



Neuro-urology for the Gynaecologist Workshop 14 Monday, 23 August 2010, 14:00-17:00

Time	Time	Topic	Speaker
14:00	14:10	Introduction	John Lavelle
14:10	14:20	Introduction of Cases	John Lavelle
14:20	14:40	Presentation of Neurogenic bladder	Donna Deng
14:40	15:00	Recognition and complications of Neurogenic bladder	Craig Comiter
15:00	15:30	Discussion & Cases	
15:30	16:00	Break	
16:00	16:10	Principles and Practical Management of Neurogenic Bladder	Chris Smith
16:20	16:30	Discussion	
16:30	16:50	Office based care of the neurological patient	Michael Kennelly
16:50	17:00	Discussion	

Aims of course/workshop

Neurogenic bladder is a finding that is obvious in patients with Spinal Cord Injury, and Multiple Sclerosis, however the neurogenic bladder may also be found in patients with prior pelvic, or vascular surgery, or may have occult neurological disease presenting with incontinence or urinary retention. The aim of this course is to provide urologists and gynecologists, who are not familiar with managing neurogenic bladder, the information to recognize, diagnose and the principals of management of a neurogenic bladder.

Objectives:

1. Recognition the characteristics of a neurologically injured bladder.
2. Correct diagnosis a neurogenic bladder and its complications.
3. Outline the primary principles of neurogenic bladder management

Educational Objectives

Since the 2010 meeting is a joint ICS & IUGA meeting, this program would be useful to urologists and gynecologists and also the allied health professionals dealing with generally able bodied patients. The primary purpose of the workshop is to help these physicians recognize the hallmarks of neurological injury to the bladder and to provide clear and concise information about the principles of management of the neurogenic bladder. It is intended to be a practical workshop with clinical examples of the various conditions and their manage

Key Learning Points and Take home messages:

1. Neurogenic bladder findings may occur in patients, without devastating neurological disease or injuries
2. Neurogenic bladder may lead to significant morbidity in affected patients.
3. The management of neurogenic bladder follows a series of clear principles

Workshop 17: Dr. John Lavelle

Discussion of Cases: Topics in presentation and consideration during the workshop.

1. Painful voiding
2. Severe incontinence
3. Urinary retention
4. "Stress Incontinence" after the Pubo-vaginal Sling.

Presentations of the Neurogenic Bladder

Case Scenario #1

58yo woman underwent radical hysterectomy for cancer. She required indwelling foley catheter for 1 week due to inability to urinate. She was able to urinate more spontaneously after the first week, but the stream was weak and slow. It is now 9 months after surgery and she is becoming more bothered by her incontinence and has been referred to you for further evaluation and possible anti-incontinence procedure.

Symptoms in patients with neurogenic bladder can include all of the following:

- Frequency
- Urgency
- Nocturia
- Urinary incontinence
- Weak stream
- Hesitancy
- Incomplete Emptying
- Retention

The past medical history should identify any neurologic condition (congenital, traumatic, metabolic, degenerative) that can affect lower urinary tract function. Patients should therefore be questioned about low back pain, previous spinal cord injury or surgery, Parkinson's disease, multiple sclerosis, cerebrovascular accident, diabetes and other pelvic surgeries. The use of medications that can affect lower urinary tract function, particularly those with anticholinergic or α -adrenergic effects, should be documented. A family history should question for such disorders as epilepsy, Huntington's disease, and degenerative conditions of the CNS.

It is important to elicit associated neurologic symptoms such as visual changes, sensory changes, motor weakness, or gait abnormalities because they may suggest a neurologic cause for the urinary symptoms (e.g., multiple sclerosis).

The motor examination primarily evaluates motor strength, but the presence of abnormal movements and signs of denervation (e.g., muscle atrophy) should also be assessed. Generally, muscle hypotonicity results from peripheral nerve injuries and myopathies, whereas hypertonicity results from suprasegmental lesions.

Sensory alterations in the genital or perianal area, fecal incontinence or constipation, Patterns of sensory loss are important because they often follow the segmental distribution of one or more spinal nerve roots and therefore may help to localize the level of neurologic deficit. Important dermatomes to remember are T10, umbilicus;

L3, front of knee; and S3–S5, perineal/perianal skin. The anterior portions of the labia are supplied by roots from the thoracolumbar spinal cord, whereas the posterior portions of the labia and perianal area are supplied by sacral roots. Disorders of orgasm may be suggestive of impairment in the innervation of the pelvic organs and the lower urinary tract.

Discrete neurologic lesions generally affect the filling/storage and emptying/voiding phases of lower urinary tract function in a relatively consistent fashion. This fashion is dependent on the area(s) of the nervous system affected; the physiologic function(s) and the contents and location of the area(s) affected; and whether the lesion or process is destructive or irritative. The acute dysfunction produced may differ, for a variety of reasons, from the chronic one.

Voiding dysfunction after pelvic plexus injury occurs most commonly after abdominoperineal resection and radical hysterectomy. The true incidence of neurogenic vesicourethral dysfunction after various types of pelvic surgery is unknown, because there are no prospectively studied series of patients with preoperative and postoperative urodynamic evaluation. The incidence has been estimated to be 20% to 68% of patients after abdominoperineal resection, 16% to 80% after radical hysterectomy, 20% to 25% after anterior resection, and 10% to 20% after proctocolectomy (1). These are estimates drawn from past literature, and the current incidence is most likely significantly lower, owing to the use of nerve-sparing techniques during these types of pelvic surgery. It has been estimated, however, that in 15% to 20% of affected individuals, the voiding dysfunction is permanent (2).

When permanent voiding dysfunction occurs after radical pelvic surgery, the pattern is generally one of a failure of voluntary bladder contraction, or impaired bladder contractility, with obstruction by what seems urodynamically to be residual fixed striated sphincter tone, which is not subject to voluntarily induced relaxation. Often, the smooth sphincter area is open and nonfunctional. Decreased compliance is common in these patients, and this, with the "obstruction" caused by fixed residual striated sphincter tone, results in both storage and emptying failure. The patient often presents with urinary incontinence that is characteristically most manifest with increases in intra-abdominal pressure, and suggestive of stress urinary incontinence. Alternatively, patients may present with variable degrees of urinary retention.

References

1. Blaivas JG, Chancellor MB: Cauda equina and pelvic plexus injury. In Chancellor MB, Blaivas JG (eds): Practical Neurourology. Boston, Butterworth-Heinemann, 1995b, pp 155–164.
2. McGuire EJ: Clinical evaluation and treatment of neurogenic vesical dysfunction. In Libertino JA (ed): International Perspectives in Urology, vol 11. Baltimore, Williams and Wilkins, 1984, pp 43–56.

Presentations of the Neurogenic Bladder

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Suprapontine Lesions

- Reflex detrusor contractions
 - Loss of cerebral regulation of voiding
 - Normal coordinated sphincter function
 - Incontinence due to bladder overactivity
 - Sensation may be deficient or delayed
- Purposeful increase in sphincter activity during overactive detrusor contraction
 - Pseudo-dyssynergia
- Rarely detrusor areflexia

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Suprasacral Spinal Cord Lesion

- Detrusor Overactivity
 - Incontinence from DO – without sensation
- DSD common
 - Smooth sphincter synergy T6-S2
 - Smooth sphincter dyssynergia (above T6)
 - BOO from DSD can cause retention
- Bowel dysfunction
 - Overactive bowel, increased colonic wall activity
 - Anal sphincter tight
 - Stool retention
 - Incontinence due to impaction/constipation
- Autonomic dysreflexia (above T6)

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Neurogenic SUI

- Typical of T11-T12 injury
- Intrinsic sphincter deficiency
 - Also pelvic floor muscular weakness
- Treatment to increase resistance
 - Periurethral bulking – poor long term efficacy
 - Especially if CIC performed
 - Urethral sling
 - Fascial sling at bladder neck
 - Spiral sling / wrap-around
 - Expect CIC to continue

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Disease below S2

- Detrusor underactivity/areflexia
- Diminished compliance during filling
- Open smooth sphincter
 - Parasympathetic or sympathetic dysfunction
- Striated sphincter residual resting tone NOT under voluntary control
- Lack of SC mediated peristalsis of bowel
 - Mesenteric plexus peristalsis only
 - If pudendal neuropathy
 - Non contractile EAS, puborectalis, loss of rectal angle
 - Constipation and incontinence

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Interruption of peripheral reflex arc

- Similar to distal cord or nerve root injury
- Detrusor areflexia
- Diminished compliance
- Smooth sphincter incompetence
- Striated sphincter fixed residual tone without voluntary relaxation

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Subsacral Lesions
(Cauda Equina or Peripheral Nerves)

- Detrusor hypocontractility (acontractility)
 - Impaired emptying
- Paralysis of urethral sphincter/PFM
 - Loss of outflow resistance, SUI
- Lack of SC mediated peristalsis of bowel
 - Mesenteric plexus peristalsis only
 - If pudendal neuropathy
 - Non contractile EAS, puborectalis, loss of rectal angle
 - Constipation and incontinence
- Conus Lesion
 - Poor compliance with competent sphincter

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Routine care of the NGB woman

- Outcome of upper tracts related to combination of detrusor and sphincteric function
 - Hyper-reflexic detrusor detrimental to upper tracts when sphincter fails to relax simultaneously
 - Weak sphincter protects kidneys
 - But permits incontinence

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Likelihood of upper tract deterioration

- Suprapontine – rarely
 - Unless significant outlet obstruction
- Suprasacral lesions – at risk
 - Chronically elevated intravesical pressure
 - DSD
- Conus – at risk
 - Poor bladder compliance with competent sphincter

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Occult Neurological Disease

- Woman without history of neuropathy presents for evaluation of voiding dysfunction
- Suspicious if:
 - History of subtle non-urological neurological complaints associated with urinary sx
 - Numbness, tingling, eyesight changes, motor weakness
 - DO on urodynamics in young, nulliparous woman
 - Or sx of incontinence in young woman
 - Discoordinated detrusor and external sphincter activity
 - Neurological problem
 - Beware of learned behavioral dysfunctional voiding
 - Presence of unexplained retention
 - Especially with hypocontractile bladder

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Recognition of NGB in woman

- History, neurologic symptoms, neuro-uro exam
 - Sacral reflex examination
 - Sacral sensation
 - Anal tone
 - PF strength
- U/A, creatinine, upper tract study
 - Ultrasound, VCUG, DMSA (function)
- UDS

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Disk Disease

- Usually disk prolapse is posterolateral
 - Does not affect majority of cauda equina
 - 1%-15% with central disk prolapse
 - Compression of cauda equina
 - Thus disk prolapse anywhere in LS spine can interfere with parasympathetic and somatic innervation of LUT

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Disk Disease

- Mostly L4-L₅ or L₅-S₁
- Low back pain radiating in girdle-like fashion
- Loss of sensation/reflexes
 - Especially in perineum/perianal (S₂-S₄)
 - Lateral foot (S₁-S₂)

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Disk Disease – LUT dysfunction

- Anywhere from 27%-92%
 - Goldman and Appell, 2000
- Detrusor areflexia – 27%
 - Sx: difficulty voiding, straining
 - UDS: detrusor areflexia, normal compliance
 - Rarely DO – likely irritation of nerve roots
 - O'Flynn, 1992
- Treatment – urologic management
 - Laminectomy may not be helpful for LUT function

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Cauda Equina Syndrome

- Clinical scenario
 - Perineal sensory loss
 - Loss of voluntary control of anal and urethral sphincters
 - Loss of sexual responsiveness
- Secondary to central disk protrusion, other central spinal canal processes

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LUTS with central disc protrusion

- The classical features of acute cauda equina compression may be absent in patients with central lumbar disc protrusion.
- Painless urinary retention may be the only physical sign.
 - Often no other signs or symptoms suggesting an underlying neurological insult.
 - 2/3 recover urinary function with early intervention
 - Mosdal et al, Acta Neurochir, 1979
- High level of suspicion is necessary
 - Failure to intervene can lead to progression to full blown cauda equina syndrome

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Spinal Stenosis

- Narrowing of spinal canal, nerve root canal, intervertebral foramina
 - Nerve damage, ischemia, edema
 - Cervical cord compression to cauda equina syndrome
- Symptoms and UDS *usually* correspond to level and amount of spinal cord or nerve root damage
 - Cervical spondylitic spinal stenosis, DO or DUA
 - Depends on primary pathology affecting micturition neural axis compresses inhibitory reticulospinal tracts of myelopathy in posterior funiculus (proprioception)
- 50% improvement in LUT function with laminectomy

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Peripheral Neuropathy due to Iatrogenic Lesion (focal neuropathy)

- LUT dysfunction from damage to nerves innervating pelvic organs
 - Course of nerves through cauda equina, spinal nerve roots, sacral plexus, peripheral nerves
- Extensive pelvic surgery
 - APR, LAR, radical hysterectomy, Aorto-iliac surgery
- Damage to pelvic parasympathetic nerves
 - To bladder, genitalia

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Radical Pelvic Surgery

- Pelvic Plexus Injury
 - Radical hysterectomy, abdomino-perineal resection, proctocolectomy
 - 16%-80% with RH
 - Zullo, 2002
 - 20%-68% with APR
 - 20%-25% with anterior resection
 - 10%-20% with proctocolectomy
 - Blaivas and Chancellor, 1995
 - 15%-20% with permanent LUT dysfunction

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Radical Pelvic Surgery

- Parasympathetic decentralization
 - Increase in adrenergic innervation
 - Conversion of b3 relaxation to a-contraction
 - Sundin, 1977
 - Synaptic reorganization with new cholinergic excitatory inputs
 - Hanno, 1988
 - Urethral supersensitivity to alpha-adrenergic stimulation
 - Koyanagi, 1988, Nordling, 1981

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Radical Pelvic Surgery – Symptoms

- Storage and emptying failure
- Urinary incontinence
 - Most manifest with SUI
 - Leakage across distal sphincter
- Varying degrees of urinary retention
 - Inability to empty bladder
 - No true bladder contraction

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Radical Pelvic Surgery – UDS

- Failure of voluntary bladder contraction or impaired bladder contractility
- Decreased compliance
- Poor proximal urethral closure
 - Sympathetic/parasympathetic damage vs hydrodynamic effects of distal obstruction
- Obstruction by residual fixed striated sphincter tone
 - Loss of voluntary control of striated sphincter
 - Smooth sphincter open and nonfunctional
- Positive bethanechol supersensitivity test

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Radical Pelvic Surgery - Treatment

- Targeted history and PVR
 - to avoid secondary myogenic damage due to chronic retention/bladder distention
- Most dysfunctions are transient
- CIC is best initial treatment
 - Resist temptation to do something
 - Early rehab of LUT may improve voiding
- Normalization by 6-12 months
 - Blaivas and Chancellor, 1995

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Herpesvirus Infections

- Invasion of sacral DRG and posterior nerve roots
- Sx: urinary retention
- UDS: detrusor areflexia
 - Rytto, 1985
- Sx: Urinary incontinence
- UDS: detrusor overactivity
- Overall 4% with LUT dysfunction

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Herpesvirus Infections

pathophysiology and treatment

- Cystitis associated
 - Dysuria, frequency, retention, pyuria, hematuria
- Neuritis associated (sacral motor neurons)
 - Retention with detrusor areflexia
- Myelitis associated (meninges, SC)
 - Detrusor overactivity
 - Chen, 2002
- Treatment: supportive, reversible (CIC, pharmacotherapy)
- All patients returned to normal voiding within 2 months

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Herpes Zoster

- Urinary retention
 - Detrusor acontractility and lack of sensation
 - +/- overflow incontinence
 - Return to normal over 3 months
- Treat retention with CIC or foley
 - Avoid infection, chronic distention
 - Natural history is to regain bladder function during quiescent phase

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HIV

- Central/peripheral nervous system involvement
- Neurogenic voiding dysfunction = poor prognosis
- Sx vary with neural involvement
 - Retention with areflexia
 - OAB with detrusor overactivity
 - DSD from CNS involvement
- Overall, voiding symptoms are a modest problem in women with HIV, neuropathic dysfunction is rare and mostly in late stages of disease

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HIV suprapontine dysfunction

- HIV encephalopathy –
 - part of the acute HIV syndrome during seroconversion.
 - Associated with detrusor overactivity
- ADC (HIV-associated dementia complex, HAD)
 - Characterized by cognitive, motor, and behavioral features in adults
 - usually develops in advanced AIDS when CD4+ lymphocyte counts fall below 200 cells/mm³.
 - Associated with detrusor overactivity
- Minor cognitive motor disorder (MCMD) is now more common than ADC.
 - Since use of anti-retroviral therapy

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SLE

- Inflammation in connective tissues and small vessels of skin and systemic organs
 - Autoimmune in origin
 - Sakakibara, 2003
- Nervous system involvement in half (18%-75%)
 - Subacute myelopathy
 - Subacute and chronic encephalomyelopathy
- Sx and UDS depend on site of disease
 - Most common findings is detrusor overactivity, but also can see decreased urinary flow, detrusor underactivity, elevated PVR, DSD, sphincter denervation

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Reflex Sympathetic Dystrophy

- Severe pain with autonomic changes
 - Vasomotor disturbances
 - Follows traumatic injury
 - Unknown prevalence
- Sx are variable
 - Urinary retention, UI, SUI, urgency, frequency, nocturia
- UDS: DO, DESD, detrusor areflexia, hypersensitivity upon filling

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Radiation

- Voiding dysfunction after XRT
 - Early radiation cystitis
 - 70%
 - 4-6 weeks
 - Mostly storage dysfunction
 - Uds: reduced first desire to void, capacity, DO (33%)
 - Reduced compliance at 4-6 weeks, then recovers, then recurs at 10-12 weeks and persists
 - Fibrotic infiltration of muscle bundles, mast cells, focal SM degeneration, unmyelinated axon degeneration

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Multiple Sclerosis

- Demyelinating process most commonly involves lateral corticospinal (pyramidal) and reticulospinal columns of cervical cord
 - Voiding dysfunction and sphincter dysfunction common
 - Demyelination location
 - Cervical – nearly always
 - Lumbar 40%
 - Sacral 18%
 - Blaivas and Kaplan, 1988
- Rare upper tract damage
 - DSD, high Pdet, indwelling catheter
 - Wyndaele, 2005

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MS

- 50%-90% with voiding dysfunction
 - Presenting sx in 15%
- 37% - 72% with incontinence
 - Wyndaele, 2005
- Symptoms:
 - F/U in 31%-85%
 - Voiding sx, retention 2%-52%
- UDS: DO in 34%-99%
 - DSD in 30% (beware pseudo-dyssynergia – intact sensation)
 - Smooth sphincter synergy
 - Sphincter flaccid in 15%
 - Detrusor underactivity, areflexia in 12%-38%
 - Wyndaele, 2005

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MS and urinary retention

- Minority of patients with MS present with or develop urinary retention
 - Detrusor underactivity
 - Detrusor-sphincter dyssynergia
 - UTI

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Diabetes Mellitus

- Most common cause of peripheral neuropathy in Europe, North America
 - Prevalence: of DM in US: 1%-6%
 - Unselected patients do not report voiding dysfunction
 - When queried, 5%-59% admit voiding dysfunction sx
 - Wein and Rovner, 1999
 - Unclear if sx caused by DM

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“Classic” DM

- Sensory and motor neuropathy
 - Motor neuropathy = impaired detrusor contractility
- UDS
 - Impaired bladder sensation, increased capacity, decreased contractility, impaired uroflow, increased PVR
 - smooth or striated sphincter dyssynergia not usually seen
 - Erroneous dx: voiding with abdominal straining
 - Interference AMG pattern (pseudo-dyssynergia)
 - Abdominal straining alone does not usually open BN

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Treatment of “classic” findings

- Early institution of timed voiding
- Prevent impaired detrusor contractility from progressing to chronic distention and detrusor decompensation.
 - Tight glucose control can slow progression and slow development of abnormal autonomic tests – Clark and Lee, 1995
 - Prevention of urodynamic and histopathologic changes in diabetic rabbits – Ayan, 1999
 - Reversal of sx/urodynamic changes – Cardozo, 2002

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DM – changing view

- Traditional recognition of diabetic bladder dysfunction = voiding problem characterized by poor emptying and overflow incontinence
- Recent clinical/experimental evidence indicate storage problems such as urgency and urge incontinence in DM.
- Recent experimental evidence from studies of diabetic bladder dysfunction in small animal models show a temporal effect on diabetic bladder dysfunction.
 - Early phase diabetes mellitus causes compensated bladder function and the late phase causes decompensated bladder function.

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“Typical” DM voiding dysfunction

- UDS
 - DO 55%- 61%
 - Half with DHIC
 - Detrusor underactivity 17%-23%
 - Normal 11-13%13%
 - Detrusor areflexia 9%-10%
 - Kaplan, 1995, Chancellor and Blaivas, 1995, Starer and Libow, 1990, Ueda, 1997, Yamaguchi [5]

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CIC and UTI

- Certainty of bacteriuria
- Symptomatic UTI
 - Systemic sx rather than GU symptoms
- Preventive strategies
 - Clean technique
 - Maintenance of appropriate bladder volumes
 - Regular emptying intervals
- There are no definitive studies illustrating that incidence of UTIs is affected by sterile single-use or coated catheters compared to clean reused catheters.
 - Moore, Cochrane Database Systemic Review, 2007

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UDS

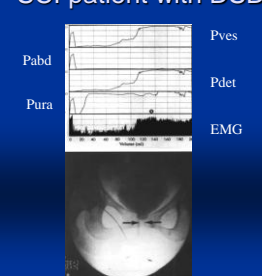
- Most definitive modality to assess dysfunction associated with NGB
 - Filling and storage
 - CMG, sphincter EMG, DLPP, ALPP
 - Voiding
 - PFS, VCUG, sphincter EMG
- Baseline: information guides management
 - Monitoring with f/u UDS q 2 years or as needed
 - Alter therapy as needed
 - Abrams, 2003

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SCI patient with DSD

Pabd

Pura




Pves

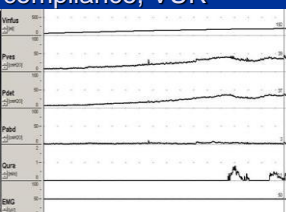
Pdet

EMG

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Video UDS - S/P radical pelvic surgery, poor compliance, VUR





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Obstruction

Previous BNS

Slide 50



Video UDS-MS

- 28 yo woman with M.S. VCUG with EMG shows DSD with fluoroscopic confirmation of obstruction at the external urethral sphincter

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Summary

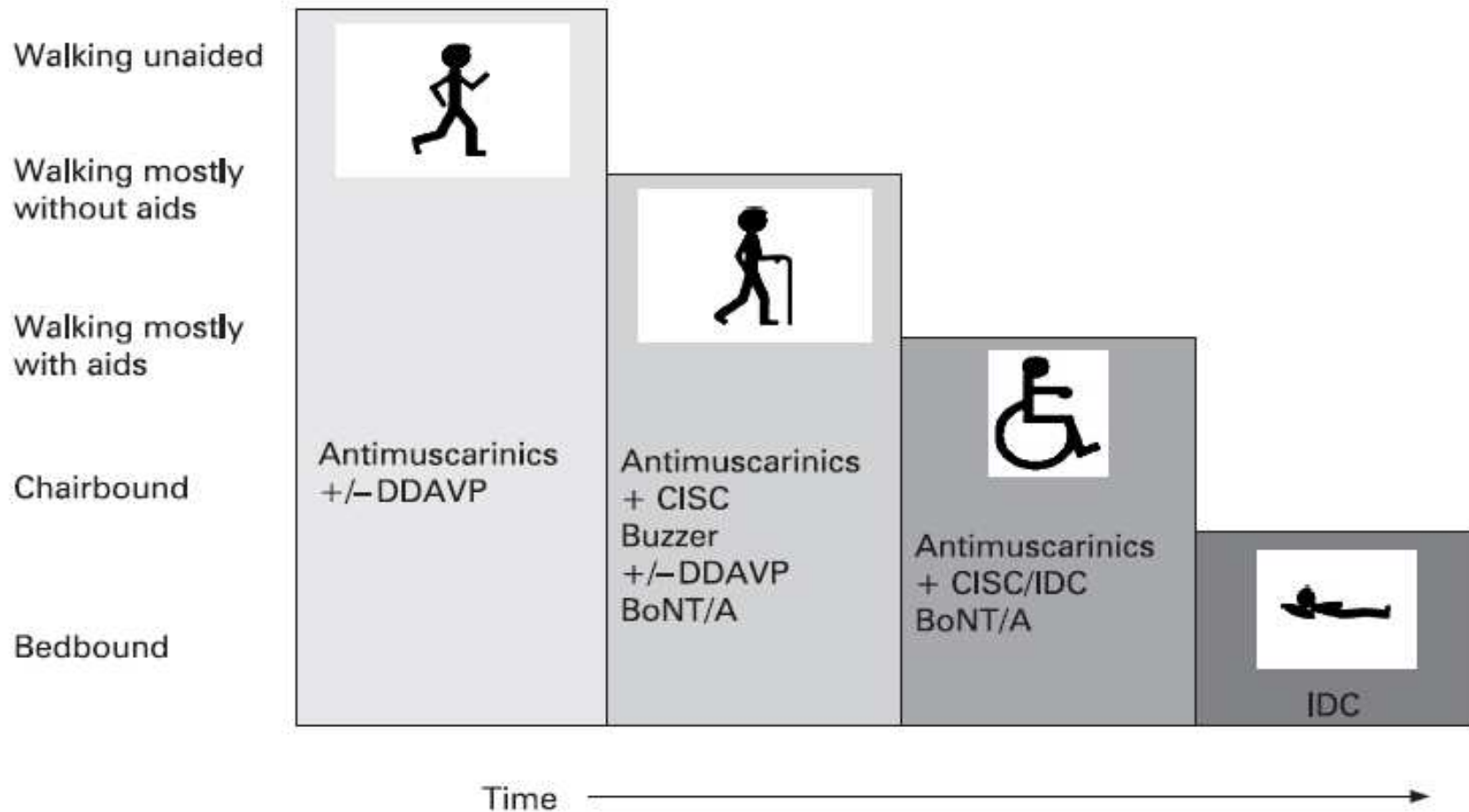
- Successful management of NGB is analogous to good parenting
- Have a high level of suspicion of occult dysfunction
- Early evaluation and intervention
- Ask for help when needed
- Be ready to change courses

Go from this:  To this: 

Principles of Management of Voiding Dysfunction Secondary to MS or Radical Pelvic Surgery

Christopher P. Smith
Associate Professor
Scott Dept of Urology
Baylor College of Medicine

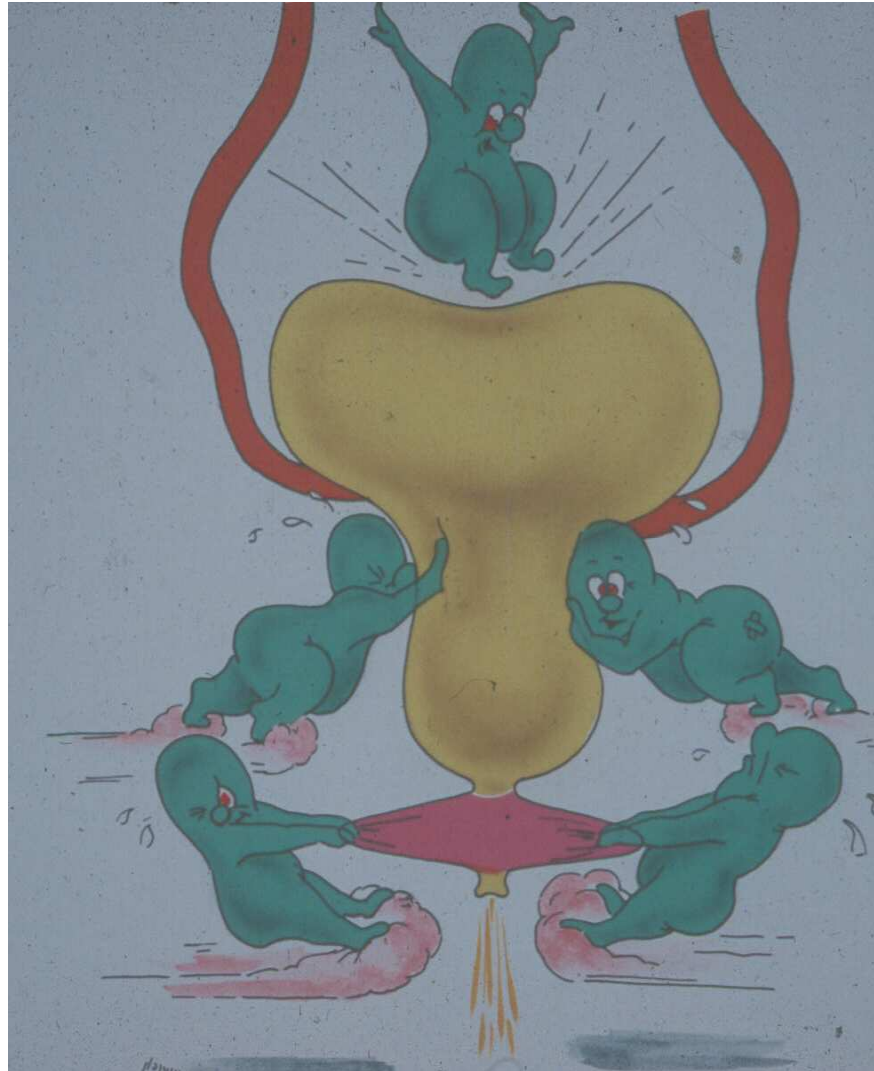
Principles of Management of LUTS in MS



Failure to Store

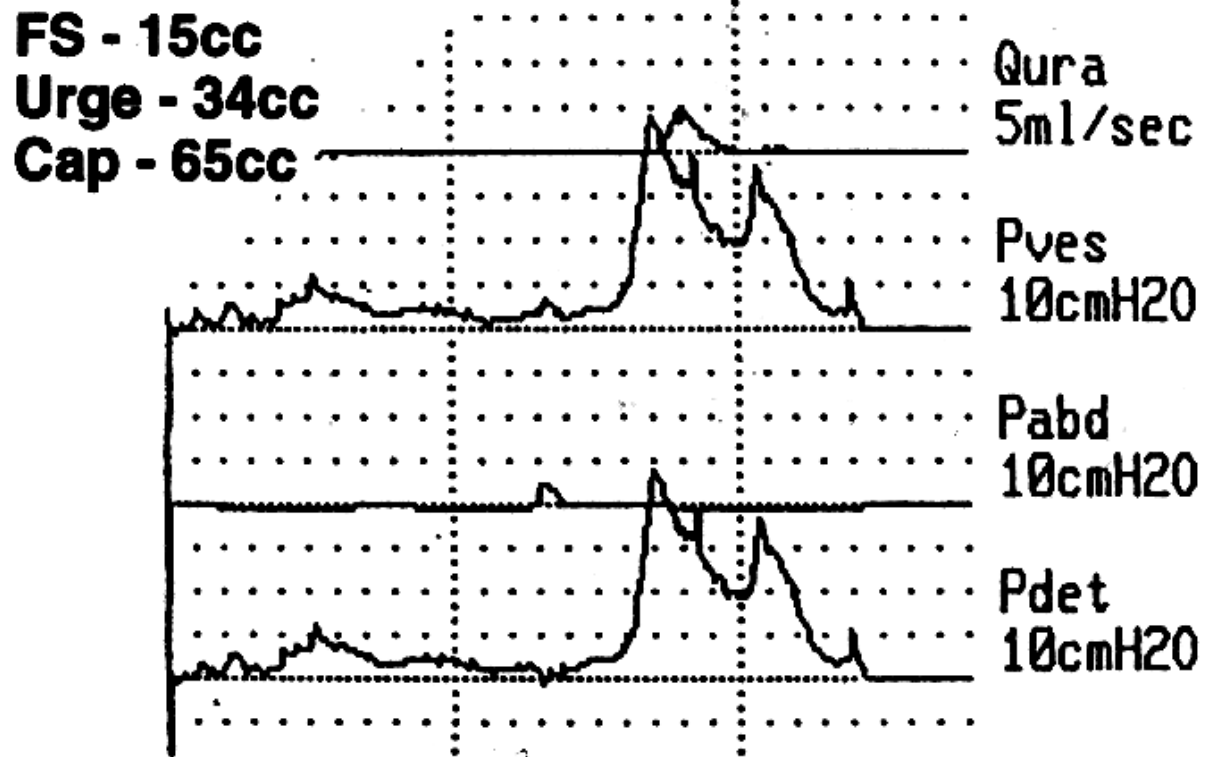
- **Detrusor Cause**
 - Detrusor Hyperreflexia
 - Loss of Detrusor Compliance
- **Sphincter Cause**
 - Denervation
 - Ablation
 - Fixed, open scar

Detrusor Hyperreflexia



35 Year Old Female with MS

Figure 1




DetrolTM
tolterodine tartrate tablets



Once-a-day
DITROPAN[®] **XL**
(oxybutynin chloride) Extended-release
tablets 5, 10, 15 mg



NEW



SANCTURATM
Trospium Chloride 20 mg Tablets

new
ONCE-DAILY
VESicare[®]
(solifenacin succinate)
tablets

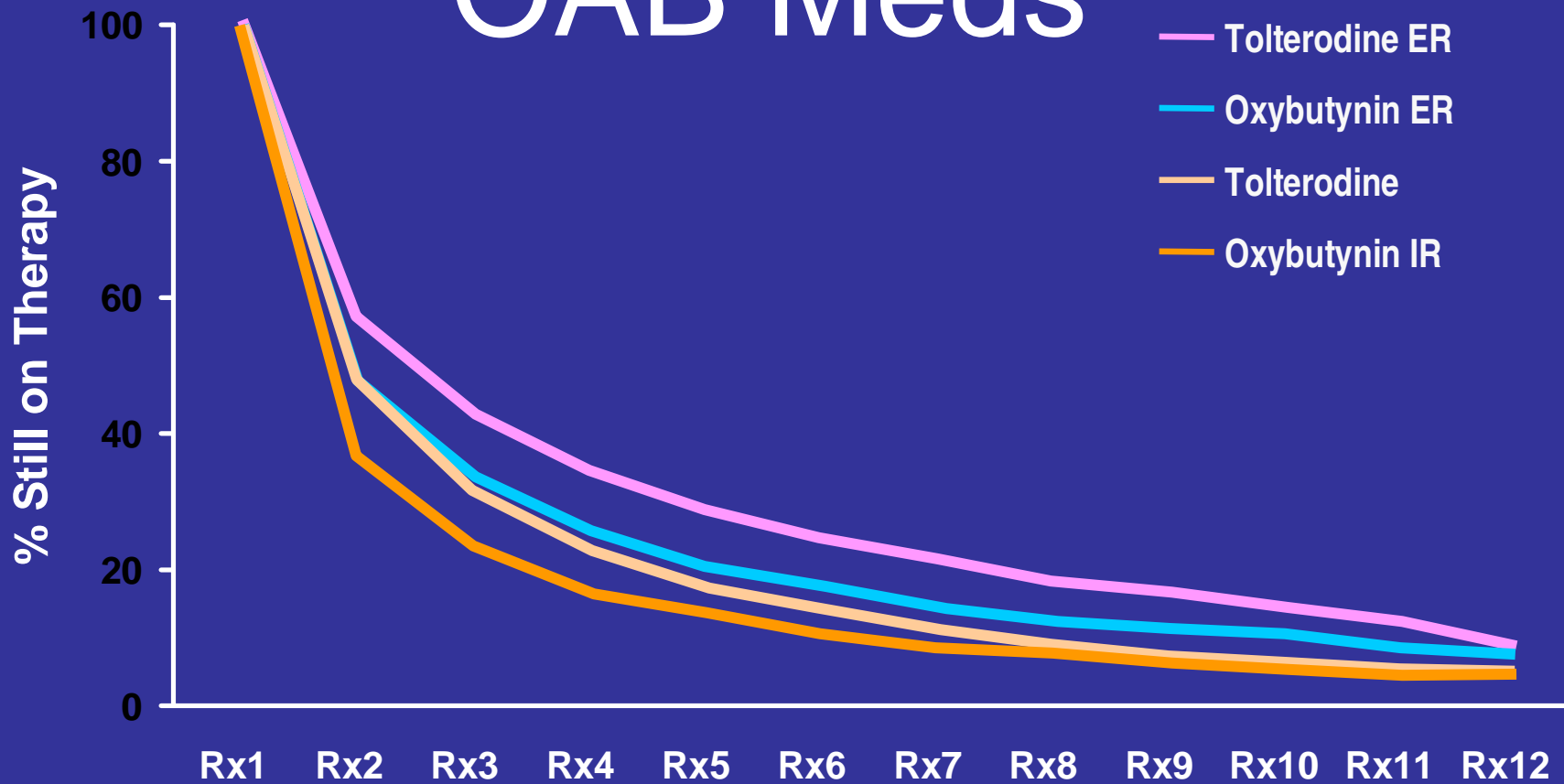


Prescri



Enablex[®]
(darifenacin)
once daily 7.5mg or 15mg

Poor Compliance with OAB Meds



Base: 26,200; New Patients: Oct-Dec 2000

Oral vs Intravesical Treatment

TABLE 2. *Urinary symptom measures*

	Mean \pm SD Baseline	Mean \pm SD Change		95% CI
		Oxybutynin	Atropine	
Bladder capacity (ml)	221.9 \pm 106.9	55.5 \pm 67.2	79.6 \pm 89.6	-0.4, 49.7
No. voiding frequency	8.8 \pm 2.9	-1.2 \pm 1.8	-1.6 \pm 2.0	-0.9, 0.2
No. incontinence events	1.7 \pm 2.1	-0.9 \pm 1.6	-0.9 \pm 1.7	-0.3, 0.3

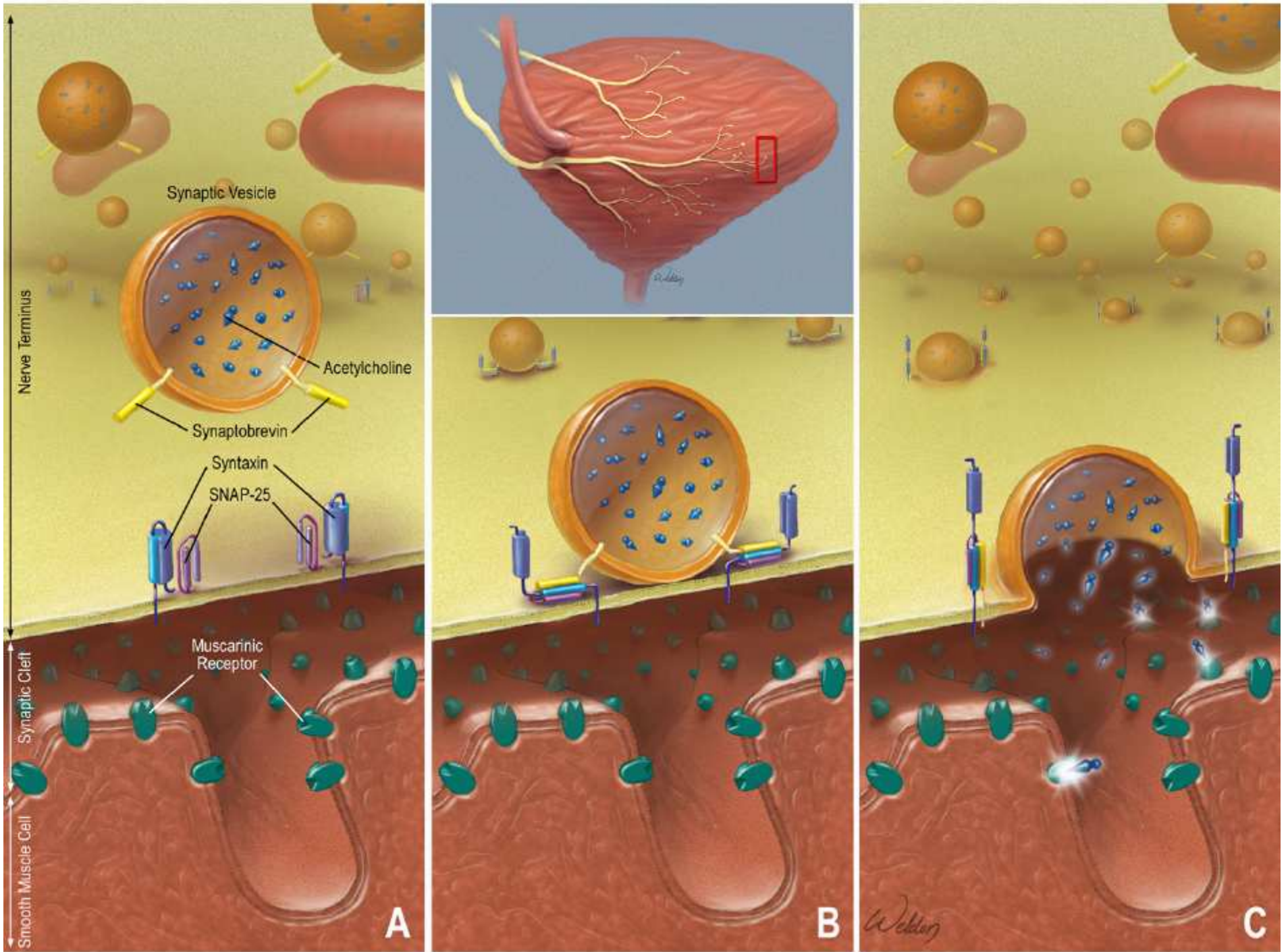
TABLE 3. *Dry mouth side effect ratings data on each participant*

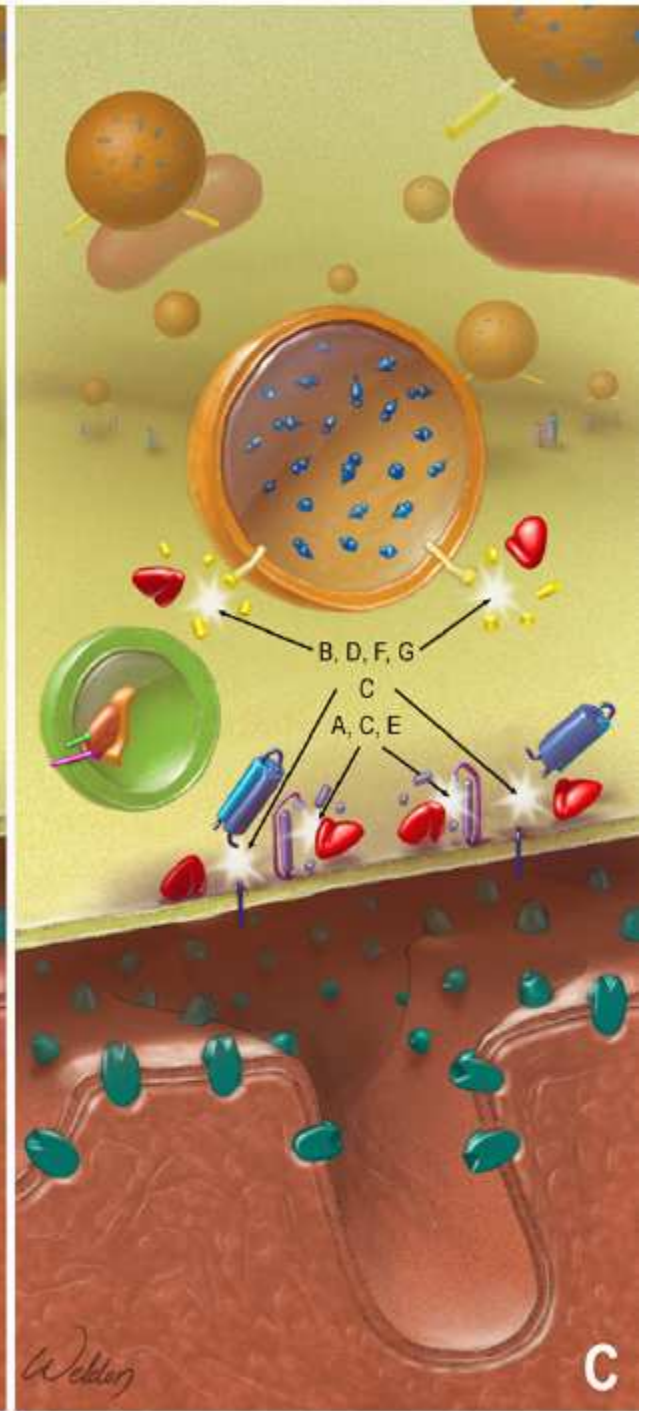
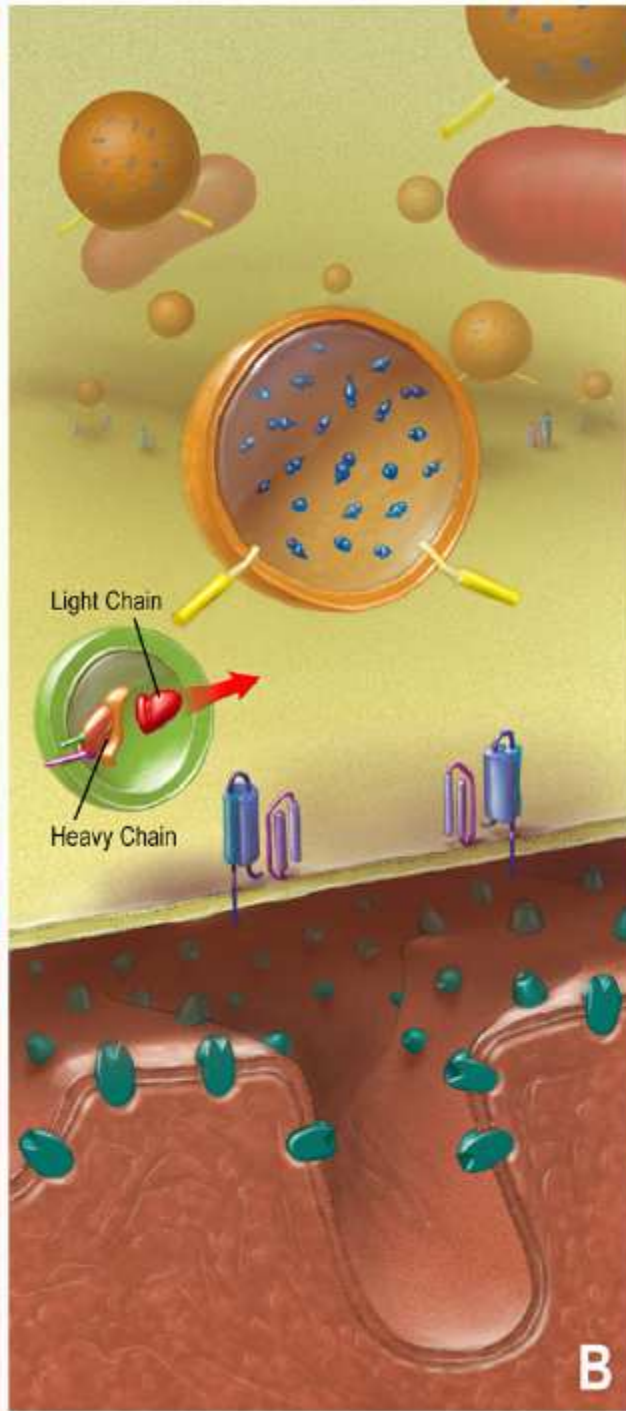
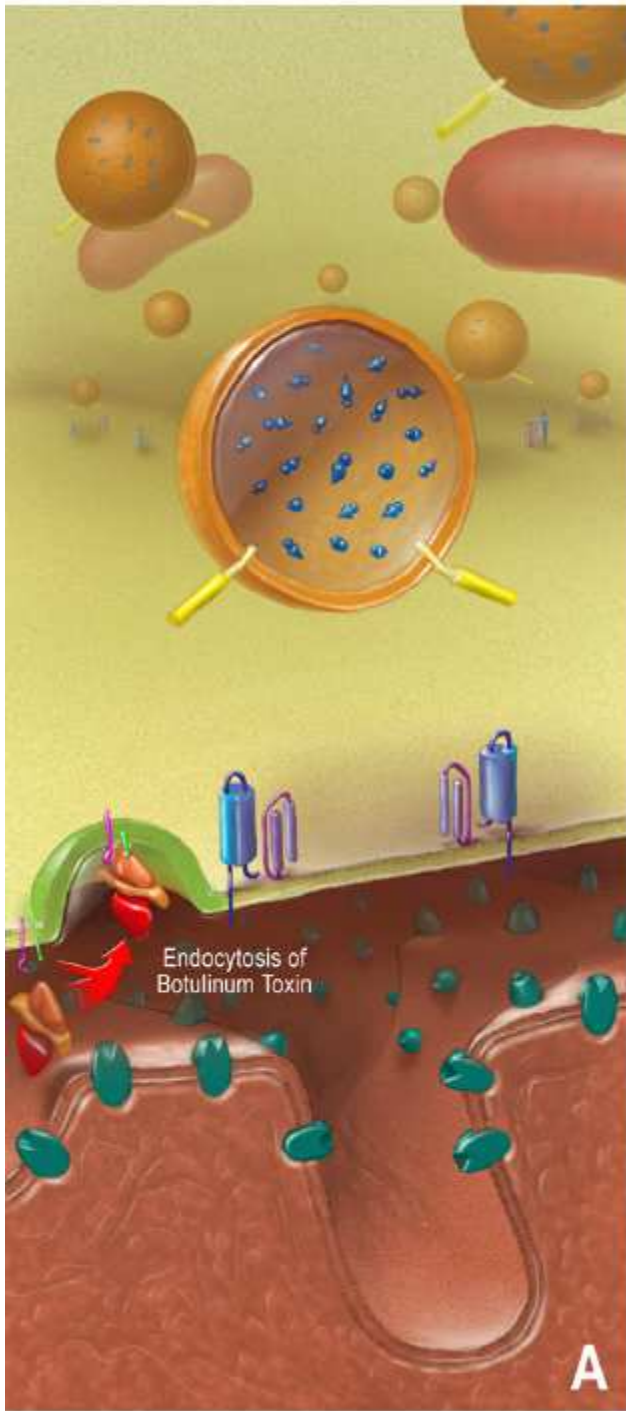
	No. Oxybutynin	No. Atropine
Better (-1)	1	6
Same (0)	17	40
Worse:		
1	25	11
2	14	0

Total of 57 participants per group.

If dry mouth was rated as 1 (a little) at baseline and then 1 (a little) after atropine arm, change data would show 0, ie the same, and if dry mouth was rated as 2 (a lot) after oxybutynin arm, change data would show 1, ie worse.

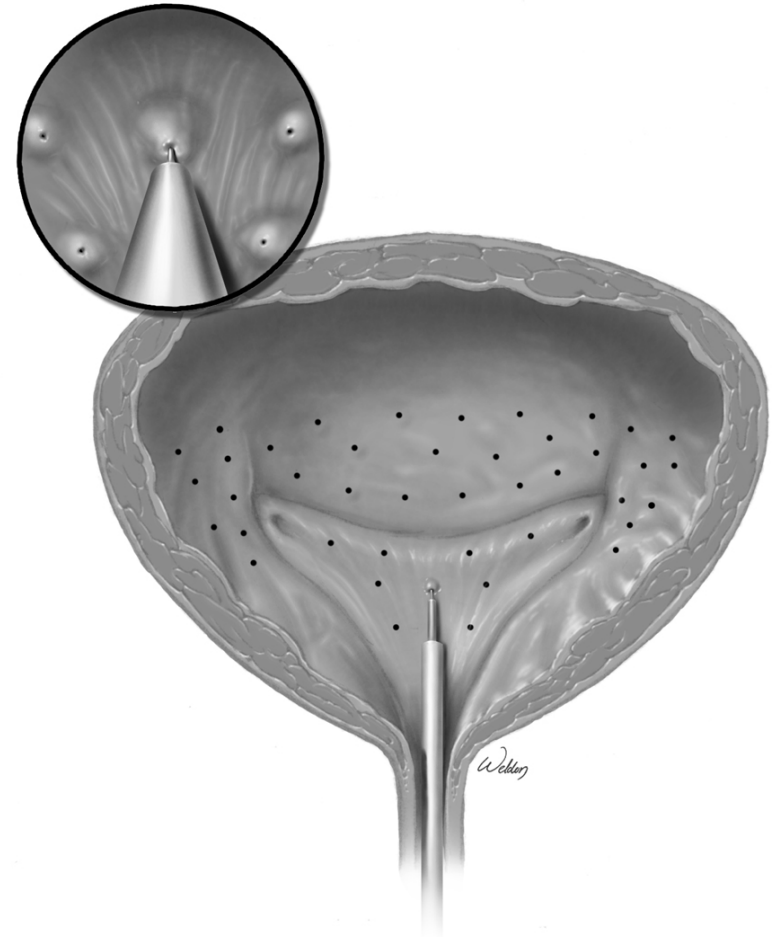
Botulinum Toxin for Overactive Bladder



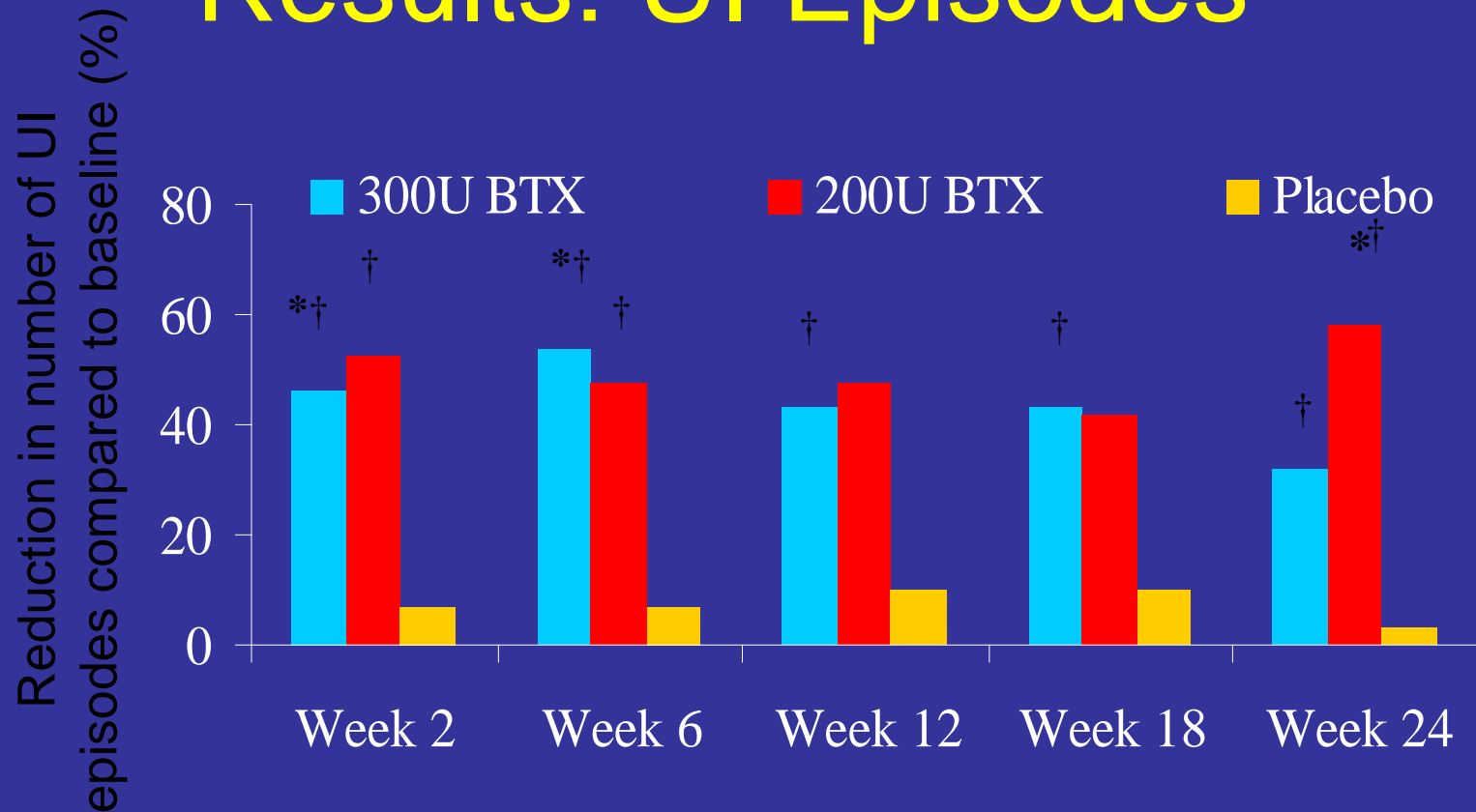


Rigid Cystoscope Bladder Injection Technique

- Dilute 100-300 U of Botox into 10-30 ml of saline
- Inject targeting the trigone, base of the bladder and lateral walls
- Rigid cystoscope: 25 Gauge Cook® Williams needle, inject approximately 0.5-1.0 ml into 20-30 sites submucosally



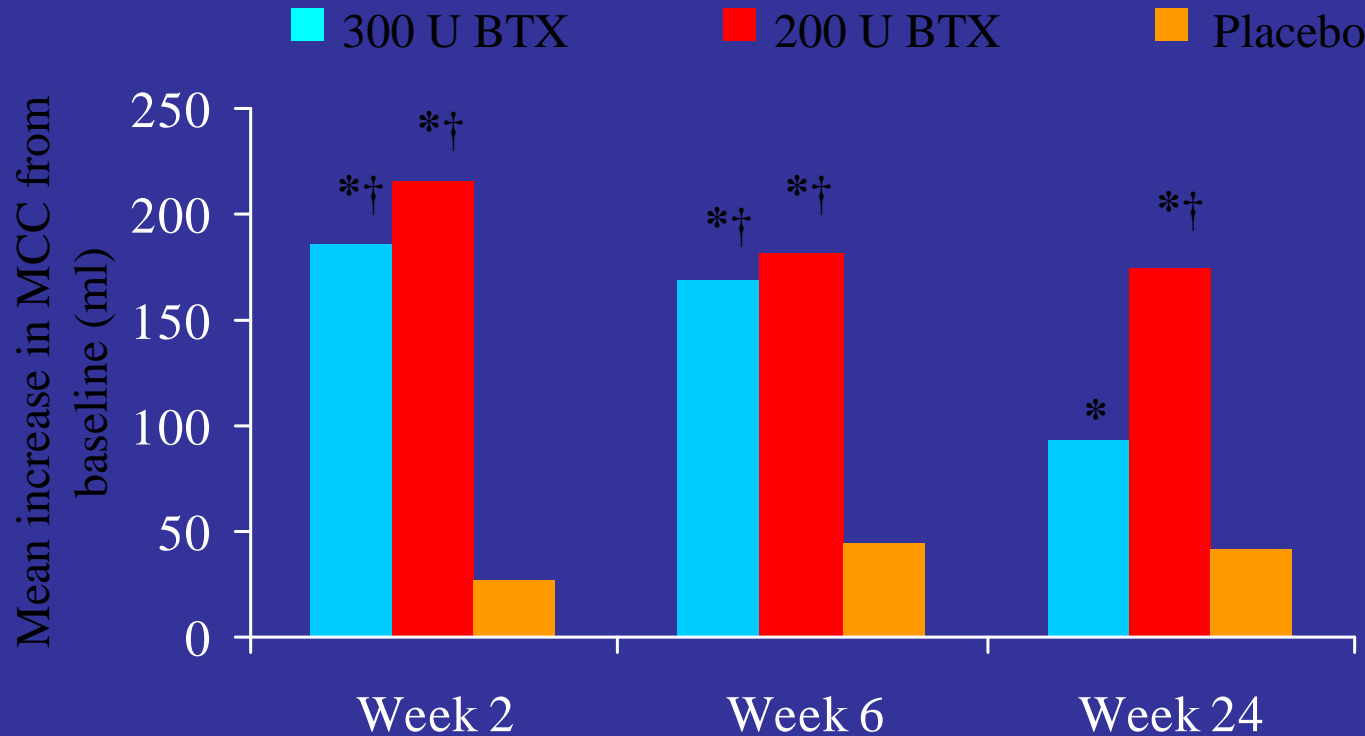
Results: UI Episodes



* $p < 0.05$ for differences between BTX group and placebo

† $p < 0.05$ for difference within-group changes from baseline

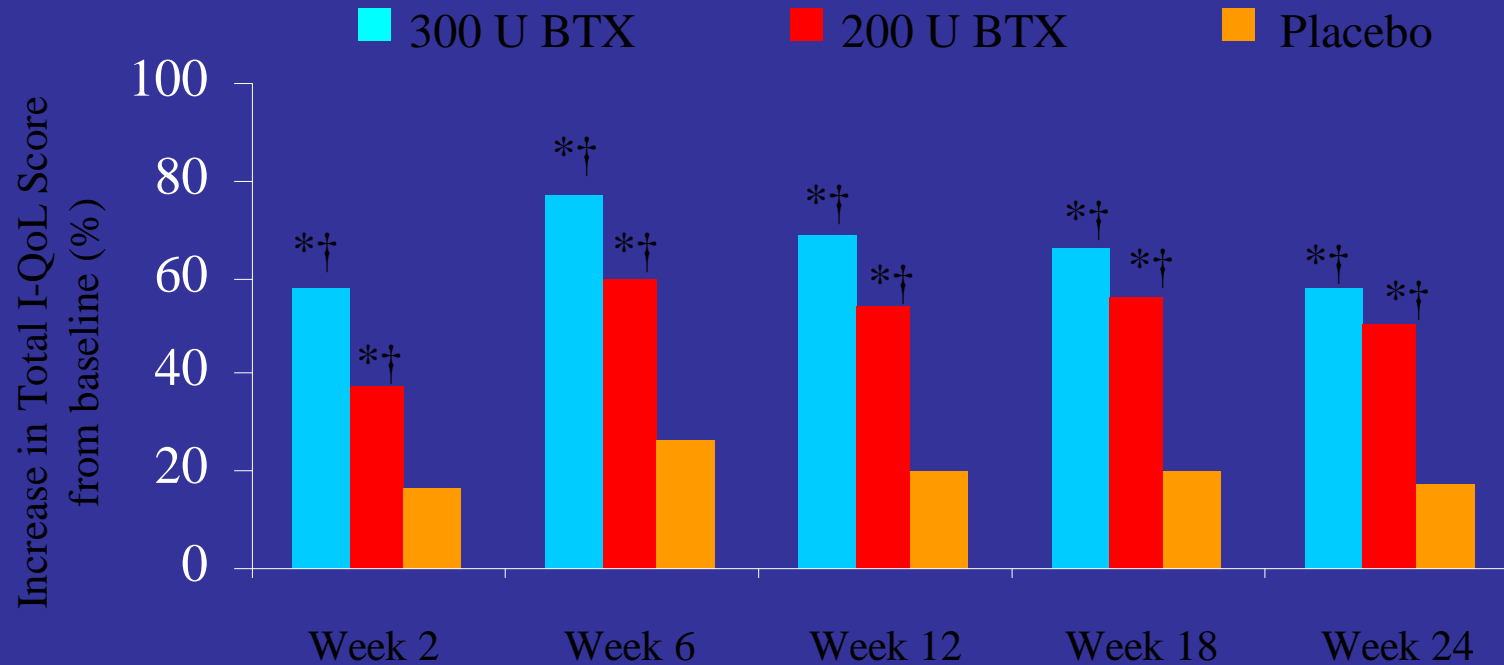
Results: Urodynamics – MCC



* $p < 0.05$ for within-group changes from baseline

† $p < 0.05$ for pairwise contrasts between BTX groups versus placebo

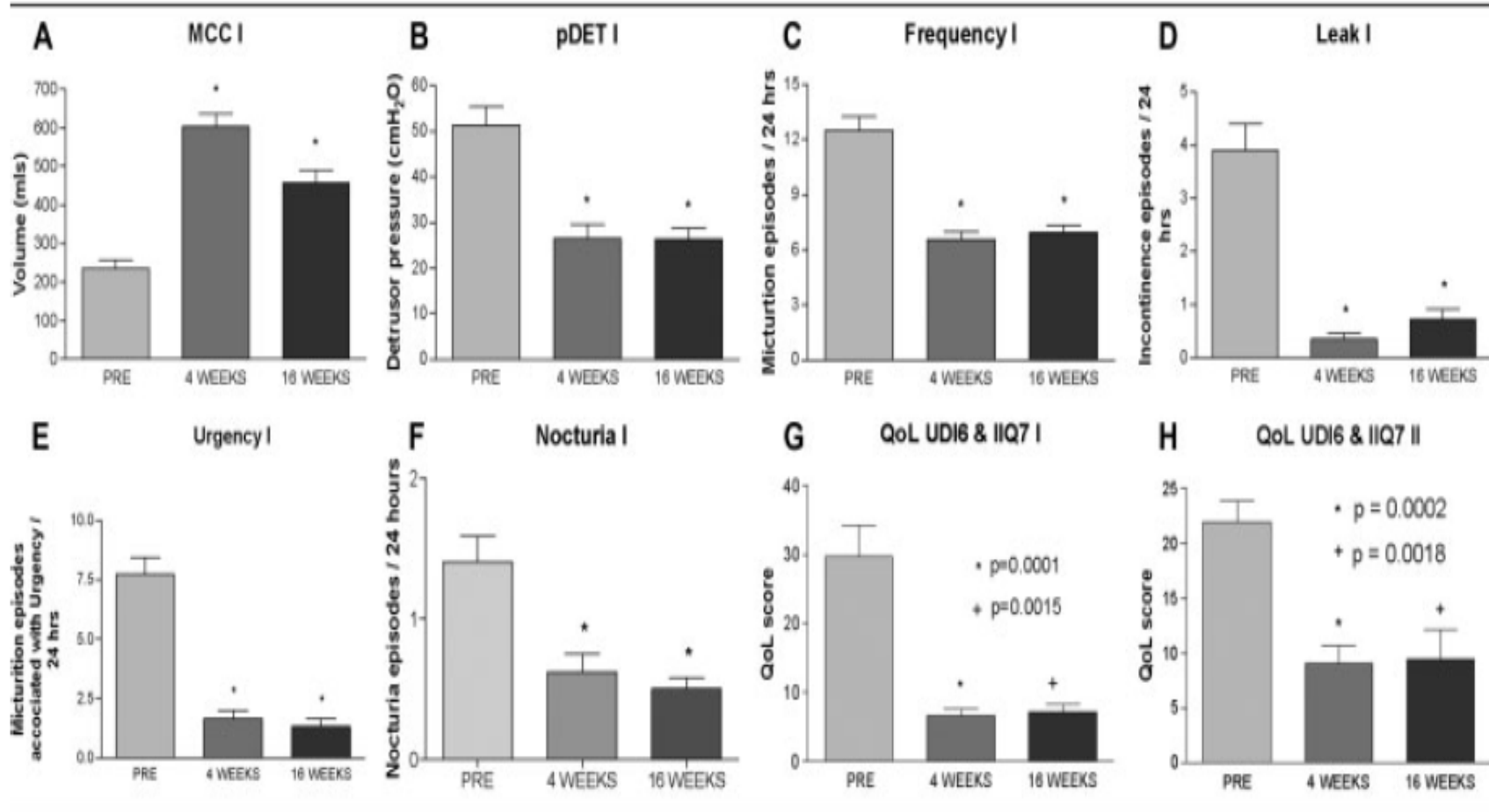
Results: Quality of Life



* $p < 0.05$ for pairwise contrasts between BTX groups and placebo

† $p \leq 0.002$ for within-group differences from baseline

BoNT-A for MS



BoNT-A for MS

Table 3. Changes from Baseline in the Use of Procontinence Medication (Number and Percentage of Patients on Medication, Mean Dose of Anticholinergics Used) at 4 and 16 Weeks after the First Botulinum Neurotoxin Type A Treatment

Study Time Point	Patients Taking Procontinence Medication (% of total)	Number of Patients Taking Most Common Types of Procontinence Medication		Mean Dose of Anticholinergic Medication Used \pm SEM, mg				
		Anticholinergics	Desmopressin	Oxybutynin	Tolterodine	Propiverine	Trospium	Solifenacin
Pretreatment	33/43 (76.7%)	29	8	19.3 \pm 2.1 (n = 14)	4.1 \pm 0.2 (n = 12)	45 (n = 1)	40 (n = 1)	5 (n = 1)
4 weeks	12/43 (27.9%)	11	2	5.5 \pm 1.9 (n = 7)	1.3 \pm 0.6 (n = 4)	0 (n = 0)	0 (n = 0)	0 (n = 0)
16 weeks	12/43 (27.9%)	10	2	6.4 \pm 2.1 (n = 7)	1.0 \pm 0.5 (n = 3)	0 (n = 0)	0 (n = 0)	0 (n = 0)
<i>p</i> (pretreatment vs 4 weeks)	<0.0001			0.0004 ^a	0.0002 ^a	NA	NA	NA
<i>p</i> (pretreatment vs 16 weeks)	<0.0001			0.0006 ^a	<0.0001 ^a	NA	NA	NA

^a*p* values refer to statistical comparisons (Fischer's exact and paired *t* test) for before botulinum neurotoxin type A (pre-BoNT/A) injection versus 4 weeks and pre-BoNT/A versus 16 weeks. The percentage of patients taking antiincontinence medication was markedly reduced after successful treatment with intradetrusor BoNT/A. A significant reduction was noted in the mean dose of oxybutynin and tolterodine used at both follow-up visits.

SEM = standard error of the mean; NA = not applicable.

Electrical Neuromodulation

PTNS for MS

TABLE II. The Effects of PTNS on Urodynamic Variables for the Comparison of Baseline and After PTNS Data in MS Patients

Urodynamic variables	Baseline value, mean \pm SD (range)	PTNS, mean \pm SD	P-value
<i>First involuntary detrusor contraction</i>			
At volume (ml)	124.2 \pm 37.6 (60–185)	217.5 \pm 66.4 (94–347)	0.000
P_{detmax} (cmH ₂ O)	43.7 \pm 20.2 (14–97)	29.7 \pm 10.2 (13–51)	0.005
<i>Maximum cytometric capacity</i>			
At volume (ml)	199.7 \pm 29.3 (128–263)	266.8 \pm 36.9 (198–342)	0.000
P_{detmax} (cmH ₂ O)	48.8 \pm 21.4 (18–98)	35.8 \pm 10.5 (21–59)	0.001
$P_{det}Q_{max}$	35.8 \pm 8.8 (21–53)	24.7 \pm 7.6 (10–37)	0.002
Q_{max} (cmH ₂ O)	11.6 \pm 2.1 (7–15)	13.2 \pm 3.5 (7–22)	0.003
PVR (ml)	82.9 \pm 72.5 (0–276)	48 \pm 26.6 (0–107)	0.006

SNS for MS

TABLE 2

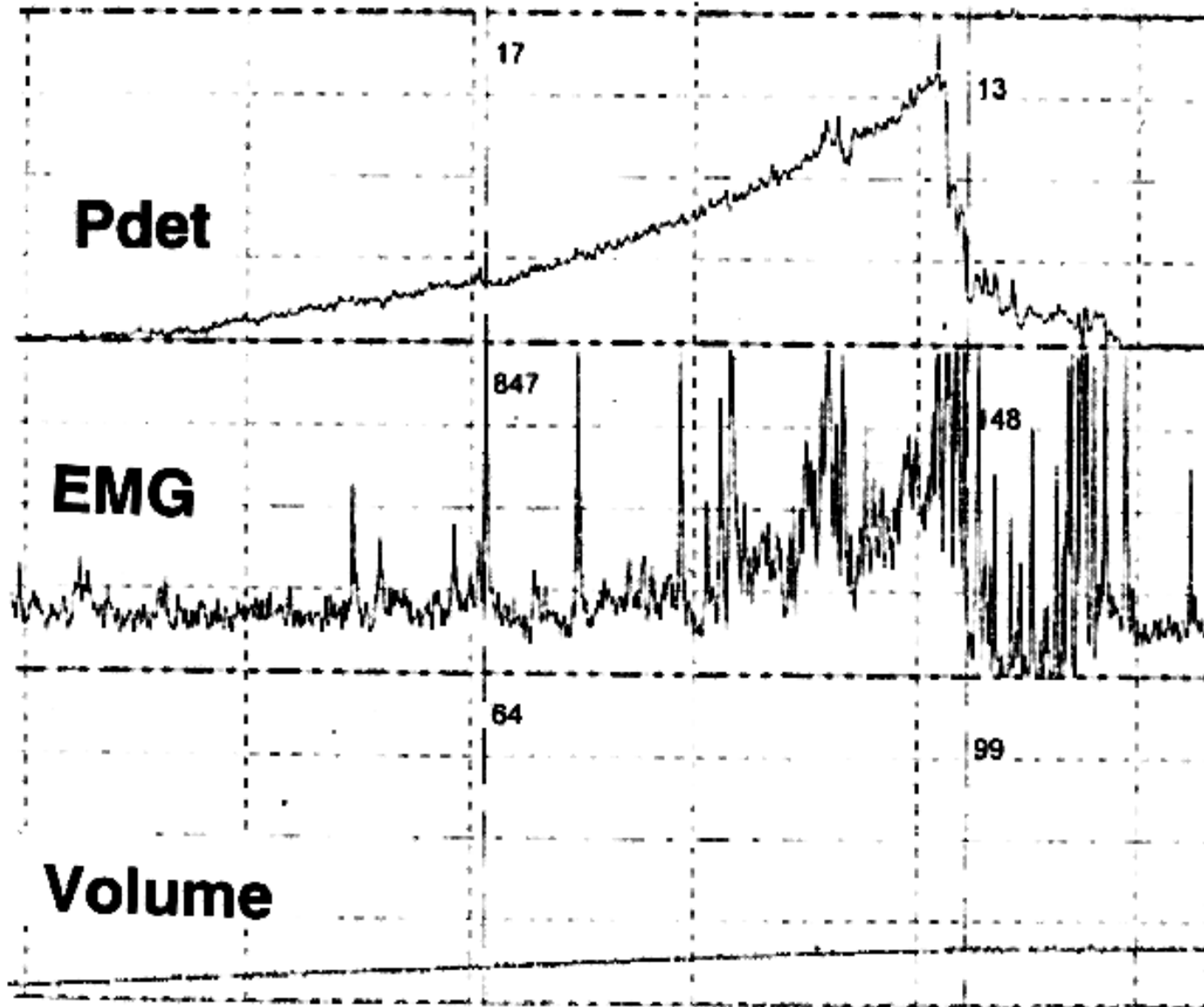
Preoperative and postoperative InterStim trial 4-day voiding diary events

	Mean frequency*	Mean nocturia	Mean number of incontinence episodes per 24 h	Mean number of pads per 24 h	Mean number of ISC per 24 h
Preoperative	10.5	2.6	4.0	3.5	3.8
Postoperative	6.0	0.8	1.3	1.0	1.6
Reduction, %	43	69	68	72	58
SD,	1.4	1.7	2.4	1.9	1.9
<i>P</i> value	<.0001	<.0001	<.0001	<.002	<.02

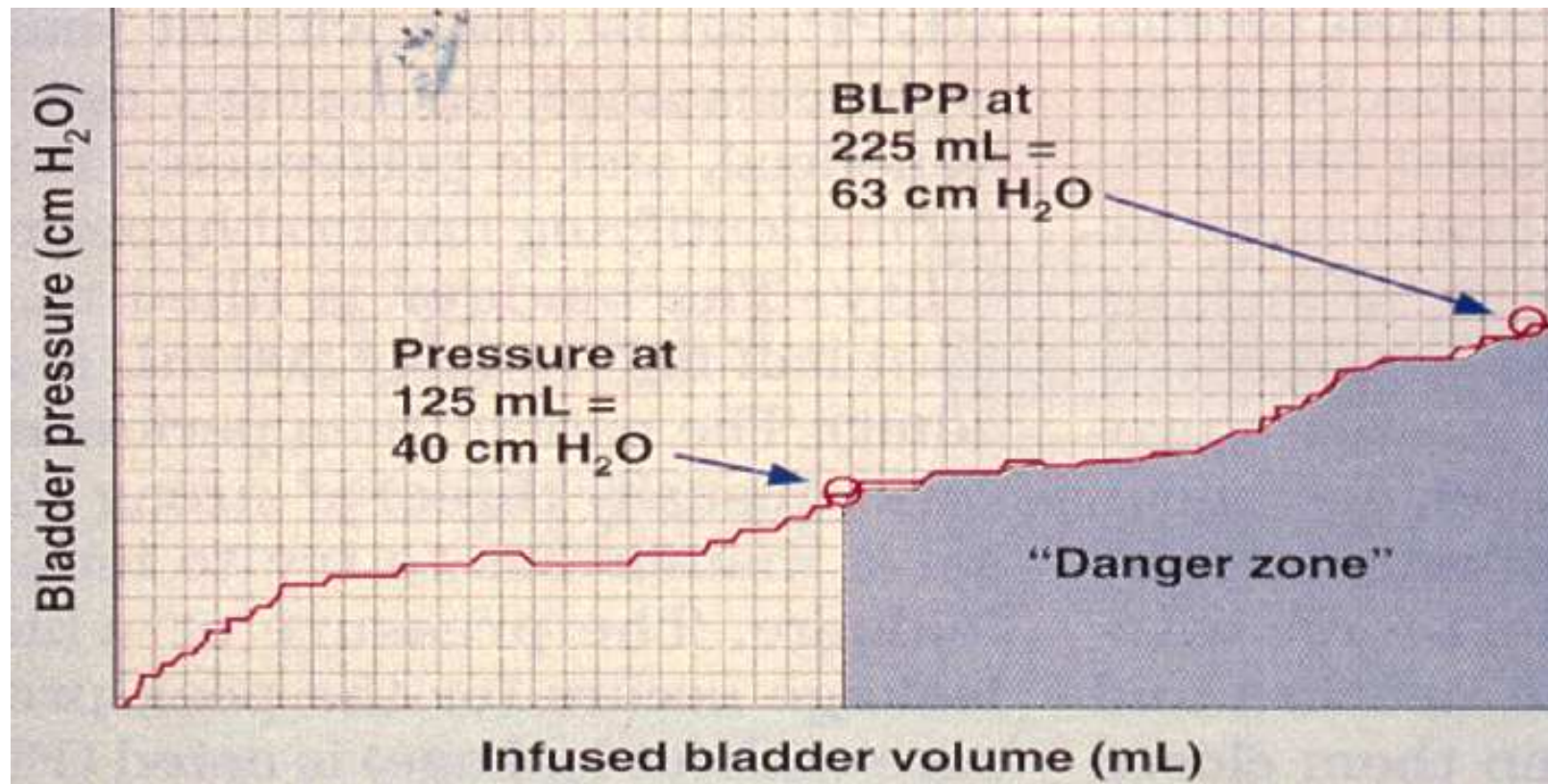
ISC, intermittent self-catheterization.

*Number of daytime voids.

Loss of Compliance



High Detrusor Leak Point Pressure



Elevated Intravesical Pressure

- In MMC patients: LPP > 40 cmH₂O: 68%
Reflux 81% Hydroureteronephrosis ¹
- In SCI patients: Decreased compliance:
39% Reflux 64% Hydroureteronephrosis ²
- Reflux resolve spontaneously with
normalization of intravesical pressure ³

1 McGuire et al: J Urol 126:205, 1981

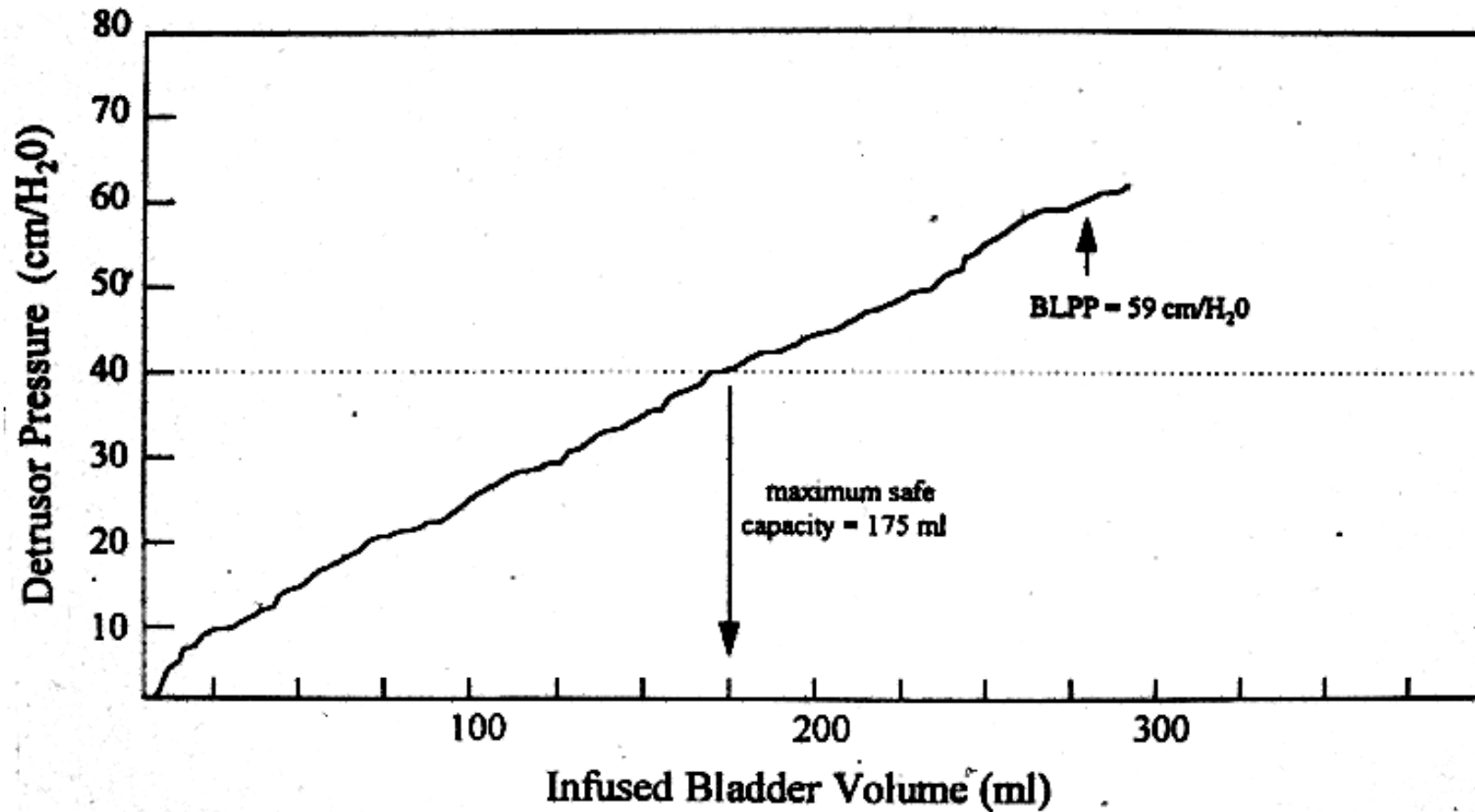
2 Hackler et al: J Urol 141: 1390, 1989

3 McGuire, Savasanto J Am Para Soc 8:28, 1985

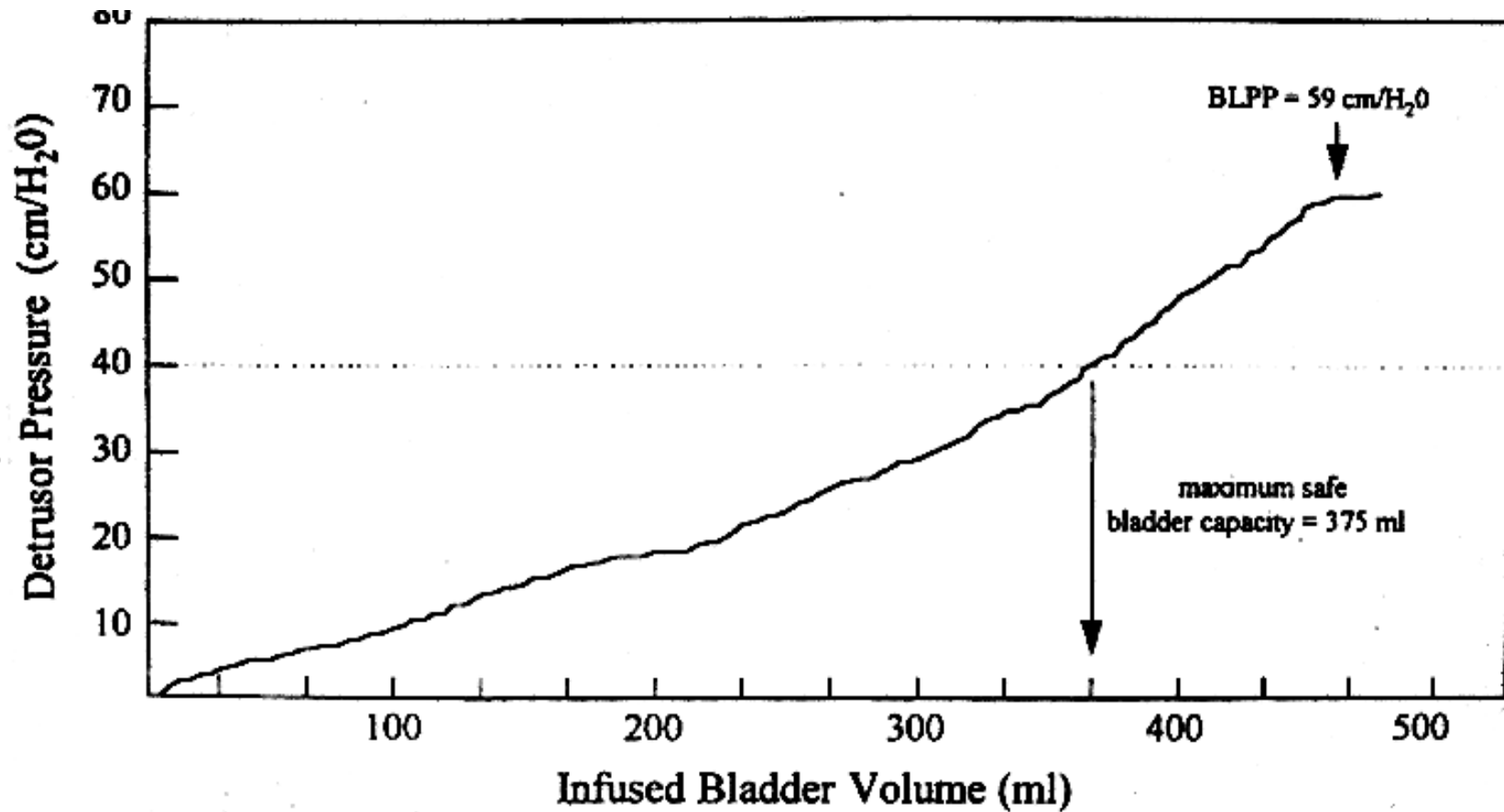
Bladder Leak Point Pressure

- Serves as your guide for storage pressure and indicates the pressure generated to overcome the outlet with voiding
- Store at $< 35 \text{ cm H}_2\text{O}$
- Void at $< 40 \text{ cm H}_2\text{O}$

Poor Detrusor Compliance Before Anti-Muscarinic Treatment



Filling Pressures After Antimuscarinic Treatment

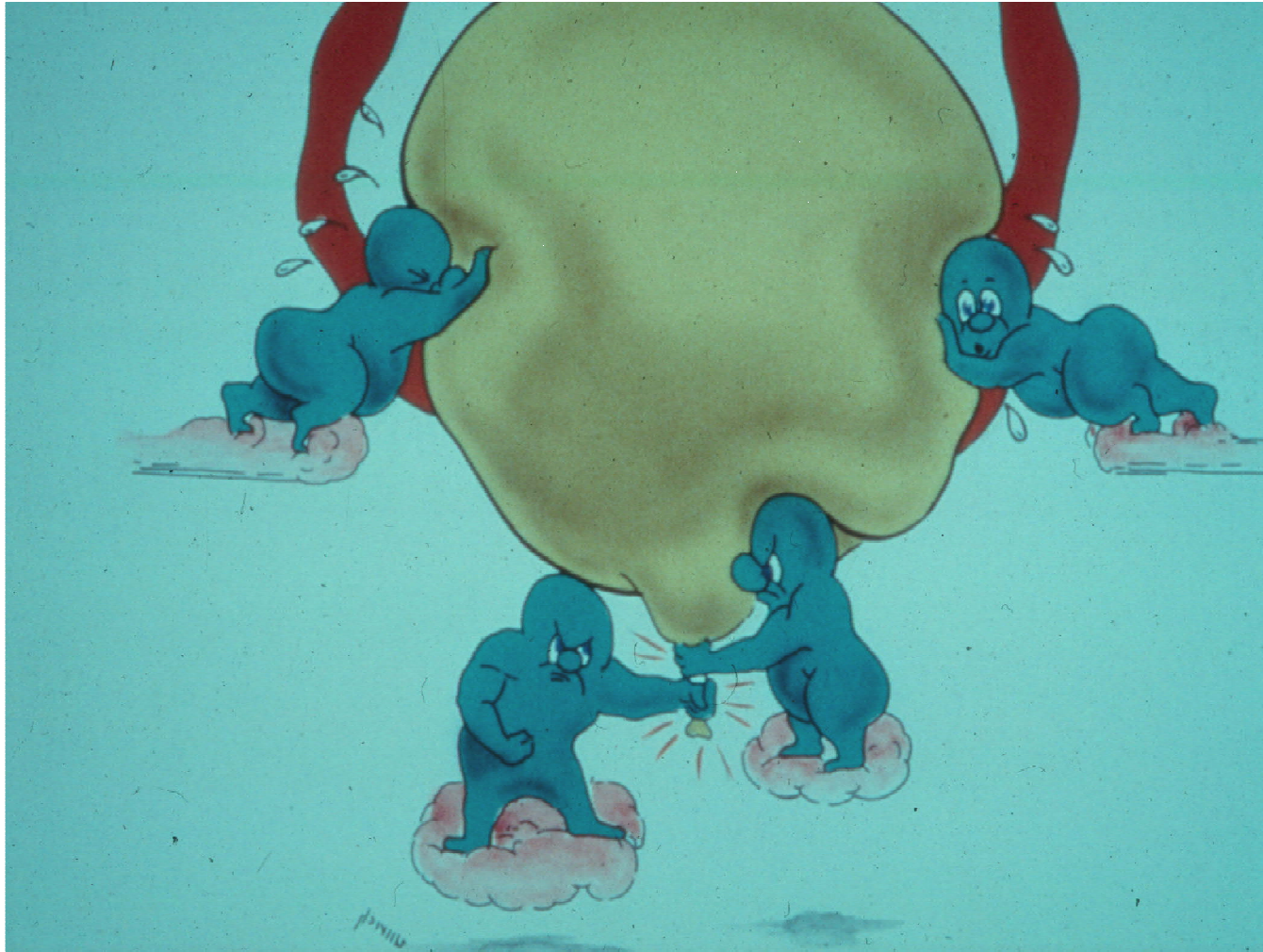


Failure to Empty

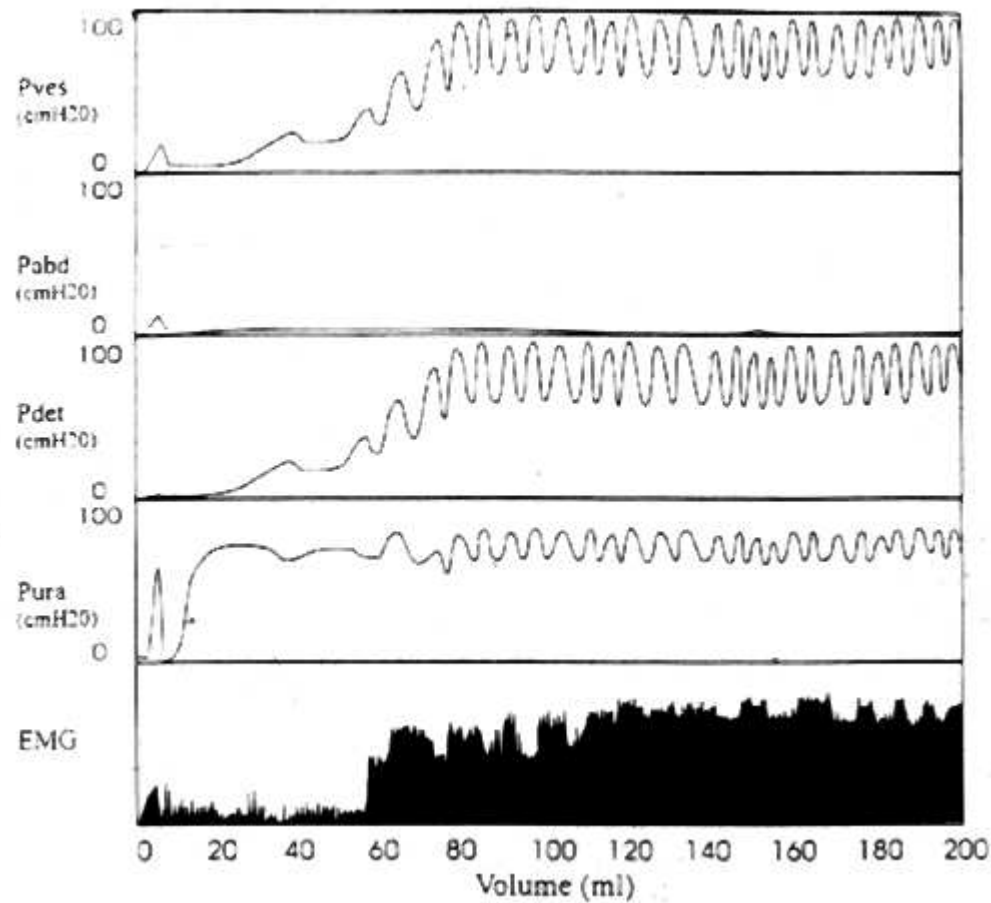
Should think of treatment of neurogenic lower urinary tract dysfunction in components:

- Detrusor function - Is detrusor contraction of significant magnitude to achieve emptying?
- Sphincter function - Is sphincter creating excessive outlet resistance?

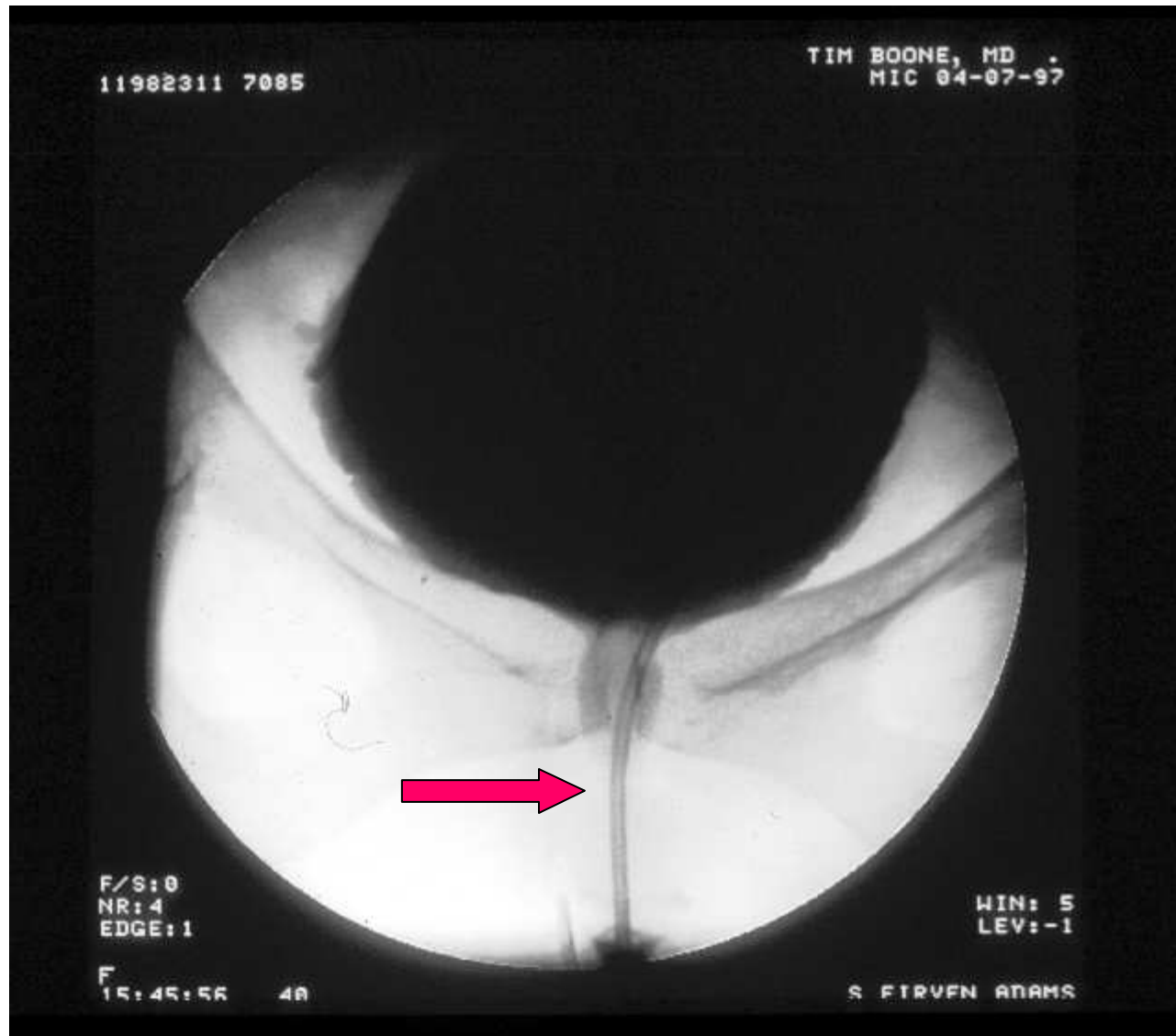
Detrusor-Sphincter Dyssynergia



Detrusor External Sphincter Dyssynergia (DESD)



Detrusor – Sphincter Dyssynergia



Indwelling Catheterization

Risks

- Chronic infection, stone formation
- Tissue erosion (traumatic hypospadias, bladder neck/ urethral destruction)
- Bladder wall fibrosis, urothelial neoplasia
- Vesicoureteral reflux, hydronephrosis
- Nephrolithiasis, renal failure
- **Avoid indwelling catheterization except as last resort**

Indwelling Catheterization

- McGuire and Savastano (1986)
 - 22 women with SCI managed with indwelling catheters
 - 54% - IVP changes
 - 54% - autonomic dysreflexia
 - 100% - recurrent stones
 - 54% - non-functional urethra
 - 46% - urethral erosion
 - 92% - febrile UTI's
 - 92% - leakage around the catheter

Squamous Cell Carcinoma

- Represents 3 - 6% of all bladder tumors
- 20X risk of SCCa in patients managed with chronic catheters
- Tumors in patients managed with chronic catheters are usually diagnosed at a more advanced stage and do not respond well to chemo or XRT

Intermittent Catheterization¹

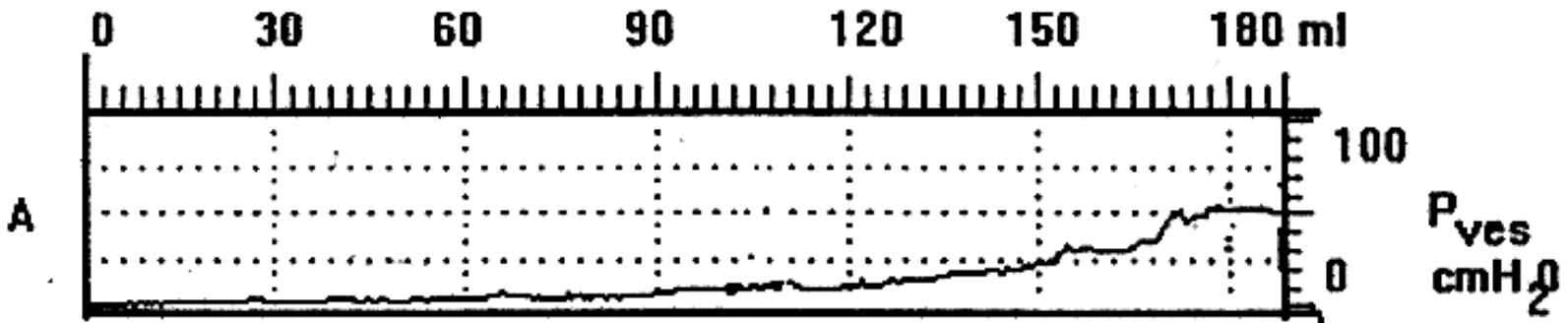
- Need to assure low-pressure storage
- Urodynamics essential to check compliance and leak point pressure²
- Hydronephrosis and reflux may resolve³
- Urinary tract becomes colonized⁴
- Treat only clinical UTI (fever, hematuria, epididymitis)
- Complications of strictures, false passages

1. Lapidus et al, J Urol 107:458, 1972
1990

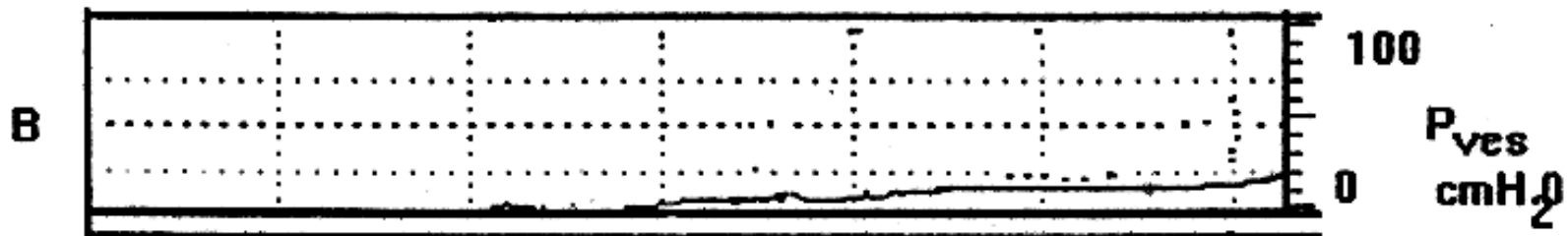
3. Wyndale & Maes, J Urol 143:906,

2. McGuire & Savastano J Urol 129:775, 1983 4. Maynard & Diokno, J Urol 132:943, 1984

Intermittent Catheterization

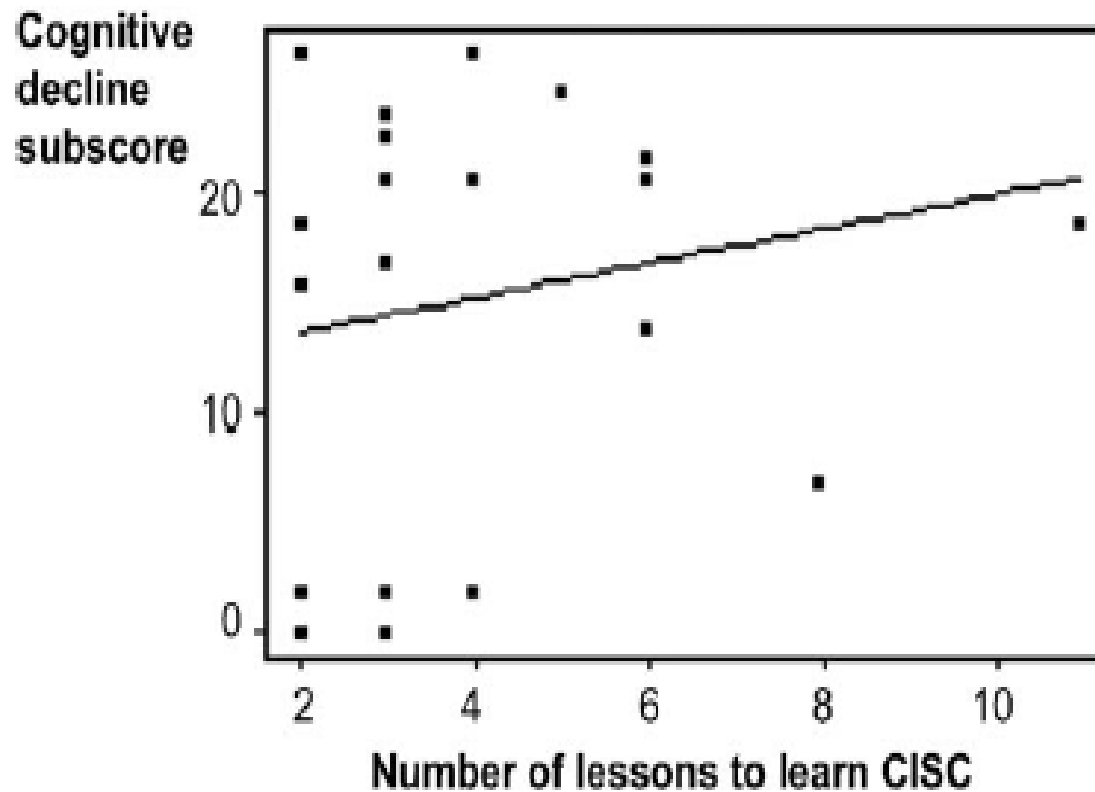


**Foley catheter for 5 years
Leak around the catheter at 180mls.**



**Intermittent self-catheterization
Normal compliance and total capacity > 400 mls.**

CIC for MS



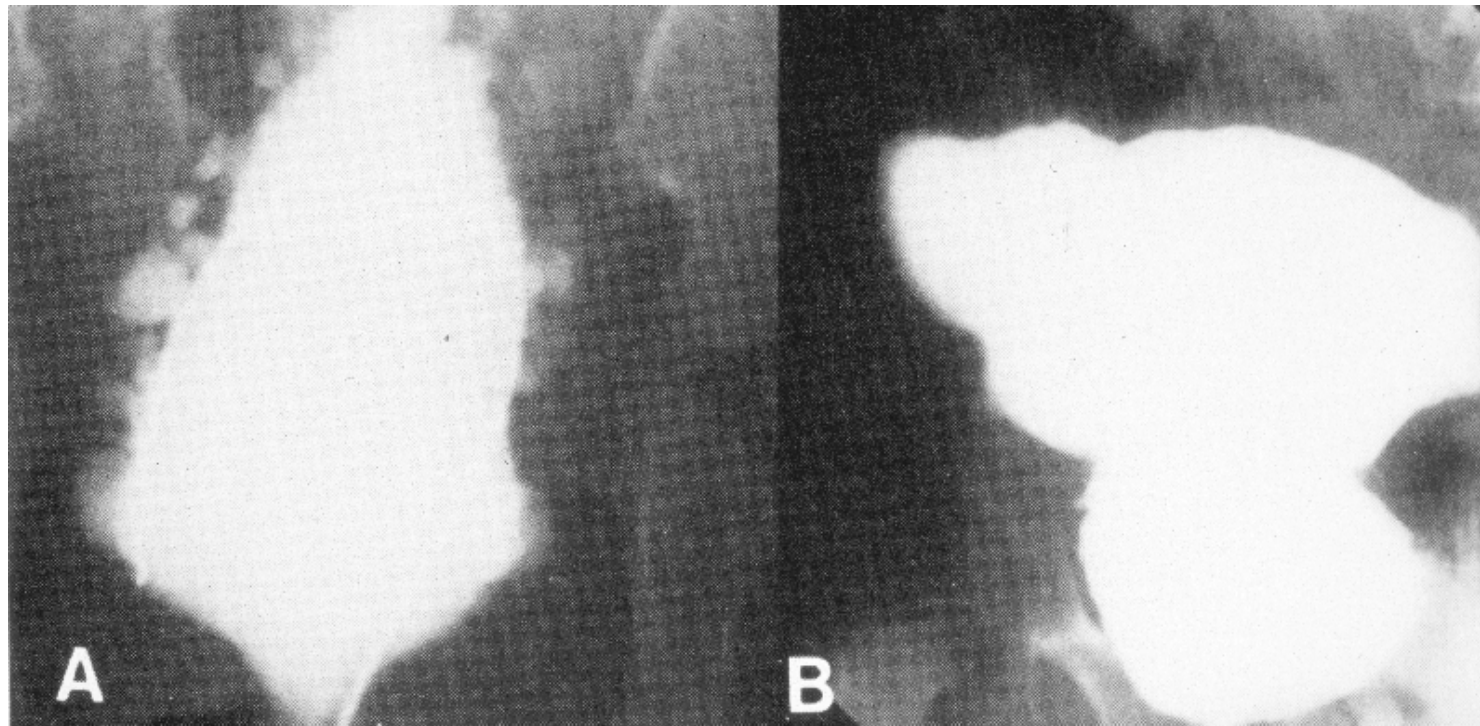
Surgical Alternatives

- Suprapubic Catheterization
- Suprapubic Catheterization with Urethral Closure
- Bladder Augmentation
- Bladder Augmentation with Continent Catheterizable Stoma
- Incontinent Ileovesicostomy
- Continent Urinary Diversion
- Ileal Conduit Urinary Diversion

Surgical Alternatives: Failure to Store

Augmentation Cystoplasty

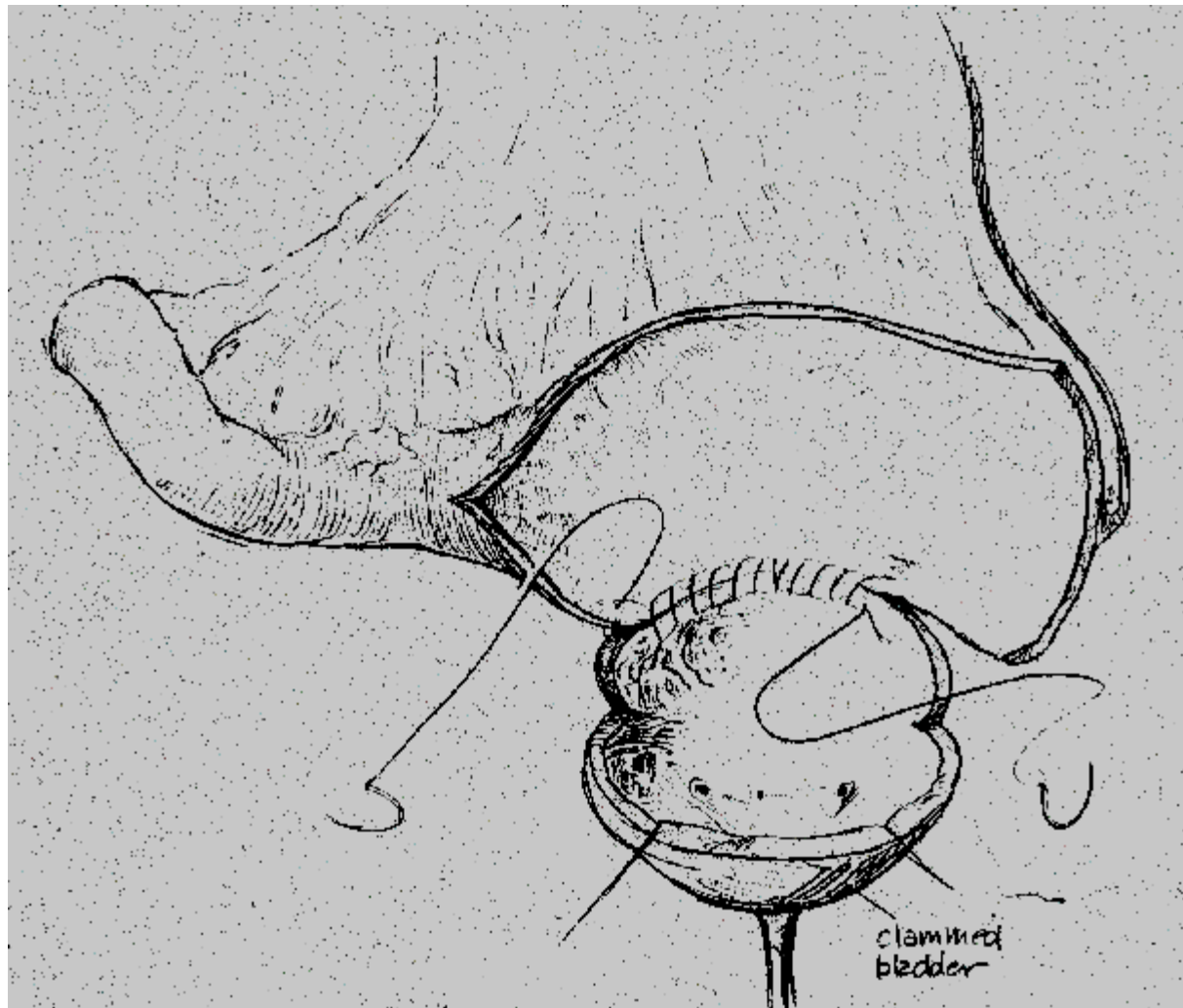
- Can use small or large bowel
- Transverse bladder incision facilitates placement of the augmentation
- Reflux - consider reimplantation for high grade reflux, +/- grade 3, grades 4 or 5
- Small but real risk of carcinoma in the augmented bowel segment



Cutaneous Ileocystostomy

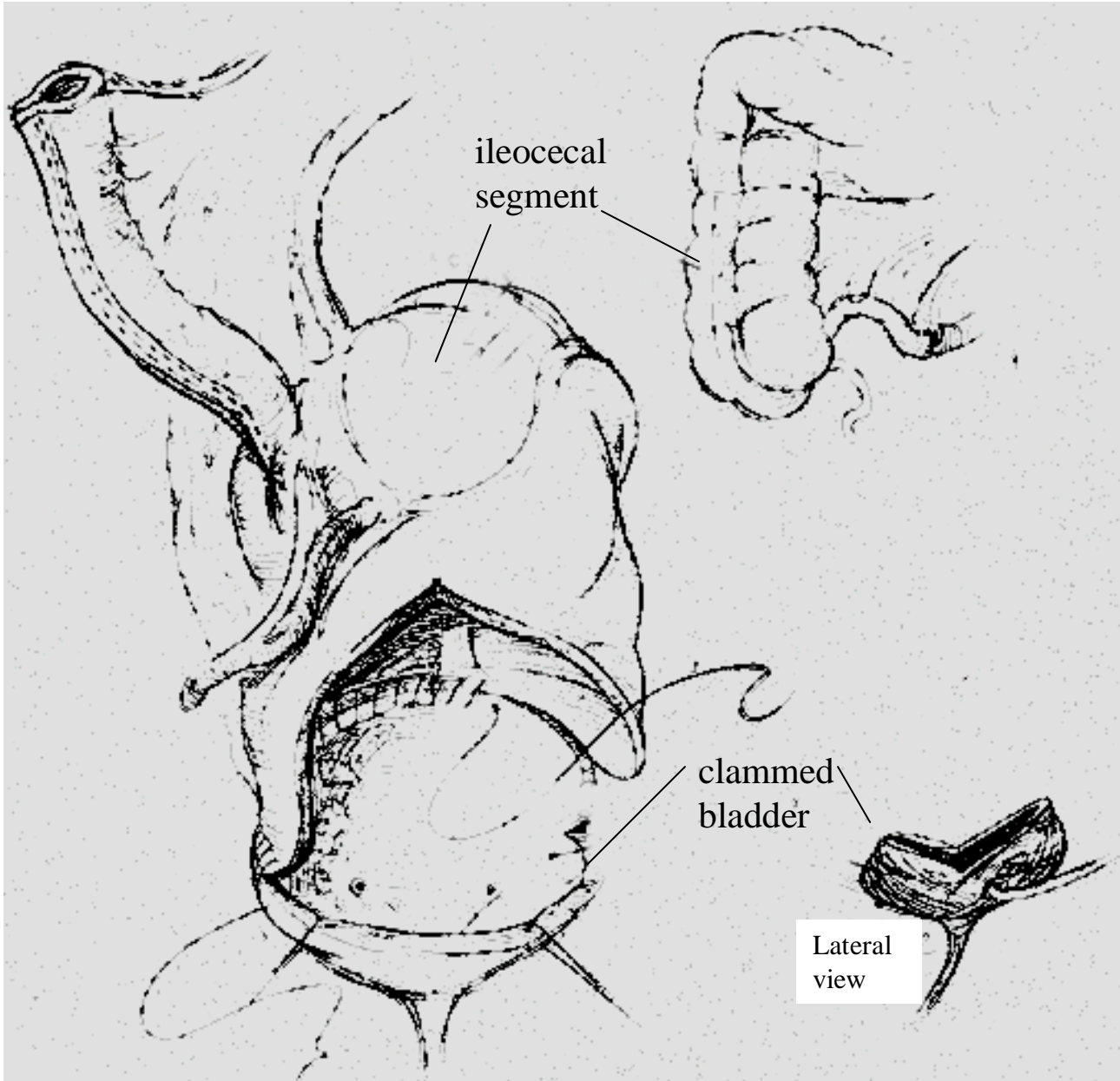
- Tetraplegic male who cannot maintain condom cath because of penile retraction
- Women without dexterity to self-cath
- An ileal conduit “bladder chimney” provides low-resistance to outflow
- Avoids complications of foreign body
- Risks less than cystectomy with ileointestinal anastomoses

Ileovesicostomy



Continent Augmentation Cystoplasty

- Utilize ileocecal segment
- Continent cutaneous stoma
- No ureteral reimplantation if no reflux
- Preserves fertility potential
- No transfer needed to do self cath
- No need for urethral catheterization although it's still available



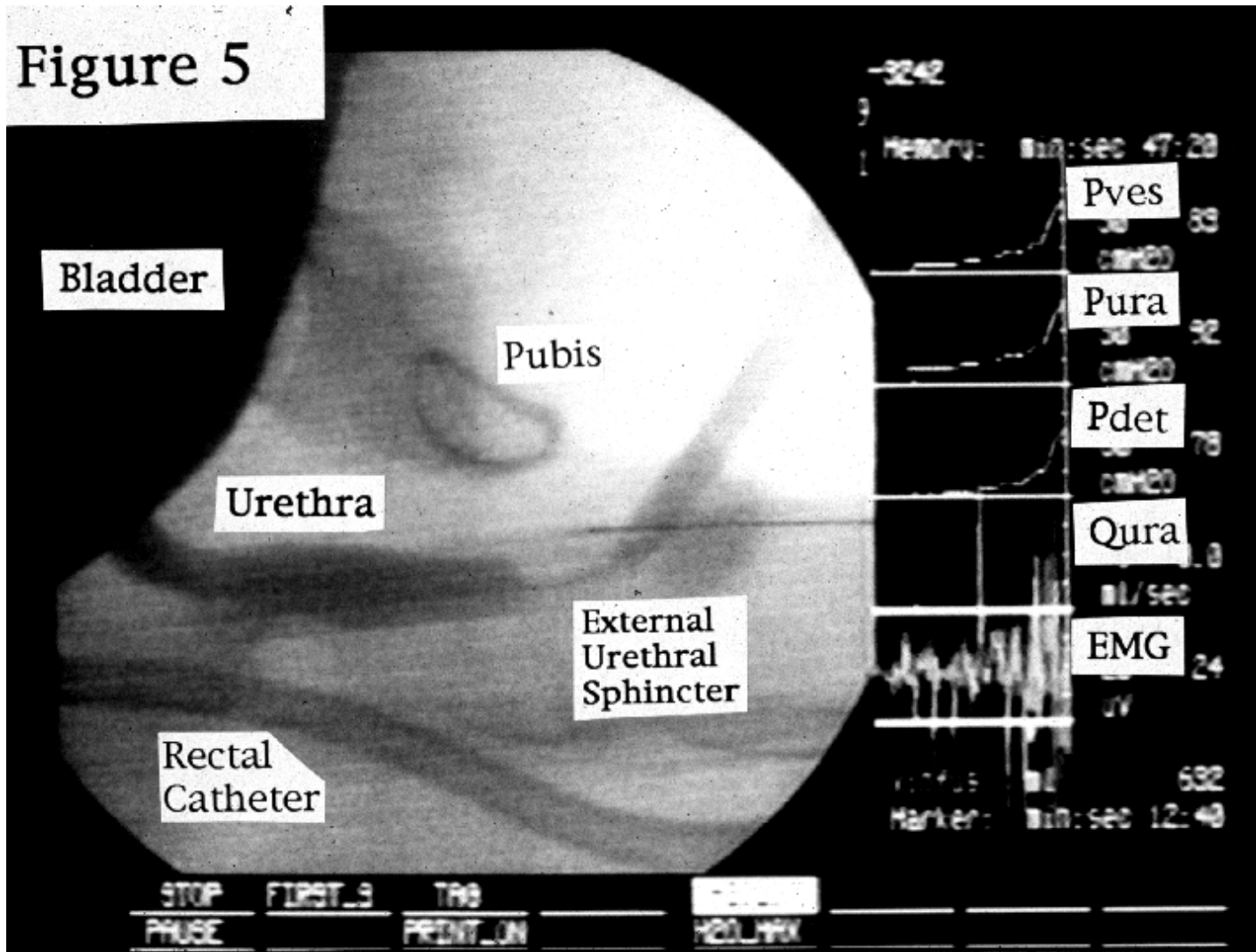


Surgical Options Failure to Store Due to Outlet Failure

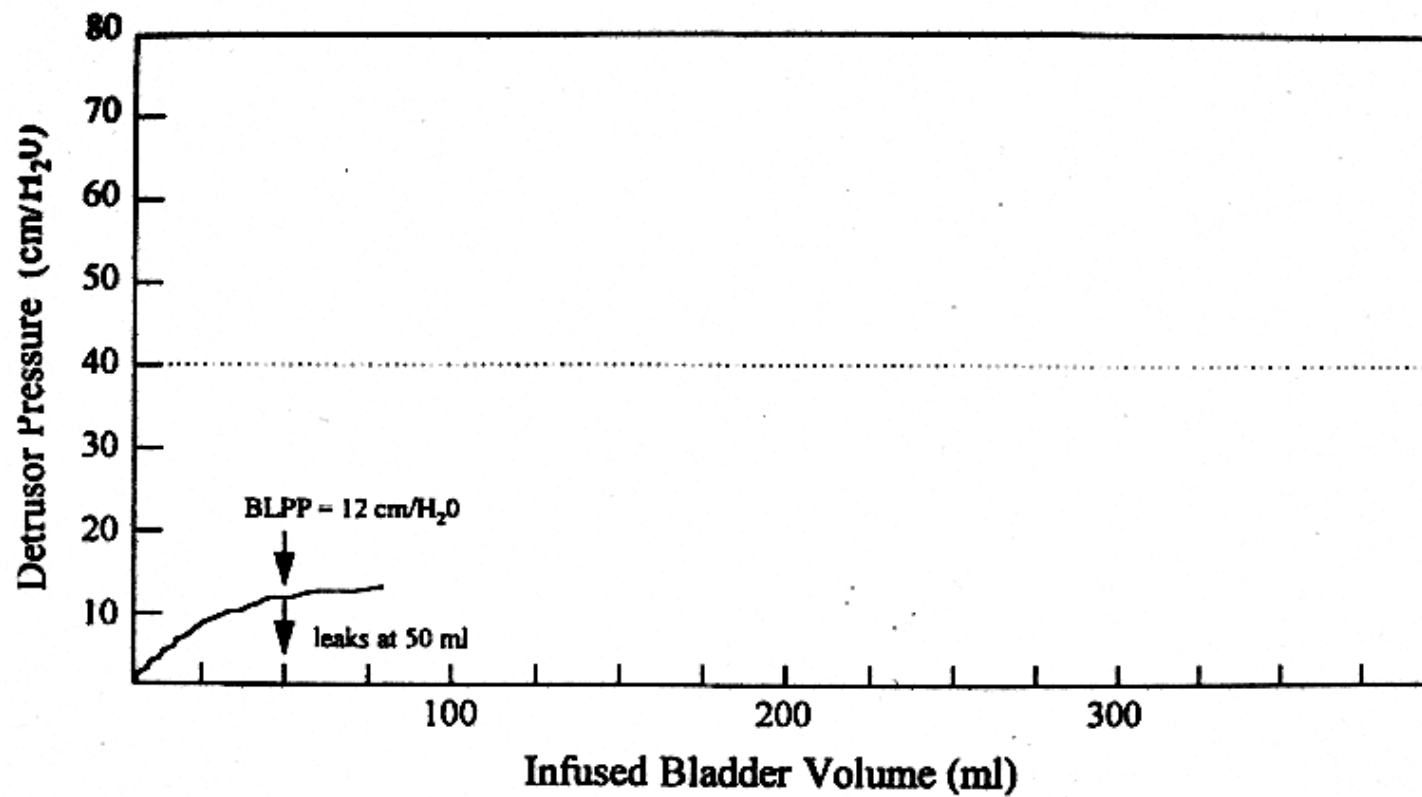
- Urethral implants: Injectables
- Bladder Neck Sling
- Artificial Urinary Sphincter
- Closure of Bladder Neck/Urethra

Surgical Alternatives: Failure to Empty

Figure 5



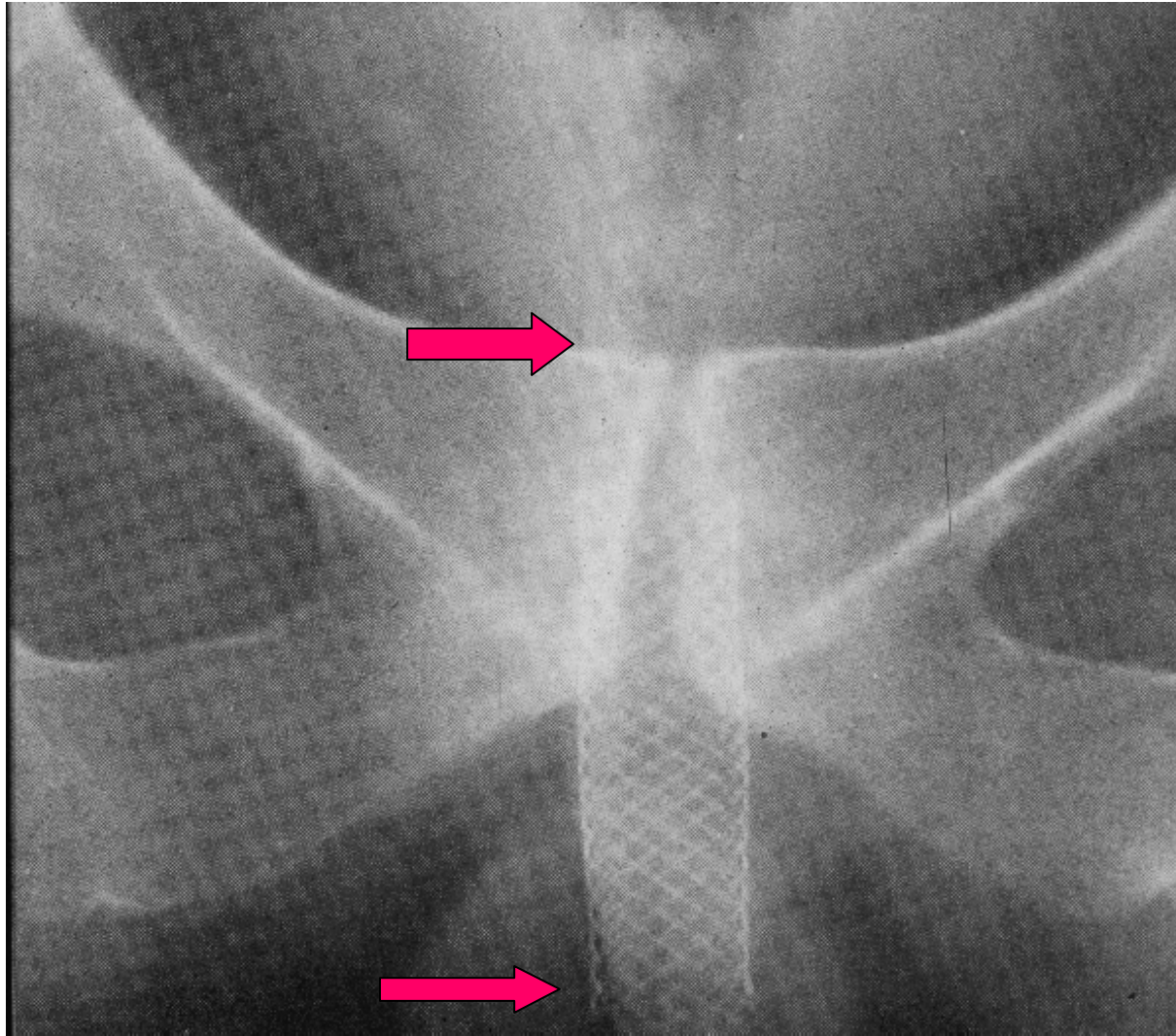
BLPP After Sphincterotomy



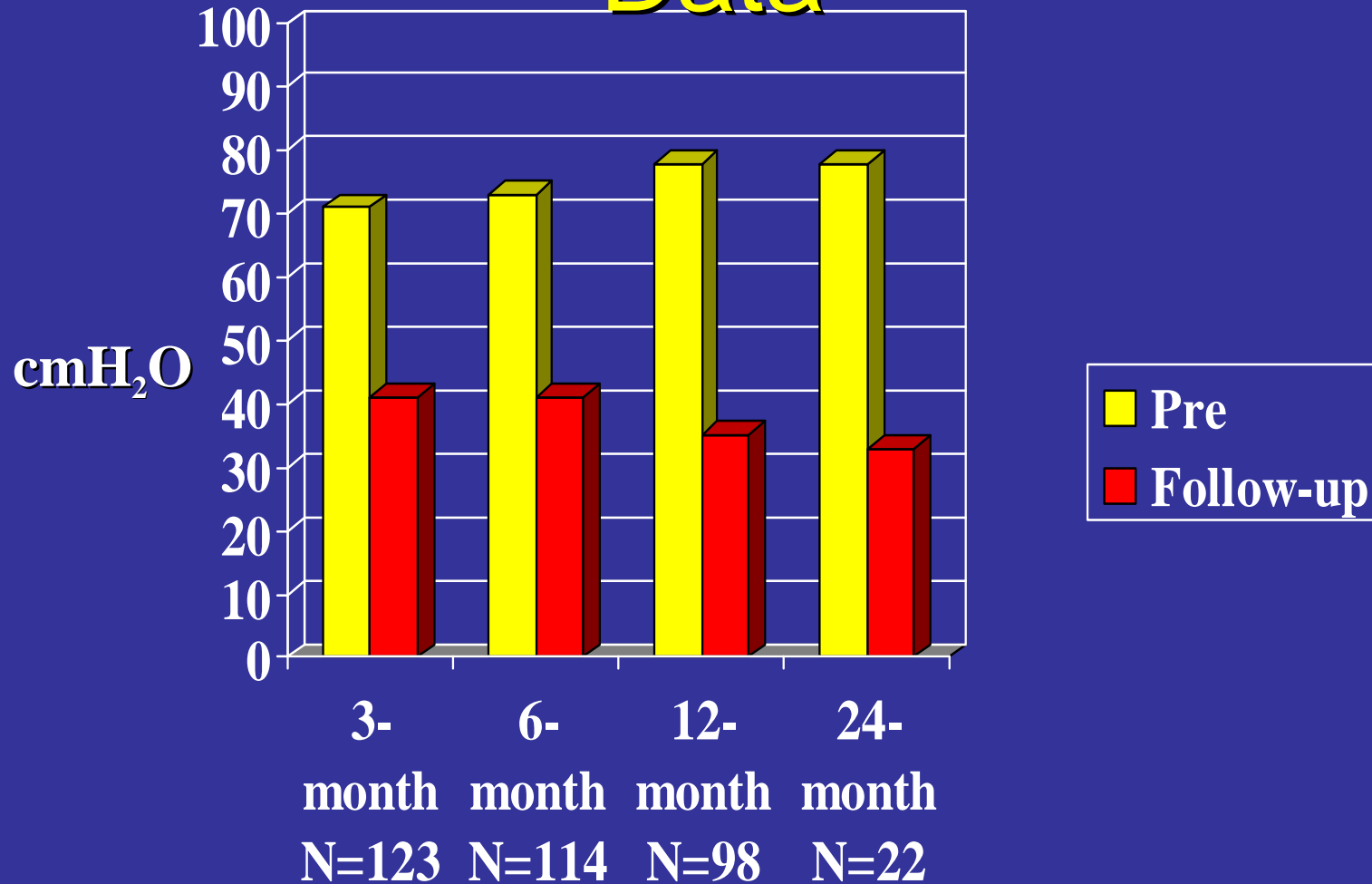
UroLume™ for DESD

- 160 patients (18-74 yrs) from 15 SCI Centers
- SCI (94%), MS (5%), Spinal Cord ischemia (0.5%), Spinal Cord tumor (0.5%)
- Mean SCI duration - 9 years (1-19 yr)
- Cervical (74%), Thoracic (22%), Lumbar (2%), Sacral (2%)
- 45/160 (28%) at least one TRUS

Urolume™ Stent for DESD



Voiding Pressure: Matched Data



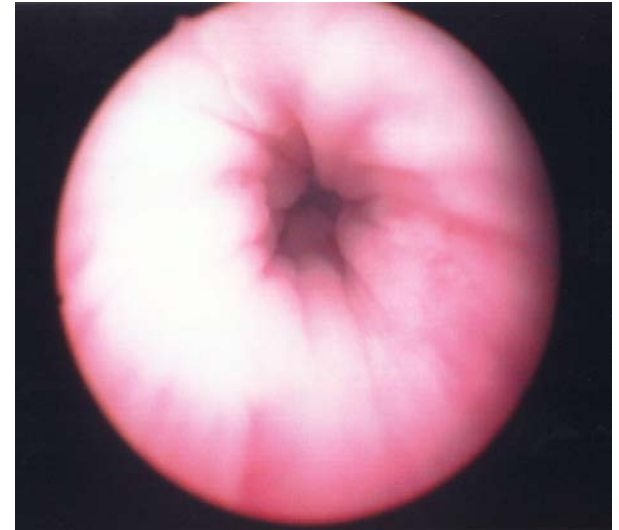
$p < 0.001$ Pre-Insertion vs. each follow-up period

Botulinum Toxin for Detrusor Sphincter Dyssynergia

Sphincter Injection Technique

Men

- Dilute 200 units BTX (Botox) with 4 ml saline
- Men: transurethral injections in the striated sphincter (25-G Cook® Williams needle at the 3, 6, 9 and 12 o'clocks)
- Flush with 0.3 ml of saline to not waste any toxin in needle



Urethral Treatment Outcome

<u>Urethral injection (N=68)</u>	<u>Pre treatment</u>	<u>Post injection (6 months)</u>	<u>p- value</u>
Retention require catheterization (percent)	41 (60%)	7 (12%)	
Residual urine volume (ml) (n=32)	240 _± 51	88 _± 53	0.01
Maximal voiding pressure (cmH ₂ O) (n=27)	81 _± 35	52 _± 21	0.01
Cystometric capacity (cmH ₂ O) (n=27)	198 _± 79	241 _± 61	0.54
Number of patients with stress urinary incontinence requiring pads	2	3	

n= patients of patient with data before and 6 months after BTX-A

Questions?

Slide 1

How Do We Promote
**Office-Based Care of the
Neurological Patient**

Michael J. Kennelly, MD, FACS
Carolinas Rehabilitation
Carolinas Medical Center
Charlotte, NC

Slide 2

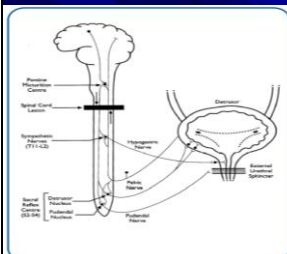
Office-Based Neurology
Overview

- Epidemiology
- Evaluation
- Barriers to Care
- Office Promotion
- Resources



Slide 3

Neuroanatomy of the LUT
Autonomic and Somatic



Coordination of micturition involves control by two main centres in the CNS:

(1) the **Pontine Centre** in the brainstem, which is responsible for co-ordinated activity between the detrusor and the bladder outlet, and

(2) the **Sacral Centre** which controls local reflexes and initiates detrusor contraction.

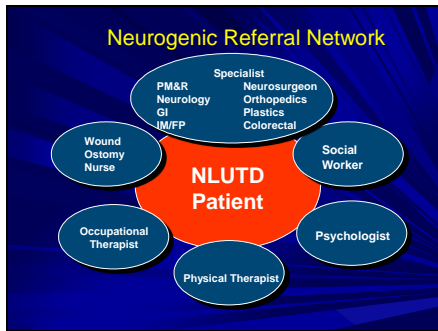
Spinal cord injury (SCI) disrupts descending motor and ascending sensory pathways, preventing normal control of micturition (illustrated in Figure 1).

Slide 43

What Can We Do to Promote Office Based Care?

- Be sensitive
- Train staff
 - Educational aspects of NLUTD
 - Technical skills training –
 - Transfer training
 - Catheter skills
 - Bowel program
 - Autonomic Dysreflexia
 - Sexual Devices
 - Referral Network

Slide 44



Slide 45

What Can We Do to Promote Office Based Care?

- Be sensitive
- Train staff
- Anticipate patients needs
- Optimize office environment
- Have adaptive equipment available
- Engage patient as an active participant
- Join and participate in SIG society & activities

Slide 52

Consumer Societies

www.nationalmssociety.org www.ucp.org www.parkinson.org

PARALYZED VETERANS OF AMERICA

www.pva.org

Slide 53

SCI Professional Societies

www.ascipro.org www.iscos.org

ASIA
AMERICAN SPINAL INJURY ASSOCIATION
www.asia-spinalinjury.org

Slide 54

Thank You
