Urinary Incontinence in the Elderly: Impact of Neurogenic bladder

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**Neurogenic Bladder (NGB)**

- Disorder of LUT function caused by an abnormality of innervation
- Not all patients with neurologic diseases & incontinence have NGB
- Patients without neurologic diseases may have incontinence due to subtle unrecognized neurologic abnormalities
Significance

- Neurogenic bladder plays an important role in our understanding of urinary incontinence in the elderly
- High prevalence of NGB in the elderly
- Underlying neurologic disorder can not only be the cause of incontinence, but also can pose a major obstacle to its treatment
<table>
<thead>
<tr>
<th>Condition</th>
<th>Prevalence</th>
<th>Incidence</th>
<th>Incontinence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s Disease</td>
<td>5,400,000¹⁹</td>
<td></td>
<td>60-70%²⁰</td>
</tr>
<tr>
<td>Stroke</td>
<td>4,500,000¹ (survivors)</td>
<td>795,000⁸ (600,000 new strokes)</td>
<td>36%¹²</td>
</tr>
<tr>
<td>Parkinson’s disease</td>
<td>1,000,000²</td>
<td>60,000²</td>
<td>27%¹³</td>
</tr>
<tr>
<td>Normal pressure hydrocephalus</td>
<td>750,000³</td>
<td>2 – 20 /million⁹</td>
<td>95%¹⁴</td>
</tr>
<tr>
<td>Multiple sclerosis</td>
<td>400,000⁴</td>
<td>10,000⁴</td>
<td>79%¹⁵</td>
</tr>
<tr>
<td>Spinal cord injury</td>
<td>276,000⁵</td>
<td>12,000¹⁰</td>
<td>81%¹⁶ (bladder dysfunction)</td>
</tr>
<tr>
<td>Lumbar Spinal Stenosis</td>
<td>8,500,000⁶</td>
<td></td>
<td>50%¹⁷ (bladder dysfunction)</td>
</tr>
<tr>
<td>Motor Neuron Disease</td>
<td>20,000⁷</td>
<td>5,600¹¹</td>
<td>36-50%¹⁸</td>
</tr>
</tbody>
</table>

¹ CDC; ² Parkinson’s Disease Foundation; ³ Ettinger; Overview of NPH. 2015; ⁴ Hersh; Multiple Sclerosis. 2014; ⁵ SCIMS; ⁶ Matt, Spine Lumbar; Spine U, 2009; ⁷ Mehta, Surveillance Summaries. 2014; ⁸ strokecenter.org; ⁹ Radford, NPH. 2016; ¹⁰ NSCIS; ¹¹ UCSD; ¹² Brittain, Stroke and Incontinence. 1998; ¹³ Siegl, Pflege Z. 2013; ¹⁴ Shprecher, Current Neurology and Neuroscience Reports. 2008; ¹⁵ Khalaf, International Journal of MS Care. 2015; ¹⁶ Ginsberg, AJMC. 2013; ¹⁷ Wyndaele, Neurologic Urinary and Fecal Incontinence; ¹⁸ Nubling, ALS Journal. 2014; ¹⁹ alz.org; ²⁰ b&bf
Impact of Neurologic Disease on Incontinence

- **Reduced cognitive function:**
  - Decreased awareness & concern about bladder sensations & events
  - Loss of voluntary sphincter control
  - Difficulty complying with treatment strategies

- **Reduced mobility**
  - Getting to bathroom in time

- **Poor hand function**
  - Intermittent catheterization, appliances & pads
State-of-the-Art Knowledge

- Pathophysiology of incontinence
- Physiology of micturition
- Neurophysiology of micturition
- Efficacy of incontinence treatments
Causes of Incontinence

- Bladder (common)
  - Involuntary detrusor contractions (IDO, NDO)*
  - Cognitive or sensory abnormalities > uncontrollable voiding due to lack of awareness or concern
  - UTI

- Sphincter (rare)
  - Neurogenic (e.g. thoraco-lumbar lesions)*
  - Non-neurogenic (e.g. ISD*, urethral hypermobility)

- Extra-urethral (very rare) e.g. fistula, ectopic ureter

*neurogenic etiology
Physiology of Micturition

Storage

Voiding

Stop

Pure

Pdet

Q

EMG

1

2

3

4

5

6

7

8
Normal Uroflow

Detrusor contraction

Command to void

No rise in pdet during filling

Sphincter relaxation
Sacral Micturition Center

• Primitive micturition reflex center

• Parasympathetic (pelvic nerve) → detrusor contraction

• Somatic (pudendal nerve) → sphincter contraction

Detrusor external sphincter dyssynergia
Sacral Spinal Lesions

--- = parasympathetic
--- = somatic

PMC
T12 - L1
S2-4
Sacral Spinal Lesions

- Loss of micturition reflex
- Detrusor areflexia
- Smooth & striated muscles of the urethra keep the sphincter closed

*Urinary retention*
Detrusor areflexia

Urinary retention

No sensation during bladder filling

Strains to void

Detrusor areflexia
Thoraco-lumbar Sympathetic Lesions

- = parasympathetic
- = sympathetic

blocks transmission

contracts sphincter

PMC

T 12-L1

S 2-4
Thoraco-lumbar Lesions

- Loss of smooth muscle sphincteric function
- Loss of striated sphincter control
- Detrusor areflexia or overactivity
- +/- low bladder compliance

*Paradoxical urinary retention & sphincteric incontinence*
Sphincteric incontinence

Steep rise in vesical pressure, but no detrusor contraction

Low bladder compliance
Suprasacral Spinal Lesions

parasympathetic
somatic
sympathetic
central
C fiber
Detrusor External Sphincter Dyssynergia

- Involuntary sphincter contraction during involuntary detrusor contraction
- Due to a neurologic lesion between sacral & pontine micturition center
- Poses serious urologic risks – UTI, urosepsis, stones, hydronephrosis, renal failure
Involuntary detrusor contraction

Obstruction due to sphincter contraction

Involuntary detrusor contraction

Involuntary sphincter contraction

EMG 168
Cortical Centers

- Conscious awareness of bladder sensations & events
- Assess the propriety, social context & timing of micturition
- Voluntary control of micturition
Suprapontine Neurologic Lesions

- Micturition reflex usually intact
- When micturition is affected, there is usually loss of voluntary control
- +/- loss of awareness & concern
- +/- loss of voluntary sphincter control
Suprapontine Lesions

parasympathetic
somatic
sympathetic
central
C fiber
Involuntary detrusor contraction

No awareness of bladder filling or urge to void

Involuntary detrusor contraction
Suprapontine Neurologic Lesions

- The "neurogenic bladder" poses no threat to health unless there is an underlying urologic condition such as
  - Urethral obstruction (prostate, prolapse)
  - Prostate or bladder cancer
  - Incomplete bladder emptying
  - Recurrent UTI
Pathophysiology of NGB

- **Suprapontine lesions:**
  - Loss of voluntary control

- **Suprasacral lesions:**
  - Detrusor sphincter dyssynergia

- **Thoracolumbar lesions**
  - Neurogenic sphincteric incontinence
  - Detrusor areflexia or overactivity

- **Sacral lesions:**
  - Detrusor areflexia

Knowledge Gaps & Research Opportunities
Understanding Urgency and Involuntary Detrusor Contractions

• Type I: patient has urgency and/or urge incontinence, but no IDC at urodynamics

1. Command to void

2. Relaxes sphincter

3. Voluntary detrusor contraction

1st urge = 80 ml

severe urge = 105 ml

Capacity = 346 ml

FSF = 66 ml

Voids normally
Understanding Urgency and Involuntary Detrusor Contractions

- **Type I:** symptoms of overactive bladder, no IDC at urodynamics
- **Type II:** IDC present; patient is aware, can contract his sphincter, abort the detrusor contraction and prevent incontinence
1. Involuntary detrusor contraction
2. Involuntary sphincter relaxation
3. Incontinence
1. sudden urge
2. involuntary detrusor contraction
3. voluntarily contracts sphincter
4. no incontinence, urge subsides
5. aborts detrusor contraction
OAB Understanding Urgency and Involuntary Detrusor Contractions

- **Type I:** symptoms of overactive bladder, no IDC at urodynamics
- **Type II:** IDC present; patient is aware and can abort the IDC
- **Type III:** IDC patient aware, cannot abort but can temporarily maintain continence by contracting the sphincter; once the sphincter fatigues is incontinent.
1. sudden urge
2. involuntary detrusor contraction
3. voluntarily contracts sphincter
4. Aborts incontinence
5. can't hold any longer
6. incontinent
Understanding Urgency and Involuntary Detrusor Contractions

- **Type I:** symptoms of overactive bladder, no IDC at urodynamics
- **Type II:** IDC present; patient is aware and can abort the IDC
- **Type III:** IDC patient aware, cannot abort but can temporarily maintain continence by contracting the sphincter
- **Type IV:** IDC, no awareness or control
1. No awareness of bladder filling or urge to void

2. Involuntary detrusor contraction

3. Incontinent
Detrusor hyperreflexia with Impaired Detrusor contractility (DHIC)

Type 3 OAB
Impaired Detrusor Contractility

Resnick & Yalla, Detrusor hyperactivity with impaired contractile function. An unrecognized but common cause of incontinence in elderly patients. JAMA (1987),12;257(22):3076
1. Urgency
2. Involuntary detrusor contraction
3. pdet@Qmax = 28 cm H₂O
4. Qmax = 5 mL/S
5. VOID: 5/180/570/4
Knowledge Gaps & Research Opportunities

- What stimuli > urgency, IDO & NDO?
  - wall tension, pdet, volume?
  - constituents in urine, caffeine?
  - neurotransmitters, receptors?
- Is IDO just a subtle NDO?
- What neural pathways, transmitters & receptors are involved in aborting urgency & DO
Knowledge Gaps & Research Opportunities (cont’d)

- Etiology & Rx of impaired detrusor contractility & DHIC

- Better Rx for:
  - Urgency & DO
  - Detrusor sphincter dyssynergia