Urinary Incontinence: Etiology, Pathophysiology

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1. Introduction

Urinary Incontinence (UI) is defined as the complaint of **involuntary loss of urine**. Multiple types of UI have been subcategorized in both men and women. The **most prominent types are stress urinary incontinence** (**SUI**), **urgency urinary incontinence** (**UUI**), **and mixed urinary incontinence (MUI)**. SUI is defined as the complaint of involuntary loss of urine with effort or physical exertion. UUI is the complaint of involuntary loss of urine associated with urgency and MUI encompasses symptoms of both urgency and stress. Additional types of urinary incontinence frequently encountered in urologic practice include overflow incontinence secondary to anatomic or functional obstruction, postural incontinence, nocturnal enuresis, continuous incontinence, insensible incontinence, and coital incontinence.[1,2] Specific information regarding the geriatric population, including transient causes of incontinence, may be reviewed in the Core Curriculum section "**Geriatrics: Urinary Incontinence and Voiding Dysfunction**."

Despite the prevalence and cost of UI many patients defer seeking help. Common misperceptions in the community include the idea that UI is a "normal" part of the aging process and that only limited and inadequate solutions are available.

UI also affects men although etiology and risk factors generally differ between genders. Although much of this section will be focused on female UI, evaluation and non-surgical treatment, the principles herein are generally applicable to either gender with the obvious exception of vaginal pessaries and devices.

Patient education materials describing general symptoms and causes of urinary incontinence are available through the **Urology Care Foundation**.

2. Epidemiology

The estimated prevalence of incontinence is subject to variations in definitions, **survey methods**, **and populations assessed**. Approximately 50% of women reporting UI indicated SUI as the primary or sole symptom of incontinence.[3] Data from the U.S. National Health and Nutrition Examination Survey (NHANES) confirm these approximations; between 2001 and 2004, **49.6% of women reported any UI**, with 49.8% reporting pure SUI, 34.4% reporting mixed UI (MUI), and

15.9% reporting pure urgency UI (UUI).[4] Among community-dwelling men, the prevalence of UI increased with age: 11% of men 60 to 64 years old reported UI versus 31% in men 85 years of age or older.[5] The prevalence of UI in institutionalized persons is markedly higher than in the general population, with rates reported at 10% to as high as 84% among adults in long-term care facilities.[6,7]

3. Costs

The estimated annual direct cost of UI in women and men in 1995 USD totaled over $16 billion, which increased to over $19 billion by the year 2000.[8,9,10,11] Over $12 billion was spent specifically for female incontinence, with 82% ($13.12 billion dollars) specifically associated with female SUI. Total spending was associated with routine care such as incontinence pads (70%), nursing-home admissions (14%), treatment (9%), complications (6%), and establishing a diagnosis (1%). Analysis of Medicare claims as part of the Urologic Diseases in America Project revealed a dramatic increase in spending between 1992 to 1998, with expenditures increasing from $128 million to $234 million.[12] More recent data highlights continued growth in the financial burden associated with UI, with estimated total national cost of UUI in 2007 of $65.9 billion, and projected costs of $76.2 billion in 2015 and $82.6 billion in 2020.[13]

The estimated annual cost of UI in men in the United States was $3.8 billion per year in 2001.[11] **A continually aging population will undoubtedly result in commensurate continued increases in the financial burden of UI.**

4. Risk Factors, Pathophysiology, and Evaluation

Associated risk factors and pathologic mechanisms contributing to UI are intimately dependent on the specific type of incontinence. In general, the pathophysiology of urinary incontinence can be divided into deficits of urine storage and/or urine emptying. **Common disorders are classified as ‘ failure to store’ due to bladder overactivity and/or sphincteric weakness and ‘‘failure to void’’ due to obstruction or hypocontractility**.[14] However, many aspects of detrusor and urethral function are inter-related, and several mechanisms provoking incontinence may overlap in any given patient.[15] Anatomic contributions of the pelvic floor musculature and load bearing structures such as supporting ligaments are superimposed upon the delicate neurovascular anatomy required for continence, therefore most UI is multifactorial.[16]

Common risk factors for UI during aging in both genders include neurologic disease, **diabetes**, **depression, dementia**, **chronic cough**, **recurrent cystitis**, **pharmacologic therapies**, **endocrine disorders**, **stool impaction**, **and reduced mobility**.[17,18] The impact of occult neurologic compromise with advancing age may encompass a substantial yet underrecognized component of the pathophysiology of geriatric incontinence.[19]

Further information regarding incontinence in the geriatric patient is addressed in the Core Curriculum section "**Geriatrics: Urinary Incontinence and Voiding Dysfunction**."

Common neurogenic causes of UI, addressed in detail in the Core Curriculum section "**Neurogenic**

Bladder", may include stroke, Parkinson's disease, multiple sclerosis, spinal cord injury, and diabetes. If neurologic disease is suspected, clinicians should address this prior to embarking on invasive treatment as prognosis and management of the condition may alter therapeutic decisions.[20] Findings that should raise suspicion for an undiagnosed neurologic condition include new onset/acute symptoms, incontinence in the absence of retention, insensate leakage, concomitant numbness/weakness, alterations in speech or gait, cognitive deficits, or autonomic dysfunction.[21]

Optimal treatment requires accurate classification of UI as SUI, UUI, or MUI with predominance of SUI or UUI. The basics of an evaluation for stress urinary incontinence include a thorough history including assessment of bother, general examination including neurologic assessment, focused pelvic/genital exam eliciting Valsalva to assess for SUI, post void residual assessment and urinalysis .[19,20] A voiding diary may provide additional information when formulating a treatment plan. In women, examination should include assessment of overall pelvic support (including the urethra), the integrity of the vaginal epithelium, presence of incontinence following provocative stress maneuvers (i.e. cough, Valsalva), voluntary contractibility of the pelvic floor muscles, evaluation of pelvic pain, and signs of inflammation and/or infection. A thorough genitourinary exam in male patients should include external examination to evaluate for anatomic abnormality, meatal scarring, signs of infection or inflammation, and an assessment of the prostate via digital rectal examination. Per the AUA, evaluation for patients with OAB and UUI included a thorough history, general physical exam and urinalysis. Assessment of post void residual is not required in the initial assessment for UUI, however it is so readily available and potentially informative to the urologist that it should be considered routine. [22] Further testing (e.g. cystoscopy, urodynamics) is not indicated before initiating conservative treatments unless patients or complex, there is concern for associated underlying conditions such as recurrent febrile urinary tract infection (UTI), bladder outlet obstruction (BOO), significant symptomatic pelvic organ prolapse (POP), neurogenic bladder, bladder stones, foreign bodies in the urinary tract, or bladder or pelvic tumors.[22] Further information can be found in AUA guidelines on management of **SUI** and **OAB**.

4.1 Risk Factors and Pathophysiology of UI in Women

The most common risk factors for UI include female gender and increasing age.[23] **Additional risk factors contributing to SUI in women include increased parity**, **higher body mass index** (**BMI**), **vaginal delivery**, **hormone replacement therapy**, **diabetes**, **and family history.**[24,25,26] There is emerging appreciation for the role of BMI in pelvic floor disorders; multiple epidemiological studies have established obesity as a strong independent risk factor for UI.[27]

The mechanical trauma and neurovascular disruption which occur during vaginal delivery result in anatomic laxity, compromising urethra and bladder neck support and contributing to development of SUI, frequently following the first delivery.[28] Traction and crush injury to the perineal branch of the pudendal nerve further contributes significantly to postpartum incontinence.[29] Additionally, pelvic organ prolapse related to such anatomic distortion may produce mechanical obstruction, leading to overflow incontinence, urethral hypermobility leading to SUI, and detrusor functional alterations

leading to UUI.[30,31]

AUA guidelines for **evaluation and surgical management of female SUI** are available.

Patient education materials describing SUI causes, symptoms and treatments are available through the **Urology Care Foundation**.

The contribution of race and ethnicity as risk factors for UI is uncertain. Some studies have reported higher incidence of UI in Caucasians compared to African-American or Asian women, whereas others have reported greater risk in African-American and Hispanic women.[32,33,34,35,3]

Urinary fistulas are a less common source of incontinence associated with substantial morbidity. **While the majority of genitourinary fistula in Western nations result from surgery or radiation therapy, obstetric trauma is the predominant culprit in developing nations**[36] Prolonged, obstructed labor results in the **"obstructed labor injury complex", consisting of pressure necrosis injuries to bowel, bladder, urethra, and vagina and neurologic; injury to pudendal innervation pathways**[37]

For further information regarding fistula disease, please refer to the Core Curriculum section **Urinary Fistulas**.

UUI is a symptom resulting from defects in the functional or sensory components of urinary storage. Often coupled to symptoms characteristic of "overactive bladder" (OAB) including frequency and nocturia, UUI may result from involuntary detrusor contractions.[38] Since UUI represents the manifestations of a symptom complex, assigning a single pathophysiologic mechanism is usually not feasible in women without a clear neurologic lesion (e.g. spinal cord injury, multiple sclerosis, etc.).[39]

Idiopathic UUI is associated with advancing age, **outlet obstruction**, **and pelvic floor disorders such as pelvic organ prolapse**.[40] MUI, encompassing symptoms of both SUI and UUI, commonly manifests in patients with both anatomic and functional deficits.[41] Guidelines for **evaluation and management of OAB and UUI are provided**.

Patient education materials describing OAB causes, symptoms and treatments are available through the **Urology Care Foundation**.

4.2 Risk Factors and Pathophysiology of UI in Men

In men, UI often results from either iatrogenic insult from radiation or surgical interventions, bladder outlet obstruction due to benign prostatic enlargement (BPE) or is secondary to urethral stricture (USD)/meatal disease. Bladder outlet obstruction (BOO) may be associated with detrusor overactivity, detrusor underactivity, and/or anatomic changes such as bladder trabeculations and diverticula. These developments may contribute to lower urinary tract symptoms (LUTS) including UUI.[42] **Overflow incontinence**, the involuntary leakage of urine associated with incomplete bladder emptying, may be a direct consequence of severe BOO from BPE or USD[15] however, overflow incontinence may be better characterized by its contributing components once they have been established by advanced evaluation (e.g. urinary retention with detrusor overactivity at large volumes,

altered compliance, etc.)

Incontinence after prostate treatment may include SUI, UUI, or MUI. These urinary conditions have a significant impact on patients' quality-of-life .[43,44,45] **Deficits in sphincteric function, known as intrinsic sphincter deficiency (ISD), represent the primary pathophysiology of post-prostatectomy incontinence, although detrusor instability may contribute or even predominate in select cases, particularly when there is a history of radiation or severe pre-prostatectomy storage lower urinary tract symptoms** . Overflow incontinence may also occur after prostate cancer treatment, often secondary to vesicourethral anastomotic stricture. Risk factors for post-prostatectomy incontinence include advanced age, prior transurethral prostate surgery, and adjuvant or neoadjuvant radiation.[46,47]

See the AUA/SUFU Guideline on **incontinence after Prostate Treatment**.

5. Non-surgical Management of Urinary Incontinence

A practical approach to the treatment of UI involves trial of conservative management before embarking on surgical intervention, as conservative therapies are often effective, well-tolerated, and safe.

Conservative therapies for UI include bladder retraining and behavioral therapy, dietary modifications, lifestyle changes, pelvic floor rehabilitation, vaginal pessaries, urethral inserts, and pharmacotherapy.[48] SUI that is refractory to conservative management may warrant surgical intervention with a sling or urethral bulking agent surgical interventions (see Core Chapters "**Surgery for Female SUI**" and "**Surgery for Male SUI**."). Refractory cases of overactive bladder (OAB) and UUI may warrant a trial of onabotulinumtoxinA, percutaneous tibial nerve stimulation/modulation or sacral nerve stimulation (see Core Chapter "Evaluation and Treatment of Overactive Bladder").[22,24]

6. Voiding Diaries

Voiding diaries are important in the treatment of UI for several reasons:

a. Keeping the diary involves the patient in their treatment program; this starts the educational process essential for behavioral therapy.
b. The diary provides semi-objective baseline information to precisely characterize the problem and measure treatment efficacy.
c. The largest recorded volume may be used as a functional, non-invasive estimate of bladder capacity.
d. The time between recorded voids may be used as an initial voiding interval for bladder retraining programs.

7. Behavioral Therapy

Behavioral therapy is based on *operant learning,* which is intended to model a desirable activity to reproduce a normal behavior. In this case, the normal and desirable behavior is urinary control.

Patients should be educated on anatomy and function of the lower urinary tract and how continence is maintained in the normal state. **Patients should also be educated on factors contributing to their UI**.

Bladder training is recommended as a first line treatment for UI.[25,26,28,49,50,51] The goal is to regain urinary control and prevent or inhibit severe urgency symptoms. The fundamental goals of bladder training include voluntary timed voiding prior to severe urgency and breaking the learned cycle of responding to the first sense of urinary urgency. Repeated pelvic floor muscle contractions, or "quick flicks", inhibit urgency by activating a spinal reflex that induces inhibition of the detrusor muscle, resulting in relaxation of the bladder. **Patients are advised to *STOP* whatever they are doing**, ***SIT*** **if possible**, ***SQUEEZE*** **their pelvic floor muscles in rapid quick succession**, **and *DISTRACT*** **themselves until the urgency subsides**.

A normal voiding frequency is every three to four hours during the day. Bother usually becomes significant when frequency approaches every 2 hours or more.[52] Although sleeping the night through is most desirable, getting up once during the night to void is of minimal bother to most and considered normal. [53] Timed voiding is the process of urinating at a fixed interval to preempt urgency symptoms. The initial interval can be set based on the shortest time between voids as recorded in a bladder diary or empirically at every 2-3 hours. Variations of timed voiding include scheduled voiding – for cognitively intact independent individuals - and prompted voiding – for cognitively impaired, dependent and/or institutionalized individuals.

8. Dietary Modifications

Except for fluid management and weight loss for overweight individuals, most dietary and lifestyle recommendations for managing UI have not been well-studied. Nevertheless, common sense strategies often prove helpful.

Daily fluid intake of up to 2 Liters/day is generally recommended with adjustment for body size and insensible losses (e.g. sweating). [54] Fluid restriction has previously been advocated for management of both SUI and OAB/UUI. However, fluid restriction may increase problems with urinary tract infections and constipation and in extreme cases may progress to dehydration and hypotension. Fluid restriction will also produce more concentrated urine which has been postulated to act as a bladder irritant that may in turn initiate detrusor overactivity.

High caffeine intake (> 400 mg/day average) is associated with detrusor overactivity on urodynamic testing and therefore is a likely cause of OAB symptoms.[29,55] An association between reductions in caffeine consumption and UI symptoms has been reported in several small studies.[29,30,56,57] Therefore, reduction of caffeine is advised for OAB patients to reduce UI.

Many other beverages and foods have been suggested as "bladder irritants", but definitive data are generally lacking.[32,33,58,59] It is reasonable for patients to do a step-wise trial of elimination (one food/beverage at a time) to identify dietary factors that may exacerbate their symptoms. Specific items that have been postulated to trigger bladder symptoms include:

- Alcoholic beverages
- Coffee or tea (with or without caffeine)
- Carbonated beverages
- Other items noted in the literature but without clear supporting evidence:
    - citrus juices/fruits,
    - tomato-based products,
    - spicy foods,
    - artificial sweeteners,
    - chocolate,
    - sugar/honey/corn syrup

9. Weight Loss

Obesity and *weight gain* are important risk factors for UI, and *weight loss* is an effective treatment.[27,34,35,3,60,61] Epidemiologic data demonstrate a linear relationship and causal association between obesity (body mass index [BMI]and waist circumference) and UI in women.[36,62] **Weight gain is an independent risk for incident SUI and UUI**.[62] **Women who gain 5 to 10 kg in body weight after age 18 have a 44% higher chance of weekly UI compared to women who stay within 2 kg of their baseline weight**; **this relationship persists regardless of initial weight**. The risk was 4-fold greater in women who gained 30 kg or more. Moderate weight loss (average of 8%, 7.8 kg) in overweight or obese women (BMI > 25) with severe UI (at least 10 UI episodes/week) was associated with a significant reduction (47%) in UI episodes.[22,27]

10. Exercise

Aerobic exercise is independently associated with reduced risk of UI in middle aged and older women.[25,49] **In women aged 37-54 years**, **the protective effect of moderate exercise** (**mainly daily walking**) **can be seen for both SUI and UUI**;[36,62] **in women aged 54-79 years**, **the protective effect was present for UI and SUI**, **but not UUI**.[38,63]

11. Smoking Cessation

Smoking is thought to be a risk factor for SUI by increasing coughing episodes and for OAB by causing bladder irritation from nicotine and toxins excreted in the urine. However, there is inadequate data to support the theory that smoking cessation effectively prevents or treats UI symptoms.[25,33,49,59] **Although studies on the effects of smoking and UI are limited, tobacco use has numerous harmful health effects and other urologic implications and should be discouraged in all patients irrespective of UI symptoms**.

12. Treating Constipation

Constipation and chronic straining have been associated with UI and OAB.[25,49] **Maintenance of a regular bowel program with appropriate daily dietary fiber is essential to the success of any**

urinary program. For those in whom diet is not adequate, daily use of a fiber supplement and polyethylene glycol (PEG) may be beneficial in relieving constipation, although the benefit of PEG specifically for OAB symptoms relative to placebo has not been established.[64]

13. Treating Associated Depression

Patients with UI are almost three times more likely to have major depression than those without (6.1% vs. 2.2%). Patients with both UI and depression have significantly greater decrements in quality-of-life and functional status than those with UI alone.[65,66] **Appropriate referrals for depression management should be made when indicated in patients with UI**.

14. Pelvic Floor Muscle Training

Pelvic floor muscle rehabilitation involves education on the anatomy and function of the pelvic floor muscles and strength training to control lower urinary tract function.[67] Pelvic floor exercises (PFE, frequently referred to as *Kegels)* have been recommended for the treatment of UI since the 1950's. The preferred term now is pelvic floor muscle training (PFMT), which emphasizes the importance of an on-going training regimen over time. As with any exercise regimen, PFMT should be sustained indefinitely to preserve the benefits.[68,69]

Improvements in continence with PFMT are thought to occur through increasing strength, power, speed and responsiveness of the pelvic floor muscles to improve overall coordination of pelvic muscle functions, including continence.

PFMT is defined as any program of repeated voluntary pelvic floor muscle contractions taught by a health care professional and should involve the most intensive PFMT supervision possible.[70]

A standard starting regimen for PFMT includes:[49]

a. Three sets of 8-12 near-maximal slow velocity contractions of the pelvic floor.
b. Each contraction should be held 6-8 seconds with an equivalent rest period between contractions.
c. At the end of each set a series of 5-10 rapid contractions are performed.
d. Exercises performed daily or every other day for 12 – 20 weeks.

PFMT is safe and efficacious based on numerous trials and should be offered as first line therapy to all women with UI (SUI, UUI and mixed UI).[49,70] **Although surgery is the single most effective treatment for SUI there is a 40-50% chance that women can achieve satisfactory control of UI through PFMT and avoid an operation.**[49]

Success with PFMT is more likely in patients with milder degrees of UI. **Independent risk factors for failure of PFMT include two or more leaks per day, presence of leakage at first cough, and use of antidepressant and/or anxiolytic medications.**

15. Biofeedback

Biofeedback is the use of electronic monitoring of an automatic bodily function to acquire voluntary control of that function. In the case of UI, pelvic muscle activity is assessed, and feedback given to the patient to aid in identification and selective contraction of the appropriate muscles. This teaches the patient how to interpret and respond to sensations of urgency or leakage that may contribute to UI.

Devices commonly used to assist with biofeedback include:

- Direct palpation of the appropriate muscles during contraction by a skilled physical therapist.
- Vaginal weighted cones for tactile feedback during contractions.[71]
- Perineometers to measure vaginal or anal pressure during squeezing; these devices may be confounded by transmission of abdominal Valsalva pressure to the probes.[68,69]

Electromyographic (EMG) probes (vaginal/anal/perineal) measure electrical activity during muscular contractions. Visual and/or auditory feedback is provided corresponding to those contractions.

16. Passive Pelvic Floor Exercise Modalities

Passive pelvic floor exercise modalities require no voluntary pelvic muscle contraction ability. There are 2 modalities of passive pelvic floor exercises.

Electrical Stimulation – Electrical stimulation is most commonly a home therapy. Since the activity is passive, it is dependent only on patient compliance, not effort. The patient can use vaginal, anal, or perineal electrodes to administer the stimulation. For treatment of SUI, high-frequency stimulation (50-200 Hz) is recommended with vaginal or anal electrodes as this frequency induces contraction of the pelvic floor and urethral muscles. For treatment of OAB/UUI, low-frequency stimulation (5-20 Hz) is recommended since this frequency activates inhibitory bladder reflexes.

Magnetic Stimulation is done in the office with the use of a magnetic chair. The patient sits in the treatment chair that emits electromagnetic waves focused on the pelvic floor muscles. The treatments consist of 20 min sessions (with both high and low frequency stimulation) 2 times per week. The advantages of this therapy are that it is non-invasive, the patient wears street clothes, and the device can be operated by a technician with minimal training required. The disadvantages are the time commitment with twice-weekly session in the office and cost. Small trials to date suggest that extracorporeal magnetic stimulation is better than control/sham for objective, voiding diary, and urodynamic outcomes and quality-of-life without adverse events.[72,73]

Acupuncture is typically performed in an office or hospital setting and requires multiple visits. Its efficacy in reducing one-hour pad weight in women with SUI vs sham was demonstrated in a pilot RCT and efficacy in improving quality of life and urodynamic outcomes in two RCTs evaluating OAB and UUI.[72,74]

17. Devices

Pessaries can be used to treat both SUI and pelvic organ prolapse (POP).[47,75] **Vaginal support**

devices used to treat SUI – with or without concomitant POP -- typically have a knob or protrusion that is oriented anteriorly in the distal vagina at the level of the proximal urethra to limit urethral hypermobility and promote urethral closure . For women who have the manual dexterity and body habitus to remove the pessary, maintenance involves removal of the pessary two nights per week to decrease the incidence of pessary-related infections or vaginal epithelial erosion. Women who are unable to remove the pessary themselves may leave the device *in situ* for up to 3-month intervals, with removal in the office by a provider and inspection of the vaginal tissues by speculum exam. **When not otherwise contraindicated**, **it is recommended that post-menopausal women who use a pessary also use local vaginal estrogen** (**vaginal cream**, **suppository**, **ring**) **to reduce the risk of vaginal infection and erosion**. Contraindications and serious complications with the use of pessaries are very rare. The Poise Impressa® is an FDA approved single-use, intravaginal device that in a small study was demostrated to be well-tolerated and effective in reducing SUI on pad weight test.[3]

18. Pharmacological Therapy

Antimuscarinic medications have been the mainstay of pharmacological therapy for OAB and UUI for many years (treatments for refractory OAB/UUI are discussed in depth in the Core Curriculum section "**Evaluation and Treatment of Overactive Bladder**.") They act primarily by blocking cholinergic receptors in the bladder to allow for an increase in bladder capacity and a decrease in uninhibited detrusor contractions.

There are a number of oral antimuscarinics on the market available in different doses and formulations (e.g. oxybutynin, tolterodine, fesoterodine, solifenacin, darifenacin and trospium) as well as a transdermal oxybutynin gel and an oxybutynin patch, marketed briefly for over the counter use in women (Oxytrol®).[76] **Comparisons between the FDA-approval studies for these antimuscarinic agents note similar efficacy**.[4] The choice of an agent depends on the cost, drug interactions and potential side effects. **Common side effects of antimuscarinic medications include dry mouth**, **constipation**, **blurred vision and cognitive impairment in the elderly**. **Antimuscarinics are contraindicated in patients with gastric retention and closed-angle glaucoma**. Overall discontinuation rates are high at 58-71%.[5,77] A recent 2012 systematic review of randomized trials noted only a small overall benefit of antimuscarinics compared to placebo for the reductions of OAB symptoms and UUI. [78,79]

A beta-agonist (mirabegron) was FDA-approved for management of OAB in 2012. Mirabegron has a lower incidence of anti-holinergic side effects (e.g. dry mouth and constipation) and similar efficacy to antimuscarinic drugs. It should be used with caution in patients who have uncontrolled hypertension. [80,81] Its efficacy appears to be similar to antimuscarinics with better tolerability. Vibegron, a novel beta-3 adrenergic receptor agonist recently approved by the FDA, has similar efficacy to antimuscarinics without the added side effect of impacting the cardiovascular system.[82] **Evidence suggesting poorer cognition and increased risk of dementia with anticholinergic medication is a concern**,[83,84,85] suggesting that beta-3 agonist may be the preferred initial medical

therapy of choice in elderly adults in the absence of contraindications or prohibitive cost.

Medical therapy should be considered early on in patients with a low maximal voided volume on a voiding diary , **patients with underlying neurologic disease**, **and patients who are unwilling or unable to participate in behavioral techniques**. Drug therapy alone is rarely curative and should optimally be initiated in conjunction with behavioral therapy and a robust PFMT program.[86,87]

Treatments for refractory OAB/UUI are discussed in depth in the Core Curriculum section "**Evaluation and Treatment of Overactive Bladder**."

19. Conclusion

UI is a prevalent problem that requires attention from primary care providers and specialists. Proper diagnosis with respect to type of UI is the first step to identifying an appropriate treatment algorithm but is not always apparent on history and physical examination. Many conservative treatment modalities exist which are safe and effective. Procedural and operative interventions exist where conservative management fails. Treatment should be consistent with patient bother, comorbidities, and tolerability for associated risks and financial costs.

20. Abbreviations

BMI: Body Mass Index
EMG: Electromyographic
FDA: Food and Drug Administration
BPE: Benign prostatic enlargement
BOO: Bladder outlet obstruction
ISD: Intrinsic sphincteric deficiency
USD: Urethral stricture disease
MUI: Mixed Urinary Incontinence
NHANES: National Health and Nutrition Examination Survey
OAB: Overactive Bladder
PFE: Pelvic Floor Exercises
PEG: Polyethylene glycol
PFMT: Pelvic Floor Muscle Training
POP: Pelvic Organ Prolapse
PTNS: Percutaneous Tibial Nerve Stimulation
Sacral Nerve Stimulation: Sacral Nerve Stimulation
SUI: Stress Urinary Incontinence
UI: Urinary Incontinence
UTI: Urinary Tract Infection
UUI: Urgency Urinary Incontinence

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Presentations

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