Vessie Hypoactive Evaluation et traitement

Gérard Amarenco
Neurogenic Underactive Bladder

• Definition and pathophysiology
• Etiologies
• Evaluation
• Treatments
DIRECTIONS: Please check the box that best describes your bladder symptoms or impact of your bladder symptoms.

1. In the past week, how often was the feeling of urge to urinate but could not go urinated?
   - Three or more times
   - Two times
   - One time
   - None of the time

1. In the past week, on a usual night, how often did you wake up in the night to urinate?
   - Three or more times
   - Two times
   - One time
   - None of the time

1. In the past week, during the day, how often did you have to urinate again after you just urinated?
   - All of the time
   - Most of the time
   - Some of the time
   - None of the time

1. In the past week, how often have you had to strain/push to empty your bladder?
   - All of the time
   - Most of the time
   - Some of the time
   - None of the time

1. In the past week, during the day, how strong was the feeling that you did not empty your bladder after you urinated?
   - Extremely strong
   - Moderately strong
   - A little strong
   - Not at all strong

**SCORE**

3 2 1 0

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**UAB-q™**

http://www.underactivebladder.org

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**Detrusor Underactivity: Pathophysiological Considerations, Models and Proposals for Future Research. ICI-RS 2013**

Gommert A. van Koeveringe¹,², Kevin L.J. Rademakers¹, Lori A. Birder², Cees Korstanje³, Firouz Daneshgari⁴, Michael R. Ruggieri⁵, Yasuhiko Igawa⁶, Christopher Fry⁷, and Adrian Wagg⁸
Definition of Underactive Bladder (UAB)

• Clinical concept: UAB ≠ UAD (likewise OAB ≠ OAD)

• Underactive bladder: a voiding disorder (difficulty with bladder emptying)

• Definition of UB => inability to produce an effective voiding contraction sufficient to empty the bladder

• Various symptoms: weak stream, straining, feeling of incomplete emptying, hesitancy to start the stream, intermittency. Sometimes: poor sensation of bladder filling, infrequent voiding, frequency.

• Patients may have a high post void residual (PVR)
Definition of Detrusor underactivity (DUA)

- In contrary, DUA is Urodynamic concept (UD diagnostic tool for DUA)
- Defined (ICS) as “a contraction of reduced strength and/or duration, resulting in prolonged bladder emptying and/or failure to achieve complete bladder emptying within a normal time span”
- There are no recognized diagnostic criteria. Definition of “normal” strength of contraction and contraction duration are not specified.
- DUA can be secondary to BOO / decrease contractility (clear definitions)

**BOOI = PdetQmax-2Qmax**

**BCI= PdetQmax+5Qmax**

Neurogenic Underactive Bladder: a voiding disorder observed in neurogenic conditions

Apart from neurological causes, many other etiologies:

- chronic bladder outlet obstruction (BPH, prolapse, female surgery)
- metabolic diseases (diabetes)
- adverse effects of medications (anticholinergic drugs, antidepressant treatment, smooth muscle relaxants, spasmylytics)
- «lazy bladder»
- forced detrusor: overdistension following important retention > 1l
- age-related changes (affecting both detrusor muscle and central/peripheral innervation)
- myogenic changes: primary («visceral myopathy») or secondary to BOO (BPH ...) / fibrosis/collagen deposition
- idiopathic impaired detrusor contractile function
The different mechanisms of DUA:

myogenic / efferent / afferent / central control alterations
Pathophysiology: neurogenic alterations leading to UAB/UAD can be observed at different levels

- Decrease of facilitator influx from the brain
- Lesion of spinal pathways carrying influx from the brain
- Lesion of sacral center (micturition reflex integration)
- Lesion of efferent nerves leading to detrusor contraction

Increased CNS inhibition

- Lesion of spinal pathways carrying sensory influx to the brain

Dysfunction of peripheral sensory mechanisms

Afferent nerves have a fundamental role in initiating and maintaining bladder contraction.
Neurogenic Underactive Bladder

• Definition and pathophysiology
• Etiologies
• Evaluation
• Treatments
Central nervous system

Nervous system splitted in central/peripheral levels

Peripheral nervous system

Includes: plexus, roots, sacral spinal cord, peripheral nerve ( pudendal nerve)
Upper motor neuron lesions

Overactive detrusor

Sacral Parasympathetic center

Lower motor Neuron lesions

Underactive detrusor
Central nervous system

Brain lesions:
- stroke
- Parkinson
- tumors
- infectious (abscess, encephalitis,...)
- trauma

Spinal cord lesions:
- vascular
- trauma (SCI)
- tumors
- infectious (abscess, myelitis,...)
- MS
- arthrosis

Etiologies of underactive bladder in central nervous system lesions

Peripheral nervous system

Includes: plexus, roots, sacral spinal cord, peripheral nerve (pudendal nerve)
Prevalence and Impact of Urinary Symptoms Among Community-Dwelling Stroke Survivors

K.R. Britain, MA; S.I. Perry, PhD; S.M. Peet, PhD; C. Shaw, PhD; H. Dallosso, PhD; R.P. Assassa, MRCOG; K. Williams, BA; C. Jagger, PhD; J.F. Potter, DM, FRCP; C.M. Castleden, MD, FRCP

Results—A 70% response rate was achieved with the return of 10,226 questionnaires. Prevalence of reported stroke was 4% (n=423). Prevalence of urinary symptoms was 34% (n=3,197). Overall, stroke survivors had a higher prevalence of symptoms than the nonstroke population (64% to 32%, respectively). These symptoms were reported to have more of an effect on the lives of the stroke survivors compared with the nonstroke population even when adjusted for age and sex differences. This reported impact was not related to the stroke per se but to the severity of the urinary symptoms.

- OAB: urge / frequency / urge incontinence / nycturia
- voiding dysfunction 40%: slow stream / urinary retention

Problem of associated lesion (BPH) observed in this population
Voiding symptoms (poor force of stream, hesitancy, incomplete emptying) are seen in 17% to 27% of cases. Problem of associated lesion (BPH) observed in this population.
Epidemiology:
80% of SCI patients have urinary disorders

Characteristics of urinary symptoms:
- OAB: 80%
- Voiding dysfunction-retention (complete or incomplete): 80%

Impacts:
- Quality of life ...
- Urological complications: stones, infections, renal failure (more than 50% .. without specific management)
Other spinal lesions

Transverse myelitis
Chiari malformation
Syringomyelia
Spinal tumors
Cervical stenosis with myelopathy

... can lead to UAB/UAD
LUTS are observed in 32 to 97 %
(Onset : 6 years (5 to 10 years))

LUTS can reveal the onset of MS in 10%

LUTS typology :
OAB = 37% to 99%
Urinary retention = 34% to 79%

Correlations LUTS and :
- pyramidal signs – OAB
- EDSS - OAB

Table 1: Clinical presentation of VUD in MS

<table>
<thead>
<tr>
<th>Author, level of proof (LP)</th>
<th>Number of patients</th>
<th>Mean duration of MS (years)</th>
<th>Time since onset of VD (years)</th>
<th>Prevalence of urgency (%)</th>
<th>Prevalence of pollakiuria (%)</th>
<th>Prevalence of incontinence/urgency (%)</th>
<th>Prevalence of dysuria (%)</th>
<th>Prevalence of urinary retention (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amarenco, 1995, LSP1</td>
<td>225</td>
<td>13.3</td>
<td>7.8</td>
<td>72</td>
<td>42</td>
<td>63</td>
<td>46</td>
<td>24</td>
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<td>Andersen, 1976, LSP2</td>
<td>62</td>
<td>12.2</td>
<td>4.9</td>
<td>71</td>
<td>38.5</td>
<td>50</td>
<td>12</td>
<td>32.7</td>
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<tr>
<td>Awad, 1984, LSP2</td>
<td>47</td>
<td>16</td>
<td>U</td>
<td>85</td>
<td>65</td>
<td>72</td>
<td>36</td>
<td></td>
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<tr>
<td>Bemelmans, 1991, LSP2</td>
<td>40</td>
<td>5.4</td>
<td>U</td>
<td>85</td>
<td>82</td>
<td>63</td>
<td>49</td>
<td>34</td>
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<tr>
<td>Betts, 1993, LSP1</td>
<td>170</td>
<td>12</td>
<td>6</td>
<td>85</td>
<td>82</td>
<td>63</td>
<td>49</td>
<td>34</td>
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<tr>
<td>Bradley, 1978, LSP4</td>
<td>90</td>
<td>U</td>
<td>U</td>
<td>86</td>
<td>60</td>
<td>60</td>
<td>28</td>
<td>20</td>
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<tr>
<td>de Ridder, 1998, LSP2</td>
<td>30</td>
<td>U</td>
<td>U</td>
<td>36.6</td>
<td>36.6</td>
<td>80</td>
<td>25</td>
<td>8.3</td>
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<td>Eardley, 1991, LSP2</td>
<td>24</td>
<td>11</td>
<td>U</td>
<td>41.6</td>
<td>41.6</td>
<td>69.1</td>
<td>25</td>
<td>8.3</td>
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<tr>
<td>Gallien, 1998, LSP1</td>
<td>149</td>
<td>13</td>
<td>6</td>
<td>69.1</td>
<td>67.7</td>
<td>69.1</td>
<td>79.5</td>
<td>52</td>
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<tr>
<td>Giannantoni, 1998, LSP1</td>
<td>116</td>
<td>14.5</td>
<td>7.1</td>
<td>99</td>
<td>99</td>
<td>99</td>
<td>79.5</td>
<td>52</td>
</tr>
<tr>
<td>Goldstein, 1983, LSP4</td>
<td>86</td>
<td>U</td>
<td>U</td>
<td>32</td>
<td>32</td>
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<td>49</td>
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<td>Gonor, 1985, LSP2</td>
<td>64</td>
<td>13</td>
<td>4.6</td>
<td>70</td>
<td>48</td>
<td>56</td>
<td>30</td>
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<tr>
<td>Hennessey, 1999, LSP1</td>
<td>221</td>
<td>U</td>
<td>U</td>
<td>71</td>
<td>76</td>
<td>66</td>
<td>48</td>
<td>73.8</td>
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<td>Kasabian, 1995, LSP2</td>
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<td>18</td>
<td>U</td>
<td>44</td>
<td>66</td>
<td>66</td>
<td>38</td>
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<tr>
<td>Koldewijn, 1995, LSP1</td>
<td>211</td>
<td>6.3</td>
<td>U</td>
<td>38</td>
<td>38</td>
<td>38</td>
<td>26</td>
<td>27</td>
</tr>
<tr>
<td>Philp, 1981, LSP2</td>
<td>52</td>
<td>10</td>
<td>5</td>
<td>61</td>
<td>59</td>
<td>47</td>
<td>49</td>
<td>49</td>
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<tr>
<td>Porru, 1997, LSP1</td>
<td>120</td>
<td>0.1 – 9</td>
<td>U</td>
<td>36</td>
<td></td>
<td></td>
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</tbody>
</table>

VUD, vesicourethral disorder; LSP, ANAES level of scientific proof [1]; U, Unknown; DSD, detrusor and sphincter disorders; VD, voiding dysfunction.
Dysautonomia

Alterations of Autonomic Nervous System

Orthostatic hypotension
Etiologies of underactive bladder in peripheral nervous system lesions

Sacral roots

Peripheral neuropathies

Sacral plexus injury (traumatic, radiotherapy, tumors)

Sacral tumors

Disk herniation

Cauda equina and conus tumors

Sacral myelitis (herpes zoster, Lyme …)

underactive bladder is more often secondary to a lesion of the peripheral nervous system (than central lesion)
TABLE 2. LUTS in diabetic women with/without MS

<table>
<thead>
<tr>
<th>Variables</th>
<th>Non-MS group (n = 272)</th>
<th>MS group (n = 246)</th>
<th>P value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Storage symptom score</td>
<td>3.8 ± 3.4</td>
<td>5.4 ± 3.7</td>
<td>&lt;0.001b</td>
</tr>
<tr>
<td>Frequency</td>
<td>1.1 ± 1.7</td>
<td>1.2 ± 1.7</td>
<td>0.58</td>
</tr>
<tr>
<td>Urgency</td>
<td>1.1 ± 1.7</td>
<td>2.1 ± 1.8</td>
<td>&lt;0.001a</td>
</tr>
<tr>
<td>Nocturia</td>
<td>1.6 ± 1.3</td>
<td>2.2 ± 1.3</td>
<td>0.002b</td>
</tr>
<tr>
<td>Voiding symptom score</td>
<td>2.6 ± 4.3</td>
<td>3.8 ± 5.1</td>
<td>0.06</td>
</tr>
<tr>
<td>Incomplete emptying</td>
<td>0.8 ± 1.5</td>
<td>1.2 ± 1.7</td>
<td>0.05</td>
</tr>
<tr>
<td>Weak urinary stream</td>
<td>0.6 ± 1.5</td>
<td>1.0 ± 1.8</td>
<td>0.08</td>
</tr>
<tr>
<td>Intermittency</td>
<td>0.6 ± 1.5</td>
<td>1.2 ± 1.8</td>
<td>0.003b</td>
</tr>
<tr>
<td>Hesitancy</td>
<td>0.5 ± 1.3</td>
<td>0.4 ± 1.1</td>
<td>0.11</td>
</tr>
<tr>
<td>Total symptom score</td>
<td>6.5 ± 6.5</td>
<td>9.2 ± 7.6</td>
<td>0.001a</td>
</tr>
<tr>
<td>LUTS score (%)</td>
<td></td>
<td></td>
<td>0.001b</td>
</tr>
<tr>
<td>Less than 8</td>
<td>186 (69.4)</td>
<td>138 (56.1)</td>
<td></td>
</tr>
<tr>
<td>8–19</td>
<td>62 (23.1)</td>
<td>80 (32.5)</td>
<td></td>
</tr>
<tr>
<td>20 or greater</td>
<td>20 (7.5)</td>
<td>28 (11.4)</td>
<td></td>
</tr>
<tr>
<td>Quality-of-life score</td>
<td>1.6 ± 2.1</td>
<td>3.3 ± 2.4</td>
<td>&lt;0.001b</td>
</tr>
<tr>
<td>IUSS score</td>
<td>0.8 ± 1.2</td>
<td>1.6 ± 1.2</td>
<td>&lt;0.001c</td>
</tr>
<tr>
<td>OAB, n (%)</td>
<td>74 (27.2)</td>
<td>128 (52.0)</td>
<td>&lt;0.001c</td>
</tr>
<tr>
<td>Urinary incontinence, n (%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress incontinence</td>
<td>30 (11.0)</td>
<td>22 (8.9)</td>
<td>0.22</td>
</tr>
<tr>
<td>Urge incontinence</td>
<td>49 (18.0)</td>
<td>54 (21.9)</td>
<td>0.58</td>
</tr>
<tr>
<td>Uroflowmetry</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Voided volume (ml)</td>
<td>199.5 ± 85.2</td>
<td>190.3 ± 79.4</td>
<td>0.35</td>
</tr>
<tr>
<td>Peak flow rate (Qmax, ml/sec)</td>
<td>13.9 ± 7.2</td>
<td>13.0 ± 7.3</td>
<td>0.54</td>
</tr>
<tr>
<td>PVR (ml)</td>
<td>74.3 ± 30.5</td>
<td>76.0 ± 27.3</td>
<td>0.92</td>
</tr>
<tr>
<td>PVR ≥100 ml, n (%)</td>
<td>16 (7.7)</td>
<td>26 (12.3)</td>
<td>0.33</td>
</tr>
</tbody>
</table>

Tai HC et al, JCEM 2010 1143-1150

In DM, incidence of underactive diabetic bladder ranged 17% - 72%
Underactive Bladder

- Definition and concepts
- Etiologies
- Impact
- Evaluation
- Treatments
UAB: various impacts: urological alterations, quality of life bothersome symptoms, renal impairment, chronic Valsalva voiding hernia, vaginal prolapse, haemorrhoids
Underactive Bladder

• Definition and concepts
• Pathophysiology
• Etiologies
• Evaluation
• Treatments
Neurologic examination: in order to track down neurogenic etiology

Loss of touch sensation

Sensory function is evaluated by testing the lumbar-sacral dermatomes

Motor function is evaluated by testing the muscle strength and tone

Anal tone

Babinski sign

BC reflex

Muscle strength (plantar flexion), tendinous reflexes ...
Underactive bladder: clinical arguments for a neurogenic etiology

• association of urinary AND anorectal AND sexual disorders (same levels of motor/sensory integration-innervation)

• urological causes eliminated: in case of voiding dysfunction -> obstruction (BPH, prolapse)

• urinary disorders typology:
Uro-gynecological evaluation: associated lesions?

- BPH
- Prostate cancer
- Obstructive lesions in female
- Urethral stenosis
- Hemorrhoids
- Prolapse
- UTI
- Chronic Valsalva voiding leading to hernia, vaginal prolapse, haemorrhoids
- Bedsores
Cystometry

- to affirm DUA
- to precise pathophysiology
- evaluation of the prognosis

\[
BOOI = P_{det}Q_{max} - 2Q_{max}
\]

\[
BCI = P_{det}Q_{max} + 5Q_{max}
\]

Detrusor contractility

- Undercontractile: \(p_{det}Q_{max} + 5Q_{max} < 100\)
- Normal: \(100 < p_{det}Q_{max} + 5Q_{max} < 150\)
- Overcontractile: \(p_{det}Q_{max} + 5Q_{max} > 150\)

Electromyography

Urethral Sphincter
m. Bulbocavernous

Anal Sphincter

Recording EMG

Denervation
Bulbocavernous Reflex

BCR tests the integrity of the sacral reflex arc.
**Evoked Potential**

SEP tests the integrity of the somatosensory (lemniscal) pathways.

- **Stimulation Penile Nerve**
- **Stimulation Clitoral Nerve**

Latency P40 (nL < 44 ms)

Record on Scalp with Scalp Needle or Surface Electrode
Averaging: 200
Motor E.P.

MEP tests the integrity of the motor pathways

Magnetic Stimulation

Descending

Motor Nerve

Muscle

TMS coil - high current
Magnetic field
Induced electric field

Trigger

Record

Amplitude (mV)

Time (ms)

Motor Evoked Potentials

TMS Pulses
Underactive Bladder

- Definition and concepts
- Pathophysiology
- Etiologies
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First: eliminate and treat Irritative lesions
Underactive Bladder Treatment

1. Fecal impaction, UTI, stone

2. Alpha-blockers, BTX sphincter

3. Self cath

4. Sacral neuromodulation

5. Surgery

6. Derivation

Graph showing EMG and Pves over time, and a graph showing cystometry with volumes and pressures.
To discriminate neurogenic and obstructive lesions: Urethral stent inserted within urethra

Underactive bladder
Underactive detrusor

Urinary retention

Urethral stent

Micturition +

Micturition -

Urological obstruction

Neurological voiding dysfunction

Reappearance of micturition = obstruction!
Neuromodulation

Sacral Neuromodulation in Diabetic Patients: Success and Complications in the Treatment of Voiding Dysfunction

David H. Daniels, Charles R. Powell, Matthew R. Braasch, and Karl J. Kreder*
Department of Urology, University of Iowa, Iowa City, Iowa

Posterior Tibial Nerve Stimulation in the Treatment of Voiding Dysfunction: Urodynamic Data

Vera Vandoninck,¹ Michael R. van Balken,¹ Enrico Finazzi Agrò,³ John P.F.A. Heesakkers,¹ Frans M.J. Debruyne,¹ Lambertus A.L.M. Kiemenei,² and Bart L.H. Bemelmans¹*

“Band-Aid” Antenna for Tibial Nerve Stimulation

Posterior tibial nerve stimulation

... few data
Parasympathomimetic drugs
(cholinomimetic drugs)

**Direct-acting**
These act by stimulating the nicotinic or muscarinic receptors.

- Choline esters
  - Acetylcholine (all acetylcholine receptors)
  - Bethanechol (M3 receptors)
  - Carbachol (all muscarinic receptors and some nicotinic receptor)
  - Methacholine (all muscarinic receptors)

- Plant alkaloids
  - Nicotine
  - Muscarine
  - Pilocarpine (M3 receptors)

**Indirect-acting**
cholinesterase inhibitors, or drugs that promote ACh release.

- Reversible cholinesterase inhibitors
  - Donepezil, Edrophonium, Neostigmine, Physostigmine, Pyridostigmine, Rivastigmine, Tacrine, Caffeine (non-competitive)
  - Huperzine A

- Irreversible cholinesterase inhibitors
  - Echothiophate, Isoflurophate, Malathion, ACh release promoters, Cisapride, Droperidol, Domperidone

**But**: no evidence based medicine / poor efficacy / important side effects => not used / recommended in clinical practice
• the gold standard in case of urinary retention
• safe and effective (specially with new devices)
• improve quality of life (decrease UI, allows specific treatment of OAB/OAD)
• CISC avoids urological complications: decrease renal risk, UTI
Specific surgery can help CIC!

Reconstructive hand surgery

Continent cystostomy
Urethral device with inside a turbine activated by magnetic remote control

- inFlow™ is normally replaced every 29 days

- Activator: a hand-held magnetic remote control required to operate the internal valve-pump mechanism in the inFlow device

- today, no available in many countries in Europe.

Just after activation, turbine, (pump) empties the bladder
The disposable introducer makes the inFlow easy to insert
To treat and PREVENT complications ...

D-Manose

Cranberry

E. Coli

( ATB : only symptomatic UTI +++ )
Cross-talk! Necessary to take in account anorectal disorders.

- Before irrigation
- After irrigation

Pyramid showing various treatment methods:
1. Stoma
2. Sacral Anterior Root Stimulation
3. Antegrade colonic irrigation
4. Sacral Nerve Stimulation
5. Transanal irrigation
6. Digital stimulation / suppositories / biofeedback
7. Diet and fluid / lifestyle alteration / laxatives or constipating drugs
A Look into the Future
Nerve rerouting: to reinnervate the neurogenic bladder

Concept: to join healthy nerves to injured nerves to improve function (to restore micturition)
Hyperpolarization-activated cyclic nucleotide–gated (HCN) channels are intermembrane proteins that serve as nonselective ligand-gated cation channels in the plasma membranes of heart and brain cells.

HCN channels are sometimes referred to as “pacemaker channels” because they help to generate rhythmic activity within groups of heart and brain cells.

There are several potential options for pharmacological intervention:

- centrally-acting agents such as opioid antagonists, dopamine agonists
- locally-acting agents such as nitric oxide donors
- agents targeting neurogenic deficits, such as cholinergic agonists, transient receptor potential agonists
- agents targeting myogenic deficits, including M1 agonists and potassium channel blockers
Fort-de-France
les ateliers pratiques de la SIFUD