations seems to be inconsistent for this group of patients.



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Underactive detrusor: Behavioural training and drugs

JJ Wyndaele

Behavioural treatment is often used but little studied. Out of common sense it can be important though and can start by better understanding what can be the cause of symptoms one develops. Voiding diary can help to restore better awareness, pick up signals of desire to void and help reinforce them.

Regulating drinking habits, timely or timed voiding , repeating voiding, treatment of constipation, can help to restore micturition contraction.

Drugs

Current standard treatment is with muscarinic receptor agonists (e.g. betanecol) or cholinesterase inhibitors. We have good results in a combined very active reeducation process with these drugs and they have been shown to improve sensory signaling in the LUT in idiopathic cases. The selection of patients likely to respond to subcutaneous betanecol was possible on the basis of electrosensation threshold measurement. Overall the level of evidence for drugs use so far remains low in literature. The same goes for drugs aimed at lowering urethral resistance as alfa adrenoreceptor antagonists and relaxing drugs for striated muscles. A real problem is the inclusion criteria and the different dosages used to permit proper conclusions.

In future other approaches will be tried out: TRPV1 agonists and other TRP channel activators. Prostaglandin E 2 intravesically have been successful but side effects limit their use so far. More research is needed

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Underactive detrusor specific surgery

JJ Wyndaele

If more conservative and less invasive treatment fails surgery might be considered.

Several techniques have been used in the past but none was really successful, though sometimes a bladder awareness of desire to void occurred: resection of part of bladder wall, wrapping of bladder wall.

Latissimus dorsi muscle transposition has been successfully used by some groups

Bladder tissue engineering, cell therapy is studied and future will show what the clinical applicability is .

For neurogenic underactivity the role of nerve transposition needs to be evaluated further.

If incontinence is due to urethral pressure deficiency and DU makes straining to void necessary, the implantation of an artificial sphincter may be an alternative solution to suburethral tape and catheterization.

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Voiding dysfunction due to underactive detrusor : can one restore function ?

Physiotherapy : Alexandra Vermandel

Micturition is often achieved with significant abdominal activity and simultaneous perineal EMG and uroflowmetry may show an interrupted or staccato flow pattern that coincides with rises in abdominal activity.

In our practice we use a stepped strategy moving from the least to most invasive therapy in the management of patients with bladder dysfunction, underactive detrusor. Most of them use the abdomen to generate voiding pressure.

Conservative measures, such as voiding behavior modification should be tried. If conservative fails to significantly improve the symptoms of bladder dysfunction, directed therapy is used. These interventions include pelvic floor techniques, biofeedback, EMG-flow.

First we review the patient voiding habits, identify habits that deviate from the normal voiding process, and clearly describe to patient the way in which their voiding differs from the normal process. After the initial approach providing information about normal voiding habits to the patient we give advise that involves voiding behavior, fluid intake modification including times voiding schedules and treatment of constipation, if present. We set an individualized voiding regimen to establish regular voiding habits. This most often entails a timed voiding schedule with frequent voids scheduled every two to three hours during the day. Document the voiding process in a diary after implementation of the voiding behavior modification. This voiding diary should include the time and volume of each void, the time of each incontinent episode, fluid intake, the time of each bowel movement, postvoiding residual volumes.

Biofeedback of the urethral sphincter muscles can be considered as treatment options for functional bladder outlet obstruction, respectively. An underactive detrusor related at a dysfunctional voiding refers to an inability to fully relax the urinary sfincter of pelvic floor during voiding .

Biofeedback therapy teaches patients how to identify and control the muscle groups involved in voiding. It can utilize non-invasive urodynamic monitoring, such as uroflow measurement and adhesive pads on the perineum to measure sphincter activity, to measure, record, and provide immediate feedback to the patient about their bladder function. The patient can observe real-time visual displays of urinary flow and electromyography (EMG) activity. The visual feedback allows the patient to become aware of and gain control over their bladder function by teaching the patient to learn how to voluntarily relax or contract their sphincter and pelvic musculature, thus modifying the observed measurements. The patient can observe real-time visual displays of urinary flow and electromyography (EMG) activity and modify these measurements by changes in the contraction or relaxation of the sphincter or pelvic floor muscles and so activate the micturition reflex.

Clearly further controlled studies of the various intervention for underactive detrusor in adults are needed.



Notes

Record your notes from the workshop here