



Urinary Incontinence in the Elderly

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Urinary incontinence (UI), the involuntary loss of urine so severe as to have social and/or hygienic consequences for individuals and/or their caregivers, is a major clinical problem and a significant cause of disability and dependency. Urinary incontinence affects all age groups and is particularly common in the elderly.

Over the past decade, a considerable amount of research has increased our understanding of the pathophysiology and optimal treatment for this common geriatric condition. However, there is a persistent myth that UI is a normal consequence of aging. While normal aging is not a cause of UI, age-related changes in lower urinary tract function predispose the older person to UI in the face of additional anatomic or physiologic insults to the lower urinary tract or by systemic disturbances such as chronic illnesses common in the elderly.

Frail nursing home residents often have UI that can be significantly improved or cured. Persons with UI should be alerted to the importance of reporting their symptoms to a health care professional and of asserting their right to proper assessment and treatment.

Despite the increased knowledge about clinical forms, diagnostic tests, and treatments, opinions differ widely concerning the best approach to the specific forms of the disorder because of the lack of well-defined guidelines.

Because only about half of the people with incontinence in the community have consulted a physician about the problem, the true clinical extent and public health impact of UI are underestimated. The costs of incontinence have been estimated to be more than \$10 bil-

lion annually in the United States. In nursing homes alone, the costs of labor, laundry, and supplies necessary to manage incontinence and its complications are more than \$3 billion.

PREVALENCE AND INCIDENCE

The prevalence of UI in a population varies, however, depending on the definitions used, clinical severity, comorbidity, sex, and the age range of the population studied. This is reflected in the wide range of results from different epidemiologic studies of UI. Despite all of these epidemiologic limitations, a prevalence range between 15% and 30% for community-dwelling older persons is considered a fair estimate. Several studies show that within the adult age range, prevalence figures for any urine loss increase with increasing age.

The prevalence of severe incontinence, with episodes occurring weekly or more often is in the range of 5% to 8%, and the estimates for symptoms of stress and urge incontinence range from 3% to 20% and 5% to 20% respectively. Among adults more than 60 years of age, women usually have a likelihood of UI at least twice that of men. Interestingly, among adults less than 60 years of age, the sex difference appears even more pronounced. Among residents of nursing homes, the prevalence is generally above 40%, and in some facilities approaches 80%.

Little is known about the natural history of

KEY POINTS

- Frail nursing home residents often have urinary incontinence that can be improved or cured.
- There are four types of chronic incontinence: Detrusor overactivity (urge) incontinence, stress incontinence, overflow incontinence, and functional incontinence.
- Treatment is based on the type of incontinence, and may include muscle exercises, biofeedback, electric stimulation, behavioral treatments, various surgical interventions, and pharmacologic treatments, such as anticholinergic-antispasmodic agents and alpha-adrenergic antagonists.

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UI and its incidence. Currently, we do not know what proportion of cases of incontinence in community-residing adults take a chronic course and what proportion are transient. One-year incidence rates of 10% for older men and 20% for older women were observed in the MESA study.^{1,3}

URINARY CONTINENCE

Urinary continence is a severe test of the intactness of the nervous system. It is generally accepted that the bladder is controlled by centers in the pons, which determine whether it is in the storage or emptying mode. Although the brain plays a crucial role in the control of micturition, little is known about the structures involved. Three areas in the brain stem and diencephalon are specifically implicated in the control of micturition: the dorsolateral pontine tegmentum, the periaqueductal gray matter, and the preoptic area of the hypothalamus.

In the dorsolateral pons, the medial cell group is called the M-region and projects via long descending pathways to the intermediolateral cell columnae containing autonomic motor neurons of the detrusor muscle. The lateral cell group is called the L-region and sends fibers throughout the length of the spinal cord to the nucleus of Onuf innervating the pelvic floor, including the external urethral sphincter.

According to these findings, a concept is presented in which the ascending projection from the sacral cord, conveying information on bladder filling, terminates in the periaqueductal gray matter. In case the bladder is sufficiently distended that voiding is necessary, the periaqueductal gray matter stimulates the M-region, which results in micturition. The M-

region, however, also receives afferents from the preoptic area, which might be involved in the final decision to start micturition.⁴

AGE-RELATED CHANGES IN THE LOWER URINARY TRACT

Major organ systems decline with advancing age. These age-related declines are gradual, progressive, and linear, generally occurring independently of each other (Table 1). Age-related change has been hampered by the lack of a definition of normality in aging. It appears, however, that detrusor contractility, bladder capacity, and the ability to withhold voiding decline in both sexes; the maximal urethral closure pressure and length probably decline in women; detrusor overactivity increases in prevalence; and the post voiding residual (PVR) urine volume probably increases to 50 mL to 100 mL. The elderly tend to excrete most fluid intake at night because of changes in the circadian sleep-awake pattern of urine production and changes in the antidiuretic and atrial natriuretic hormones and renin aldosterone system, even in the absence of diseases such as venous insufficiency, heart failure, renal disease, and prostatic enlargement. None of the cited age-related changes causes incontinence, but all predispose to it.⁵

TYPES AND CAUSES OF URINARY INCONTINENCE

The causes of incontinence are multifactorial and may involve factors both within and outside the lower urinary tract. Urologic, gynecologic, neurologic, psychologic, environmental, and iatrogenic factors may all play a role. Several potentially reversible causes may contribute to transient as well as to chronic incontinence.

TABLE 1. Age-Related Changes Affecting Micturition

<i>Change</i>	<i>Predisposes to</i>
Detrusor overactivity (about 20% of healthy, continent persons)	Frequency, urgency, nocturia, urinary incontinence
Benign prostatic hyperplasia	Outlet obstruction with frequency, urgency, nocturia, urge or overflow UI
More urine output later in the day	Nocturia
Atrophic vaginitis and urethritis	Decreased urethral mucosal seal, irritation, urge and stress UI
Increased PVR	Frequency, nocturia, UI
Decreased ability to postpone voiding	Frequency, urgency, nocturia, UI
Decreased total bladder capacity	Frequency, urgency, nocturia, UI
Decreased detrusor contractility	Decreased flow rate, elevated PVR, hesitancy

TABLE 2. Common Causes of Transient Incontinence^a

Delirium or confusional state
Infection, urinary (symptomatic)
Atrophic urethritis or vaginitis
Pharmaceuticals
Sedatives or hypnotics, especially long-acting agents
Loop diuretics
Anticholinergic agents (antipsychotic agents, antidepressants, antihistamines, antiparkinsonian agents, antiarrhythmics, antispasmodics, opiates, and antidiarrheal agents)
Alpha-adrenoceptor agonists and antagonists
Calcium-channel-entry blockers
Vincristine
Psychologic disorder, especially depression
Endocrine disorder (hypercalcemia or hyperglycemia)
Restricted mobility
Stool impaction

TABLE 3. Medications That Can Potentially Affect Continence¹²

Type of Medication	Potential Effects on Continence
Diuretics	Polyuria, frequency, urgency
Anticholinergics	Urinary retention, overflow
Psychotropics	
Antidepressants	Anticholinergic actions, sedation
Antipsychotics	Anticholinergic actions, sedation, rigidity, immobility
Sedatives/Hypnotics	Sedation, delirium, immobility, muscle relaxation
Narcotic analgesics	Urinary retention, fecal impaction, sedation, delirium
Alpha-adrenergic blockers	Urethral relaxation
Alpha-adrenergic agonists	Urinary retention
Beta-adrenergic agonists	Urinary retention
Calcium channel blockers	Urinary retention
Alcohol	Polyuria, frequency, urgency, sedation, delirium, immobility

Many reversible or transient causes of UI can be identified, especially in persons who have incontinence *de novo* and those who have worsening severity of established incontinence. These transient causes of UI are from external processes that act on the urinary tract to precipitate incontinence. A simple mnemonic, DIAPPERS (Table 2), summarizes these causes.

The four basic types of chronic incontinence are Detrusor overactivity (urge) incontinence, stress incontinence, overflow incontinence, and functional incontinence.

Detrusor overactivity is the most common cause of UI in the elderly, occurring in 40% to 70% of those who present to the physician with complaints of incontinence. Patients with detrusor overactivity have early, forceful detrusor contractions, which occur well before the bladder is full. This creates its clinical hallmark "the abrupt sensation that urination is imminent, whether or not leakage ensues" and frequency. Patients with detrusor overactivity describe frequent losses of small to moderate volumes of urine. The PVR urine volume is typically normal (<51 mL). Detrusor overactivity can be found in conditions of defective central nervous system inhibition or increased afferent sensory stimulation from the bladder. Examples of disorders, which impair the ability of the brain to send inhibitory signals, include strokes, masses (tumor, aneurysm, hemorrhage), demyelinating disease (multiple sclerosis), Alzheimer's disease, and Parkinson's disease. Increased afferent stimulation from the bladder can result from lower urinary tract infections (cystitis), atrophic urethritis, fecal impaction, or uterine prolapse. Benign prostatic hyperplasia is a common

cause of detrusor overactivity in men. It can also produce symptoms of urinary outflow obstruction. Impaired detrusor contractility alone is an uncommon cause of UI, though it occurs in patients with diabetic neuropathy, spinal stenosis, and spinal cord injury. It has been diagnosed in conjunction with detrusor overactivity in almost one third of nursing home patients. Patients with detrusor hyperactivity with impaired contractility experience urge symptoms, but the PVR urine volume is high (>100 mL).

Stress incontinence is the most common type in early postmenopausal and younger elderly women. Although this type of incontinence can occur in men, it is usually limited to those who have had internal sphincter damage from various urologic procedures. The hallmark symptom is leakage of urine simultaneous with increases in intra-abdominal pressure caused by coughing, sneezing, laughing, bending, or exercising. In women, the etiology of urinary stress incontinence is usually pelvic relaxation resulting from multiple childbirths combined with the aging process. Drug-related causes of stress incontinence can include alpha-adrenergic antagonists.

Overflow incontinence is observed in 7% to 11% of incontinent elderly individuals and is characterized by a reduction in the force and caliber of the urinary stream, incomplete micturition, and the sensation of incomplete voiding. The two main sources of overflow incontinence are outlet obstruction and bladder contractile dysfunction, both of which lead to an increase in bladder volume. In the former condition, a physical blockage causes obstruction of urine flow, commonly caused by benign prostatic hypertrophy, urogenital cancers, severe genitourinary prolapses, and fecalomas. Dysfunction in bladder contractility can result from diabetic or alcoholic neuropathy, sacral spinal cord lesions, or the use of medications with anticholinergic properties, such as neuroleptics, narcotics, certain tricyclic antidepressants, and muscle relaxants.

Functional incontinence is a term used to describe incontinence that is predominantly related to chronic impairments of cognitive function and/or mobility that interfere with independent toileting skills. It should be a diagnosis of exclusion.⁶⁸

DIAGNOSTIC EVALUATION

The purposes of the diagnostic approach are (1) to determine the cause of the inconti-

nence; (2) to detect related urinary tract pathology; and (3) to evaluate comprehensively the patient with regard to mental and physical status, comorbidity, medications (Table 3), environment, and the available resources. The extent of the evaluation must be tailored to the individual and tempered by the realization that not all detected conditions can be cured, that simple interventions may be effective even in the absence of an exact diagnosis, and that for many elderly persons, diagnostic tests are themselves often interventions.

A focused history should include a detailed past and present medical, surgical, urologic, gynecologic, and neurologic history. A characterization of the type of incontinence (urge, stress, overflow), frequency, severity, duration, and pattern. Also, ask for associated lower urinary symptoms (straining, incomplete voiding, dysuria) precipitating factors (bowel habits, medications, fluid intake), and palliative measures (protective devices, surgical or medical treatments). The history should also include study of voiding habits (voiding/incontinence diary), and assessment of mobility, dexterity, mental status, living conditions, and social environment.

The physical examination should focus on checking for signs of neurologic disorders (dementia, delirium, stroke, Parkinson's disease, cord compression, peripheral neuropathy) and identification of general medical illnesses (heart failure, orthostatic hypotension, peripheral edema, abdominal masses). It should also include pelvic examination (atrophic urethritis, pelvic mass, laxity of pelvic floor, and prolapse) and rectal examination (symmetry of gluteal creases, perineal sensation, tone and voluntary control of the anal sphincter, fecal impaction, masses, and prostatic enlargement [though its size correlates poorly with obstruction]). A provocative stress test and measurement of PVR urine volume should be included.

In addition to the history and physical examination, other measurements that should be obtained in all patients are urinalysis (bacteriuria, pyuria, hematuria, glycosuria and proteinuria), serum creatinine or blood urea nitrogen levels, blood glucose level, and urine cytology.

Based on the findings from this initial basic evaluation, a decision for treatment or more specialized evaluation is made. Specialized studies currently available include uroflowmetry,

cystometry, cystourethrography, urethral pressure profilometry, electrophysiologic sphincter testing, videourodynamics, electromyography, ultrasonography, and ambulatory urodynamics. These numerous noninvasive and invasive tests must be used selectively. Some elderly patients may not be candidates for sophisticated studies due to inability to cooperate or a poor prognosis for correction.^{9,11}

MANAGEMENT

Detrusor Overactivity (Urge Incontinence)

Behavioral therapy is the initial step in treating urge incontinence. Bladder retraining is effective for those individuals who are independent of caregiver support and motivated to participate actively in treatment. Patients can expect 50% improvement in their incontinence. The goals are to change dysfunctional habit patterns, to improve the ability to suppress urgency, and to gradually increase bladder capacity and extend the voiding interval. Initial voiding intervals are gradually increased as the patient learns how to suppress the urge long enough to walk slowly to the bathroom and void in a controlled fashion. Pelvic floor contractions are used to inhibit the urge and postpone voiding. Pelvic muscle exercises, biofeedback techniques, and electric stimulation help strengthen the pelvic floor muscles to augment urethral closure and reflexively inhibit bladder contractions.

Routine toileting and prompted voiding are techniques used primarily in institutional settings for cognitively impaired patients with urge incontinence. In routine toileting, the patient is brought to the toilet every 2 hours with the hope of avoiding an incontinent episode, and positive verbal reinforcement is used for maintaining continence and using the toilet.

When behavioral treatments alone are unsatisfactory, anticholinergic-antispasmodic agents are the preferred pharmacologic option in the older individual because of their efficacy and side-effect profiles. Tolterodine (1 mg to 2 mg twice daily) and oxybutynin (2.5 mg to 5 mg every night and up to 3 times daily) remain the drugs of choice and can reduce urinary incontinence by 15% to 60%. These medications are associated with anticholinergic side effects, including dry mouth, constipation, bladder retention, and confusion. If drugs fail or are not tolerated, transcutaneous electrical nerve stimulation may be an alternative treatment for detrusor overactivity.

In patients with impaired detrusor contractility, the treatment goals are to reduce the PVR urine volume and prevent urine reflux to the kidneys and renal impairment. The first step is to decompress the bladder with an indwelling catheter and to correct any aggravating factors, such as medications and fecal impaction. If the bladder remains completely acontractile after bladder decompression, intermittent catheterization should be started or an indwelling catheter should be used.

Stress Incontinence

Pelvic floor rehabilitation is the first step of treatment in older individuals. Pelvic muscle exercises, also known as Kegel exercises, can decrease incontinence substantially in many motivated and cognitively intact older women. The contractions strengthen the muscles, exert closure of the urethra, and reflexively inhibit urgency symptoms. Biofeedback is extremely helpful for patients who have difficulty isolating the pelvic muscles and inhibiting involvement of abdominal, gluteal, and adductor muscles.

Pharmacologic therapies for stress incontinence are designed to increase bladder outlet resistance when pelvic floor muscle rehabilitation therapies alone are not effective. Estrogen reverses the urogenital atrophy and urethritis associated with postmenopausal aging and can be administered either orally (conjugated estrogen, 0.625 mg/day) or vaginally (estrogen cream, 0.3 mg with applicator twice weekly). Progestin (medroxyprogesterone, 2.5 mg/day) should be administered to those women with an intact uterus. Alpha-adrenergic agonists should rarely be used in older women because of the effect on hypertension, cardiac arrhythmias, and angina.

Surgery is an effective treatment for pure stress incontinence associated with urethrocele. A variety of surgical techniques for the transvaginal or transabdominal suspension of the bladder neck yield a success rate between 80% and 95% in appropriately selected patients. Urethral sling procedures pass a ribbon of fascia or artificial material beneath the urethra. The sling, fixed to the anterior body wall, serves to elevate and compress the urethra, restoring continence in 80% of patients. Frail nursing home residents may benefit from bladder neck suspension surgery and repair of pelvic prolapse when it is appropriate. Periurethral injection of substrates that compress, support, or narrow the bladder neck for

the treatment of stress incontinence can be done in carefully selected patients. The best candidates are those with intrinsic sphincter dysfunction and elderly women who are at high operative risk.

Overflow Incontinence

Treatment for overflow incontinence depends on the cause of the urinary retention. If there is anatomic obstruction, a surgical procedure is necessary. New, less invasive operations are now being done for benign prostatic enlargement, and laser prostatectomy may be an increasingly common option for older men. Transurethral prostatectomy can result in a high cure rate for patients with properly functioning bladders. Men with moderate benign prostate hypertrophy assigned randomly to have either transurethral resection of the prostate or watchful waiting had equivalent occurrence of persistent UI. In these patients, full evaluation, including urodynamic testing before surgery, is essential to rule out coexisting causes of incontinence.

Transurethral resection of the prostate can result in UI due to detrusor dysfunction or urethral insufficiency caused by sphincteric injury, bladder dysfunction, or bladder outlet obstruction. For many patients with either stress or urge postprostatectomy incontinence, treatment with Kegel exercises can reduce symptoms. When conservative therapy has failed, surgery may be appropriate. Men are candidates for periurethral bulking injections, though these techniques are better studied in women than in men. Periurethral injections seem less useful for severe postprostatectomy incontinence, but artificial sphincter implantation allows patients with severe or continual urinary leakage to gain continence with a cure rate of 66%.

An alpha-adrenergic antagonist (terazosin or tamsulosin) is effective in minimizing symptoms of prostatism. Finasteride decreases trophic androgenic stimulation of the prostate and may delay the need for surgery in selected men with a markedly enlarged prostate. Neither terazosin nor finasteride, however, should be the treatment of choice when there is urinary retention.^{12,16}

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A Crabbit Old Woman?

What do you see nurses
 what do you see?
 Are you thinking
 when you are looking at me
 A crabbit old woman,
 not very wise,
 Uncertain of habit
 with far away eyes,
 Who dribbles her food
 and makes no reply,
 When you say in a loud voice
 "I do wish you'd try"
 Who seems not to notice
 the things that you do,
 And forever is losing
 a stocking or shoe,
 Who unresisting or not
 lets you do as you will
 With bathing and feeding
 the long day to fill,
 Is that what you're thinking,
 is that what you see?
 Then open your eyes nurse,
 you are not looking at me,
 I'll tell you who I am
 as I sit here so still:
 As I use at your bidding,
 as I eat at your will,
 I'm a small child of ten
 with a Father and Mother,
 Brother and sisters who
 love one another,

A young girl of sixteen,
 with wings on her feet,
 Dreaming that soon now
 a lover she'll meet:
 A bride soon at twenty,
 my heart gives a leap,
 Remembering the vows
 that I promised to keep:
 At twenty five now
 I have young of my own
 Who need me to build
 a secure happy home,
 A woman of thirty,
 my young now grow fast,
 Bound to each other
 with ties that should last;
 At forty my young sons
 now grown and all gone
 But my man stays beside me
 to see I don't mourn,
 At fifty once more
 babies play around my knee,
 Again we know children
 my loved one and me.
 Dark days are upon me,
 my husband is dead,
 I look at the future
 I shudder with dread,
 For my young are all busy
 rearing young of their own,
 And I think of the years
 and the love I have known,

I'm an old woman now
 and nature is cruel,
 'Tis her jest to make
 old age look like a fool.
 The body it crumbles,
 grace and vigour depart,
 There now is a stone
 where once I had a heart:
 But inside this old carcase
 a young girl still swells,
 I remember the joys,
 I remember the pain,
 And I'm loving and living
 life over again,
 I think of the years
 all too few—gone too fast,
 And accept the stark fact
 that nothing can last,
 So open your eyes nurses,
 open and see,
 Not a crabbit old woman
 Look slower—see ME.

Kate

(This poem was found in the locker of a patient who died in a long-term-care hospital ward in England. The patient was thought to have a dementing illness.)

TAB 2

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