Urinary incontinence (UI) is a multifactorial syndrome produced by a combination of genitourinary pathology, age-related changes, and comorbid conditions that impair normal micturition or the functional ability to toilet oneself, or both.

PREVALENCE AND IMPACT

The prevalence of UI increases with age and affects women more than men (2:1) until age 80, after which men and women are equally affected. Of persons aged 65 years and over, 15% to 30% in the community and at least 50% in long-term care are incontinent. UI can cause morbidity, including cellulitis, pressure ulcers, urinary tract infections, falls with fractures, sleep deprivation, social withdrawal, depression, and sexual dysfunction. UI is not associated with increased mortality. UI impairs quality of life, affecting the older person’s emotional well-being, social function, and general health. Incontinent persons often manage to maintain their activities, but with an increased burden of coping, embarrassment, and poor self-perception. Caregiver burden is higher with incontinent older persons, which can contribute to decisions to institutionalize. Estimated annual UI-related costs total more than $36 billion.

THE PATHOPHYSIOLOGY OF INCONTINENCE

Normal Micturition

For older persons in particular, continence requires mobility, manual dexterity, the cognitive ability to recognize and react to bladder filling, and the motivation to stay dry.

Bladder smooth muscle (the detrusor) contracts via parasympathetic nerves from spinal cord levels S2 to S4. Urethral sphincter mechanisms include proximal urethral smooth muscle (which contracts with sympathetic stimulation from spinal levels T11 to L2), distal urethral striated muscle (which contracts via cholinergic somatic stimulation from cord levels S2 to S4), and musculofascial urethral supports. In women, these supports form a two-layered “hammock” that supports and compresses the urethra when abdominal pressure increases. Micturition is coordinated by the central nervous system: Parietal lobes and thalamus receive and coordinate detrusor afferent stimuli; frontal lobes and basal ganglia provide signals to inhibit voiding; and the pontine micturition center integrates these inputs into socially appropriate voiding with coordinated urethral relaxation and detrusor contraction until the bladder is empty. Urine storage is under sympathetic control (inhibiting detrusor contraction and increasing sphincter tone), and voiding is parasympathetic (detrusor contractor and relaxation of sphincter tone).

Age-Related Changes

Age-related changes in the lower urinary tract and micturition (Table 20.1) are found in both continent and incontinent older persons. Why some older persons develop UI and others do not remains unclear; differences in lower urinary tract (LUT) and non-LUT compensatory mechanisms may play a role.

Risk Factors

Any condition, medication, or factor that affects lower urinary tract function, volume status and urine excretion, or the ability to toilet can predispose a person to UI. Causation is suggested by temporal links between such factors and the development or worsening of UI. Risk factors in community-dwelling older persons include advanced age, parity, depression, transient ischemic attacks and stroke, congestive heart failure, fecal incontinence and constipation, obesity, chronic obstructive lung disease, chronic cough, diabetes mellitus, impaired mobility, and impaired activities of daily living. Among institutionalized older
persons, UI is associated with impaired mobility, depression, stroke, diabetes mellitus, and Parkinson’s disease; at least one third have multiple conditions. Table 20.2 lists the mechanisms by which these conditions may impair continence. Although moderate to severe dementia is associated with UI, even severely demented persons remain continent if they have mobility for transfers. Thus, UI in demented persons may not be caused by dementia, but may be a multifactorial epiphenomenon with treatable causes.

**CLINICAL TYPES OF INCONTINENCE**

Incontinence can be classified into diagnostic clinical types that are useful in planning evaluation and treatment.

**Transient Incontinence**

UI precipitated by remediable factors is called *transient incontinence*. Transient UI affects approximately one third of community-dwelling older persons and accounts for one half of the incontinence among hospitalized older persons. The causes of transient incontinence are summarized in Table 20.3. Although transient UI is more likely in persons with lower urinary tract abnormalities, correction of the precipitating factor remains effective without abolishing the specific abnormality.

**Urge Incontinence**

Urge UI is the most common type of UI in older persons. It is characterized by abrupt urgency, frequency, and nocturia; the volume of leakage may be small or large. The term *overactive bladder* refers to a condition with frequency, nocturia, and urgency or urge UI, or both. Urge UI is associated with detrusor overactivity (DO) that may be age-related, idiopathic, secondary to lesions in central inhibitory pathways (eg, stroke, cervical stenosis), or due to local bladder irritation (infection, bladder stones, inflammation, tumors). Because DO is found in healthy, continent older persons, failure of lower urinary tract and functional compensatory mechanisms may play an important role in UI. Distinctions between detrusor hyperreflexia (DO associated with central nervous system lesions) and detrusor instability (DO without such lesions) are frequently blurred in the older persons. Less common causes of urge UI are interstitial cystitis (urge UI with otherwise unexplained pelvic pain) and spinal cord injury, which results in impaired detrusor compliance (excessive pressure response to filling) or detrusor-sphincter dyssynergia (concomitant detrusor and sphincter contraction), or both.

DO may coexist with impaired detrusor contractility (detrusor hyperactivity with impaired contractility, or DHIC), with an elevated postvoid residual volume (PVR) in the absence of outlet obstruction. DHIC accounts for most established UI in frail older persons. Women can be misdiagnosed with stress UI if weak DHIC contractions are not detected, and men can be misdiagnosed with outlet obstruction because of the similarity of the symptoms (urgency, frequency, weak flow rate, and elevated residual urine). Persons with DHIC may be at increased risk for urinary retention if treated with bladder-relaxant drugs.

**Stress Incontinence**

Stress UI, the second most common type of UI in older women, results from failure of the sphincter mechanism(s) to preserve outlet closure during bladder filling. Stress UI occurs coincident with increased intra-abdominal pressure, in the absence of a bladder contraction. Leakage is due to impaired pelvic supports or, less commonly, failure of urethral closure; the latter intrinsic sphincter deficiency occurs with trauma and scarring from anti-incontinence surgery in women and prostatectomy in men, or with severe urethral atrophy. Unlike the episodic leakage of genuine stress UI, this leakage is typically continual and can occur while the person is sitting or standing quietly. Stress maneuvers may trigger DO; with such stress-related urge UI, leakage occurs after a several-second delay following the stress maneuver.

**Overflow Incontinence**

Overflow UI results from detrusor underactivity, bladder outlet obstruction, or both. Leakage is small in volume but continual. The PVR is elevated, and symptoms include dribbling, weak urinary stream, intermittency, hesitancy, frequency, and nocturia. Associated urge and stress leakage may occur. Rarely, continual leakage is due to extraurethral incontinence (eg, cystovaginal fistula).
Outlet obstruction is the second most common cause of UI in older men; most obstructed men, however, are not incontinent. Causes include benign prostatic hyperplasia, prostate cancer, and urethral stricture. In women, obstruction is uncommon and usually due to previous anti-incontinence surgery or a large cystocele that kinks the urethra.

Detrusor underactivity causing urinary retention and overflow UI occurs in only 5% to 10% of older persons. Intrinsic causes are replacement of detrusor smooth muscle by fibrosis and connective tissue (eg, with chronic outlet obstruction). Neurologic causes include peripheral neuropathy (from diabetes mellitus, pernicious anemia, Parkinson’s disease, alcoholism) or mechanical damage to the spinal detrusor afferents by disc herniation, spinal stenosis, or tumor.

**ASSESSMENT OF URINARY INCONTINENCE**

The multifactorial nature of UI in older persons requires a comprehensive diagnostic evaluation, with a careful search for all possible causes and precipitants beyond a focus on specific genitourinary diagnoses. Table 20.4 lists the key points in the evaluation of UI in older persons. The evaluation must determine the cause(s) of UI and exclude serious conditions.

Routine urodynamic testing is optional and usually is not needed. Precise diagnosis is most important when surgical treatment is being considered for stress UI or outlet obstruction, because surgery is ineffective for DO, DHIC, and detrusor weakness that present with similar symptoms. Geriatric UI is multifactorial, and lower urinary tract pathology is rarely the only cause. A focus on urodynamic diagnosis detracts from more relevant precipitants. Moreover, some treatments are effective for several types of UI (see below). Thus, urodynamics should be considered only before surgical intervention, if the diagnosis is unclear, or when empiric therapy has failed. Cystometry determines only bladder proprioception, capacity, detrusor stability, and contractility; carbon dioxide cystometry may be unreliable. Simultaneous measurement of abdominal pressure is necessary to exclude abdominal straining and detect DHIC. Fluoroscopic monitoring, abdominal leak-point pressure, or profilometry tests detect and quantify stress UI. Pressure-flow studies detect obstruction. Peak urine flow rates \( \geq 12 \text{ mL per second} \) without straining for voids of at least 150 to 200 mL reliably exclude obstruction. Low flow rates are nondiagnostic, and precise diagnosis requires urodynamic evaluation.

Bedside cystometry may detect DO and measure bladder capacity. The PVR is measured by catheterization; then the bladder is filled through a syringe attached to the catheter. DO is identified by a rise in fluid level in the syringe column. Although bedside cystometry has moderate sensitivity and specificity for DO in ambulatory older persons, its true utility is unclear. Among community-dwelling older persons, its benefit over history and physical examination is unknown. When it is used in long-term-care patients, low-pressure DHIC contractions can be missed, and it may be difficult to differentiate DO from abdominal straining.

**MANAGEMENT**

**Overview**

Because age-related changes render older persons more vulnerable to developing UI from factors such as medical illnesses and medications, correction of those factors alone often improves continence. Relieving the most bothersome aspects of UI for the patient is key. A stepped strategy moving from least to more invasive treatments should be used, with behavioral methods tried before medication, and both tried before surgery. Treatment that simply decreases the number of UI episodes may not be sufficient for persons most bothered by the timing of UI, nocturia, or leakage with exercise. Cure often requires multiple visits. Evidence for the efficacy of UI treatment is summarized in Table 20.5, Table 20.6 and Table 20.7.

**General Measures**

Fluid management includes avoiding caffeinated beverages and alcohol, and minimizing evening intake if nocturnal UI is bothersome. Constipation should be reduced. If pads and protective garments are used, they should be chosen on the basis of gender and the type and volume of UI. Because these products are expensive, some patients may not change pads frequently enough. Medical supply companies and patient advocacy groups publish illustrated catalogs for product selection.
Urge Incontinence

Behavioral treatment for urge UI employs two principles: frequent voluntary voiding to keep bladder volume low, and retraining of central nervous system and pelvic mechanisms to inhibit detrusor contractions and leakage. Cognitively intact persons can use bladder retraining, with timed voiding while awake and suppression of precipitant urges by relaxation techniques. The initial toileting frequency (based on a voiding record) uses the shortest interval between voids. When a precipitant urge occurs, patients are instructed to stand still or sit down and concentrate on making the urge decrease and pass: to take a deep breath and let it out slowly, or to visualize the urge as a wave that peaks and then falls. Once in control of the urge, they should walk slowly to a bathroom and void. After 2 days without leakage, the time between scheduled voids is increased by 30 to 60 minutes until the person voids every 3 to 4 hours without leakage. Although its marginal benefit is unproven, many experts find biofeedback an important adjunct to improve teaching and execution of pelvic muscle exercises to control urge UI. Successful bladder retraining usually takes several weeks; patients need reassurance to proceed despite any initial failure. (See the Appendix, for an example of patient instructions for bladder retraining and bladder urge control.)

For cognitively impaired patients, behavioral methods include habit training (timed voiding, with the interval based on a person’s usual voiding schedule), scheduled voiding (timed voiding usually every 2 to 3 hours), and prompted voiding. Prompted voiding has three components: regular monitoring with encouragement to report continence status, prompting to toilet on a scheduled basis, and praise and positive feedback when the person is continent and attempts to toilet. Persons most likely to respond to prompted voiding are those with who void fewer than four times every 12 hours during the day and those who toilet correctly over 75% of the time in an initial trial. These methods require training, motivation, and continued effort by patients and caregivers; special attention and staff reinforcement is needed in institutionalized settings to ensure continued treatment success.

When behavioral methods alone are unsuccessful, bladder-suppressant medications can be added. Although medications usually do not abate DO, they can improve UI and frequency. Drug choice is based on efficacy, side effects, and comorbid conditions (eg, avoiding anticholinergic effects for a person with constipation). Lack of response to one agent does not preclude response to another, and low-dose combinations may work when side effects occur with higher doses of single agents. Oxybutynin has both antimuscarinic and musculotropic action; the initial dosage is 2.5 mg two to three times daily (5 mg once daily for extended release), followed by titration as needed up to 20 mg per day. Quick onset of action makes regular release useful when protection is needed at specific times. Extended-release oxybutynin (5 to 20 mg once daily) allows once-daily dosing and fewer side effects. Anticholinergic side effects can be limiting; constipation and compensatory fluid intake for xerostomia may exacerbate UI, and xerostomia predisposes a person to caries. The PVR should be monitored if UI worsens, as it may indicate retention that requires lowering the dose. Tolterodine (2 mg twice daily) has similar efficacy and possibly decreased xerostomia, especially with extended release (4 mg daily). One case report suggests that it may increase the international normalized ratio in persons on warfarin. The dose of tolterodine should not exceed 2 mg daily if the patient is taking medications that inhibit cytochrome P-450 3A4 (eg, erythromycin, ketoconazole).

Other agents including propantheline, dicyclomine, imipramine, hyoscyamine, calcium channel blockers, and nonsteroidal anti-inflammatory agents have scant efficacy data. Flavoxate is ineffective. Vasopressin was found to decrease nocturnal voids in a small randomized trial in healthy older persons, yet its expense and risks of congestive failure and hyponatremia argue against routine use.

In patients with DHIC on bladder-suppressant medications, careful monitoring for urinary retention is crucial. Double voiding (waiting and trying to void again before leaving the toilet), Valsalva’s maneuver, or Credé’s maneuver (pressure applied to the bladder) may improve bladder emptying. Constipation should be treated. If acceptable to the patient, retention may be induced with medication and the bladder emptied by intermittent clean catheterization several times daily.

Sacral nerve neuromodulation by an implanted S3 electrode may decrease severe refractory DO, but reimplantation is required in one third of patients. Augmentation cystoplasty surgery has high morbidity and is reserved for patients with profound DO (usually younger persons with poorly compliant bladders from neurologic disease).

Stress Incontinence
Pelvic muscle exercises (PME) strengthen the muscular components of urethral supports and are the cornerstone of noninvasive treatment for stress UI. PME, like strength training, employs a small number of isometric repetitions at maximal exertion. Unfortunately, there is much professional and lay misinformation about PME; persons who report failing PME trials may have used inadequate methods. PME requires motivated patients and careful instruction and monitoring by health professionals; poor adherence may occur even with close monitoring. PME instruction should focus on isolation of pelvic muscles; avoidance of buttock, abdomen, or thigh muscle contraction; moderate repetitions of the strongest contraction possible (3 sets of 8 to 10 contractions held for 6 to 8 seconds 3 to 4 times a week); and contraction for progressively longer times (up to 10 seconds, if possible). (See the Appendix, for an example of PME instructions for patients.) Biofeedback may help patients perform correct muscle contraction and monitor progress. The efficacy of other adjuncts—such as electrical or magnetic stimulation of pelvic muscle contractions, and progressively weighted cones retained in the vagina during ambulation—is less clear. Pessaries may benefit women with stress UI exacerbated by bladder or uterine prolapse. (See Gynecologic Diseases and Disorders.)

Systemic or topical estrogen may reduce stress and urge UI in some patients with atrophic vaginitis and urethritis. Several epidemiologic and intervention studies, however, have not found estrogen to be beneficial for stress UI. Topical agents (estrogen creams, vaginal tablets, or slow-release rings) may be preferable. α-Adrenergic agonists stimulate urethral smooth muscle contraction; however, no pure α-agonists are currently available. Estrogen increases α-receptor responsiveness and may potentiate α-adrenergic agonists. Imipramine’s α agonist and anticholinergic actions have been used in women with mixed stress and urge UI, but if the PVR increases, stress leakage may worsen, and efficacy data is scant.

Surgical correction offers the highest cure rates for stress UI at a cost of some increased morbidity. The surgical approach depends on the underlying defect and coexistent prolapse. For urethral hypermobility (genuine stress UI), bladder neck suspension procedures that repair urethra support defects are preferred (eg, transvaginal colposuspension). Complications include urinary retention and vaginal wall prolapse. The Marshall-Marchetti-Krantz abdominal procedure has greater morbidity. Anterior colporrhaphy and needle suspensions are less effective and not recommended. For intrinsic sphincter deficiency, sling procedures using autologous or synthetic material to support the urethra, and periurethral bulking injections with collagen or autologous fat are preferred. Increasingly, slings also are used for genuine stress UI (urethral hypermobility).

Treatment for men with postprostatectomy stress UI often is difficult. For milder cases PME and bulking injections can be helpful, whereas severe cases often require management with protective garments or catheters. Artificial sphincter replacement can be effective but has high reoperation rates (up to 40%), even with experienced surgeons.

**Overflow Incontinence**

Treatment of overflow incontinence depends on its cause. For outflow obstruction from benign prostatic hyperplasia, a range of medical and surgical alternatives are available (see Prostate Disease). Outlet obstruction should be considered in women with previous vaginal or urethral surgery; treatment by unilateral suture removal or urethropagation (remobilization of adhesions) can restore continence.

For detrusor underactivity, treatment is supportive. Drugs that impair detrusor contractility and increase urethral tone should be decreased or stopped, and constipation treated. Bethanechol chloride is ineffective except possibly for patients with overflow UI who must remain on anticholinergic agents (eg, antidepressant or antipsychotic medications). Intermittent clean catheterization can provide effective management; sterile intermittent catheterization is preferred for frailer patients and those in institutionalized settings. Bladder emptying may improve with Credé’s or Valsalva’s maneuvers during voiding, double voiding, or simply unhurried voiding.

**Catheters and Catheter Care**

Indwelling catheters cause significant morbidity, including polymicrobial bacteriuria (universal by 30 days), febrile episodes (1 per 100 patient days), nephrolithiasis, bladder stones, epididymitis, and chronic renal inflammation and pyelonephritis. External collection devices also cause bacteriuria, infection, penile cellulitis and necrosis, and urinary retention and hydronephrosis if the condom twists or its external band is too tight.
Indwelling catheters should be reserved for short-term decompression of acute retention: when retention cannot be managed surgically or medically, when wounds need to be kept clean of urine, when a terminally ill or severely impaired patient cannot tolerate garment changes, or when there is persistent patient preference for catheter management despite risks. The passage of the 1990 Omnibus Budget Reconciliation Act has resulted in more appropriate and decreased catheter use in long-term care but with an increased prevalence of UI.

Several general principles guide safe and effective catheter care. Bacteriuria and infection are reduced by closed drainage systems. Topical meatal antimicrobials, catheters with antimicrobial coating, collection bag disinfectants, and antimicrobial irrigation are not effective. Although antibiotics decrease bacteriuria and infection, routine use induces resistant organisms and secondary infections such as *Clostridium difficile* colitis. Bacteriuria is universal in catheterized patients and should not be treated unless there are clear symptoms. Routine cultures should not be done because of the changing flora and failure to predict infection. In symptomatic patients, cultures should be done after the old catheter is removed and a new catheter is placed. Institutionalized patients with catheters should be kept in separate rooms to decrease cross-infection.

With acute urinary retention, decompression should continue at least 7 days, followed by a voiding trial after catheter removal (never clamping). Prophylactic antibiotics are recommended only with short-term catheterization in high-risk patients (e.g., those with prosthetic heart valves). For men with chronic obstruction, intraurethral stents may be preferred to indwelling catheters.

Risk factors for catheter blockage include alkaline urine, female gender, poor mobility, calciuria, proteinuria, copious mucin, *Proteus* colonization, and preexistent bladder stones. Changing the catheter every 7 to 10 days may decrease blockage in such patients. In the absence of risk factors for blockage, catheters need not be changed routinely as long as monitoring is adequate. If patients cannot be monitored, changing catheters every 30 days is reasonable. Persistent leakage around the catheter can be caused by irritation by a large Foley balloon, catheter diameter that is too large, bacteriuria, constipation or impaction, improper catheter positioning, or catheter materials.

Clean intermittent catheterization is an alternative for willing patients with sufficient dexterity. Strict sterility is not necessary, although good handwashing and regular decontamination of the catheters is needed. Bacteriuria can be minimized by a frequency of catheterization that keeps bladder volume < 400 mL. Stiffer catheters are easier to insert.

Resources, including Web site addresses, of use to clinicians treating patients with UI are listed in the Appendix.

ANOTATED REFERENCES


  The entire issue of the journal is devoted to geriatric incontinence evaluation and treatment. It includes good review articles on age-related changes, diagnosis, pharmacology, behavioral and surgical treatments, nocturia, benign prostatic hyperplasia, vaginal and urethral atrophy, and effects on quality of life.


  These are good reviews of stress incontinence treatment. The systematic review of pelvic floor exercises plus adjuncts such as biofeedback and electrical stimulation is well done, providing a useful summary table of levels of evidence supporting behavioral treatments. The surgical review focuses on a situation common to many older women, recurrent stress urinary incontinence after an initial anti-incontinence surgery; it provides a good review of the range of surgical techniques.


The first study is a randomized, controlled trial comparing biofeedback-assisted behavioral treatment, oxybutynin, and placebo for urge or mixed urinary incontinence in 197 ambulatory, cognitively intact, community-dwelling women aged 55 and older. Behavioral treatment resulted in significantly greater incontinence reduction (80.7%) than with drug treatment (68.5%, *P* = .04), with a parallel difference in patient satisfaction. There was a significant placebo effect, with the control group experiencing a 39.4% decrease in urinary incontinence. In the follow-up study, the authors followed women who wished at the trial’s end to cross over to the other treatment. The 13% of those in the behavioral treatment group who crossed over to drug treatment had further improvement (from 57.5% to 88.5% decrease in incontinence), as did the 42% of those in the drug treatment group who crossed to behavioral treatment (from 72.7% to 84.3% decrease). Together, these studies demonstrate the efficacy of well-done behavioral therapy and the potential to improve outcomes with stepped combination therapy in patients who are not satisfied with initial treatment.


This is the consensus summary of the recommendations of the 1998 WHO International Consultation on Incontinence, including algorithms for the initial assessment and management of urinary incontinence in children, women, men, persons with neurologic disease, and frail elderly persons. The perspective is global, taking into consideration widely variable health care resources. The algorithms, designed for initial primary care management, are simple yet informative; specialized management algorithms are available at [http://www.thelancet.com](http://www.thelancet.com). Older persons with Alzheimer’s disease and urinary incontinence are included in the “neurogenic incontinence algorithm” but may be best assessed using the “frail older person” algorithm that includes assessment of reversible precipitants.

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