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Diagnosis and Management of Urinary Incontinence for the Primary Care Physician

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Introduction

Urinary incontinence, defined as the involuntary loss of urine that is objectively demonstrable and a social or hygienic problem, is a condition that affects approximately 13 million Americans in community and institutional settings. Despite its prevalence and an estimated annual direct cost of more than $15 billion, most affected individuals do not seek help for incontinence primarily because of embarrassment or because they are not aware that help is available.[1] Urinary incontinence has a profound psychosocial effect on individuals, their families, and caregivers. It can result in loss of self esteem, social isolation, sexual dysfunction, and a decrease in ability to maintain an independent lifestyle. When patients do seek help, many clinicians are hesitant or inexperienced to discuss, diagnose, or treat the problem. It is expected that urinary incontinence will continue to be a significant healthcare problem in the elderly and institutionalized populations and will increase as the population continues to age and there is growing awareness and media attention given to this condition. The following paper will review the pathophysiology, diagnosis, and office management of urinary incontinence, with emphasis given on the role of the primary care provider.

Epidemiology

Although demographic surveys report that urinary incontinence affects 10-35% of adults and at least half of the 1.5 million nursing home residents in the United States, this number is most likely underweighted, as many patients who suffer from this condition are reluctant to report the problem to their healthcare provider due to embarrassment and social stigma. Although the prevalence of urinary incontinence increases with age, it should not be considered a normal part of the aging process. Among the population between 15 and 64 years of age, the prevalence of urinary incontinence in men ranges between 1.5-5.0%, and in women from 10-30%.[2] Incontinence is 2-3

times more common among women than men, and is primarily related to gender-specific risk factors, such as childbirth and menopause, that adversely affect pelvic support structures and result in stress urinary incontinence. Although urinary incontinence is commonly regarded as a condition affecting older, multiparous women, it is also common in young, nulliparous women and is particularly associated with strenuous physical exercise.

In the elderly population, urinary incontinence can affect 15-30% of women older than the age of 60 and more than 50% of nursing home residents.[3] It is one of the major causes of institutionalization of the elderly in this country despite the fact that one-third of all cases may be due to transient factors that may be readily treatable or undergo spontaneous remission. Appropriate diagnosis and management of urinary incontinence in the elderly by the primary care provider can have a profound effect on quality of life and may prevent the need for subsequent nursing home admission.

The economic effect of urinary incontinence has been scarcely reported due to the inability to obtain reliable prevalence, risk factor, and cost data, and because of wide diversification of treatment modalities. Recent data, estimated at more than $15 billion annually, does not consider indirect costs, such as loss of productivity due to morbidity/mortality and time costs of unpaid caregivers treating and caring for the incontinent patient. As public awareness of this condition continues to increase, it is expected that the financial costs of treating this disorder will also proportionately rise. Unfortunately, the majority of these expenditures have been directed toward control measures rather than cure options, resulting in minimal improvement. Of the $15 billion spent annually on this disorder, only 1% of this amount was spent on the diagnosis and permanent treatment of this disorder, while 60% was spent on palliative measures.[4]

Due to the increased incidence of incontinence, studies have shown that an effective prevention program would result in a reduction of approximately 50,000 annual cases.[5] Reversible conditions or risk factors associated with incontinence are presented in Table 1. Although no controlled clinical trial data exist to support the fact that risk factor interventions would result in significant reduction of incontinence incidents, severity, or prevalences, these programs are easy to institute and may result in other medically related benefits. These include weight loss and smoking cessation programs, control of hypertension and diabetes, maintenance on estrogen replacement therapy and pelvic muscle exercise programs, and alteration or adjustment of current medications. Patient education and routine follow-up are the cornerstones for an effective risk reduction program with regard to urinary incontinence.

Anatomy and Physiology of the Normal Continence Mechanism

The exact mechanism of continence in the healthy adult is incompletely understood. Current concepts emphasize a complex interaction between the bladder and urethra dependent on intact enervation and anatomic support of the bladder neck, especially in females. The bladder is a muscular organ with two primary functions—bladder storage at rest and bladder

Table 1. Reversible Causes and Risk Factors for Urinary Incontinence

- Immobility/chronic degenerative disease
- Impaired cognition
- Medications
- Morbid obesity
- Diuretics
- Smoking
- Fecal impaction
- Delirium
- Low fluid intake
- Environmental barriers
- High-impact physical activities
- Diabetes
- Stroke
- Estrogen deprivation
- Pelvic muscle weakness
- Childhood nocturnal enuresis
- Race
- Pregnancy/vaginal delivery/episiotomy

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emptying during contraction. Both phases are regulated by the autonomic nervous system, with sympathetic enervation from T10 to L2 coming via the hypogastric nerve and parasympathetic enervation coming from S2 to S3 via the pelvic plexus. This system can be modulated by somatic nerves that innervate the pelvic floor and external urethral sphincter.

Disorders that affect bladder storage (filling phase) cause urinary incontinence, while disorders of emptying (voiding phase) cause voiding dysfunction and urinary retention. In some patients, especially women, abnormalities during filling and voiding may coexist. As urinary incontinence is primarily related to the storage phase, a detailed description of the physiology of normal micturition (voiding phase) is beyond the scope of this paper.

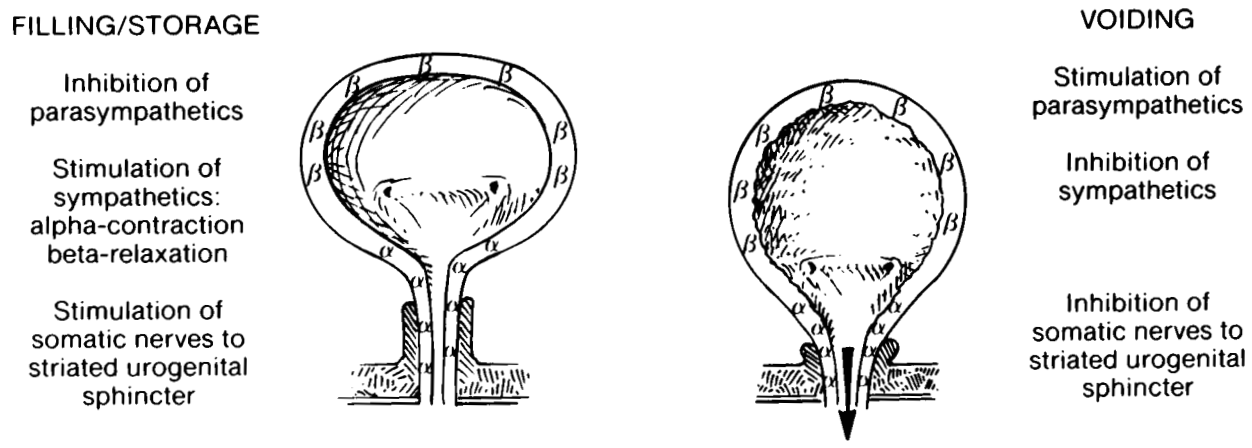
The mechanism of continence is complex. At rest, the bladder is a muscular reservoir that holds increasing volumes of urine while maintaining a low, resting pressure (accommodation) due to compliance of the bladder wall. Simultaneous sympathetic stimulation of beta adrenergic receptors within the bladder wall and inhibition of parasympathetic activity causes detrusor relaxation. Continence is maintained by a high resting pressure in the urethra at the urethrovesical junction (bladder neck) due to the musculature of the internal and external urethral sphincter, mucosal coaptation of the urethral lumen, and sympathetic stimulation of alpha adrenergic receptors in the urethra. (*See Figure 1.*)

During increases in intra-abdominal pressure, the pressure gradient between the bladder and urethra is maintained by equal transmission of pressure to the bladder and proximal urethra to ensure continence. In the female, the external urethral sphincter and levator ani muscle complex serve as a secondary continence mechanism by a reflex contraction, with resulting bladder neck closure during increased abdominal pressure. In addition, the vagina, slung like a hammock below the urethra by its lateral fascial attachments, functions as a backboard against which the bladder neck is compressed during valsalva maneuvers. During micturition, a coordinated change in the pressure relationship between the bladder (detrusor muscle) and urethra occurs. Voiding is initiated by voluntary relaxation of the pelvic musculature and urethral sphincter, followed by a detrusor contraction mediated by parasympathetic cholinergic receptors in the bladder. With an increase in bladder pressure and a decrease in urethral pressure, the pressure gradient favors micturition and bladder emptying occurs.

Urinary incontinence may occur due to abnormal increases in bladder pressure or decreases in urethral pressure that alter the low bladder pressure-high urethral pressure relationship that maintains continence at rest. Abnormal increases in bladder pressure are primarily due to spontaneous detrusor contractions (detrusor instability) with a reflex relaxation of the urethral sphincter that results in urinary leakage. Increased bladder pressure may also be due to overfilling of the bladder beyond its capacity and compliance resulting in overflow incontinence. Abnormal decreases in urethral pressure may be static or dynamic. A decrease in resting urethral tone can be caused by injury to the sphincter enervation or musculature (intrinsic sphincter deficiency). Abnormal bladder neck function in dynamic states of increased abdominal pressure or valsalva is seen in female patients with genuine stress incontinence. With relaxation of the pelvic floor muculature and fascial supports, the proximal urethra exhibits rotational descent outside the zone of abdominal pressure transmission and no longer has a fixed backboard for mechanical compression. As a result, increases in intra-abdominal pressure cause only elevated intravesical pressure, which overcomes the resting intraurethral pressure, resulting in loss of urine.

An adequate understanding of the normal continence mechanism as well as specific abnormalities resulting in urinary

Figure 1. Physiologic Basis of Storage and Voiding



Physiologic basis of storage and voiding based on characteristic distribution of sympathetic and parasympathetic receptors. Reprinted with permission from Mosby Year-Book Inc. from Walters MD and Karram MM. *Clinical Urogynecology* 1993;1-19.

incontinence is critical in determining the underlying etiology and formulating an effective treatment plan. Diagnostic evaluation is based on understanding normal bladder function at rest and assessing the lower urinary tract within these parameters.

Classification of Urinary Incontinence

Urinary incontinence is a symptom, a sign, and a condition. The International Continence Society has created a classification of urinary incontinence.[6] Accurate diagnosis of the type of incontinence will determine the appropriate treatment plan.

Genuine stress incontinence is the involuntary loss of urine that occurs when, in the absence of a detrusor contraction, the intravesical pressure exceeds the maximum urethral pressure. It is most common in women who typically complain of urinary leakage with cough, exercise, laughing, valsalva maneuver, and other activities that increase intra-abdominal pressure. Patients with severe stress incontinence may report constant leakage with minimal exertion. Genuine stress incontinence is due to urethral hypermobility or intrinsic sphincter deficiency. Urethral hypermobility, the most common cause of genuine stress incontinence, occurs when there is loss of the anatomic support of the bladder neck, allowing the proximal urethra to be displaced outside the abdominal pressure zone during straining. McGuire and colleagues have classified subtypes I, IIA, and IIB, depending on the amount of descent of the bladder base at rest and with straining.[7] This damage to the bladder neck supports may be the result of pregnancy and vaginal delivery, tissue atrophy that results from advancing age and estrogen withdrawal, or continuous stressors, such as obesity and chronic coughing. With intrinsic sphincter deficiency, the urethra exhibits decreased resting tone and no longer functions as a sphincter. Even at rest, it loses the ability to maintain a normal continence mechanism. More common in women, it is often associated with a history of pelvic radiation, previous anti-incontinence surgery, advancing age, and deenervation injuries, resulting in overt disruption of the sphincteric musculature. These patients most commonly demonstrate a fixed, rigid urethral tube, with a nonfunctioning sphincter (lead pipe urethra or Type III incontinence). In men, it is most often associated with urethral damage that occurs following transurethral resection of the prostate.

Potential stress incontinence can occur in women with advanced pelvic prolapse. These patients initially have loss of bladder neck support and resulting incontinence. Continued prolapse of the anterior vaginal wall results in kinking of the urethra with improvement of incontinent episodes but incomplete bladder emptying. Subsequent reduction of the prolapse with a pessary or surgery without concurrent bladder neck support will result in reestablishment of the incontinence symptoms.

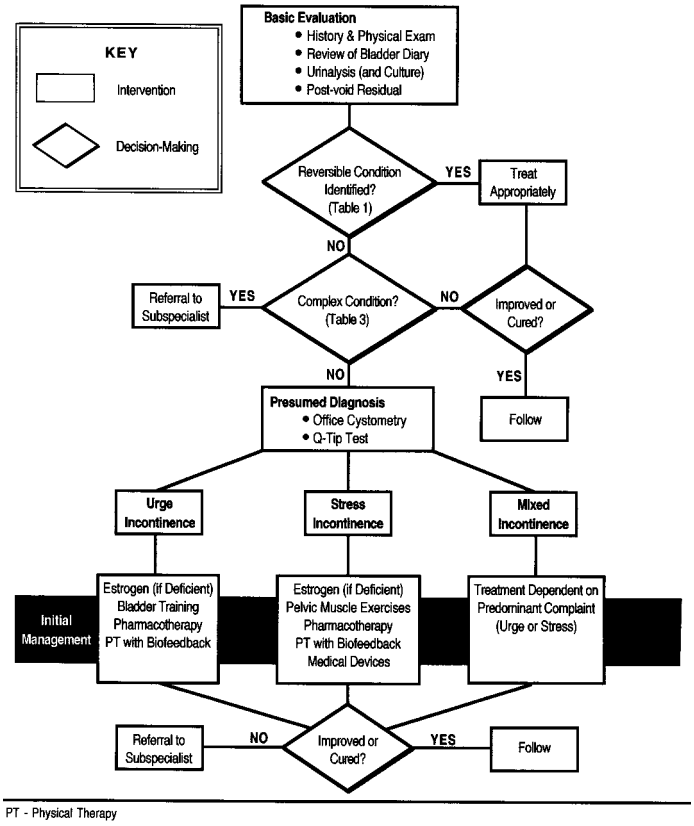
Urge incontinence is defined as the involuntary loss of urine associated with a sudden and strong desire to void (urgency). Normal micturition is under voluntary control. Spontaneous uninhibited detrusor overactivity can result in detrusor contractions with reflex urethral relaxation and urinary incontinence. Overactive detrusor function in the absence of a known neurologic abnormality is called detrusor instability; overactivity due to its disturbance of the nervous control mechanisms is termed detrusor hyperreflexia. Patients with this condition complain of an inability to control voiding and experience sudden urgency to void, which is sometimes unsuppressible. These patients commonly report coexisting urinary frequency (> 7 times/day), nocturia (> 1 time/night), enuresis, and, occasionally, pelvic pain. Although detrusor instability is most often iatrogenic, secondary causes include urinary tract infection, anti-incontinence surgery, bladder stones or foreign bodies, and bladder cancer. Suburethral diverticulum, an outpouching of the urethral mucosa, can also occasionally present with irritative bladder symptoms, such as urgency and frequency.

Detrusor hyperreflexia with impaired contractility (DHIC) is a rare disorder that may occur in some patients—especially the elderly or patients with neurologic lesions. This paradoxical condition is characterized by detrusor overactivity in combination with decreased detrusor contractility. These patients present with urge incontinence in combination with urinary retention. Diagnosis is difficult, as it can mimic other types of incontinence, and usually requires complex multichannel urodynamic testing. Mixed incontinence, a condition characterized by coexisting stress incontinence and urge incontinence, is one of the most common types of incontinence in patients presenting to their primary care physician. It typically results from compensatory responses that the stress incontinent patient self initiates. Once the initial symptom of stress incontinence is noted, the patient begins to urinate frequently to maintain a low residual of urine in the bladder and to minimize the risk of further stress-related incontinent episodes. As the condition worsens, the degree of urinary frequency increases and the bladder accommodates to a lower capacity. When the bladder distends beyond its now reduced capacity, the patient experiences sensory urgency or detrusor instability with or without associated urinary leakage and irritative symptoms, such as frequency, urgency, and nocturia. Often, however, one symptom, stress, or urge is more bothersome to the patient than the other, and therapeutic intervention is based on the predominant symptomatology.

Overflow incontinence is the uncontrollable loss of urine associated with overdistention of the bladder. This is usually caused by an underactive or acontractile detrusor or bladder outlet/urethral obstruction leading to overdistention and overflow. Failure of the bladder to empty adequately with large post-void residuals may by idiopathic, but it is usually found in conjunction with drugs, diabetic neuropathy, spinal cord injury, or radical pelvic surgery, which causes de-enervation of the detrusor muscle. In men, overflow incontinence is associated with obstruction due to prostatic hypertrophy or urethral stricture. In women, it can be associated with advance pelvic prolapse, which causes bladder outlet obstruction due to kinking of the bladder neck. Increased bladder capacities with a large post-void residual urine volume will clarify the diagnosis.

Other types of incontinence are less common but should be considered in the appropriate clinical context. Functional incontinence refers to urine loss that occurs due to factors outside the urinary tract, such as chronic impairment of physical or cognitive functioning. This type of incontinence is typically found in the elderly immobile nursing home resident. Reflex incontinence is the loss of urine due to detrusor hyperreflexia, involuntary urethral relaxation, or both in the absence of the sensation usually associated with the desire to void. This condition is most commonly seen in patients with neuropathic

Figure 2. Diagnosis/Treatment Algorithm for Urinary Incontinence



Diagnosis/treatment algorithm for urinary incontinence for the primary care provider in the office setting

bladder or urethral dysfunction due to neurologic injury. Transient and reversible causes for incontinence are often identified, particularly in elderly patients. A useful mnemonic for common reversible causes of incontinence is DIAPPERS—Delirium, Infection, Atrophic urethritis, Pharmaceuticals, Psychological factors, Endocrine disorders, Restricted mobility, and Stool impaction. Often, treatment of the inciting factor will result in immediate cure of urinary leakage. Extraurethral causes of incontinence should be included in the differential diagnosis. This is particularly relevant for women with a history of pelvic surgery, radiation, or obstetric delivery. Etiologic entities include urinary fistulas (ureterovaginal, vesicovaginal, or urethrovaginal), ectopic ureter, and urethral diverticulum. A detailed pelvic exam is required for diagnosis.

Evaluation of Urinary Incontinence

The evaluation of patients with urinary incontinence is directed toward: 1) clarifying a patient's symptoms; 2) objectively documenting loss of urine; 3) determining the etiology of the incontinence; and 4) identifying patients who require further consultation and complex testing. The large majority of patients can be appropriately diagnosed and managed in the primary care setting, and few will require outside referrals unless they have a complex condition or desire surgical correction. A step-by-step diagnostic algorithm that includes a history, physical examination, and appropriate office testing is useful in the evaluation of the incontinent patient. (*See Figure 2.*)

Accurate diagnosis of urinary incontinence begins with a careful review of the patient's history. The history should focus on the duration, characteristics, and severity of the incontinence, with particular attention to precipitating factors and reversible causes. Cystitis, foreign body, or tumor should be suspected in patients with acute onset of symptoms while chronic etiologies, such as urethral hypermobility, intrinsic sphicter deficiency, and idiopathic detrusor instability, should be considered for long-standing complaints. Concurrent lower urinary tract symptoms (i.e., dysuria, urgency, pelvic pain, dyspareunia) as well as symptoms related to the gastrointestinal tract (i.e., constipation, fecal incontinence) and genital tract (i.e., pelvic prolapse, abnormal vaginal discharge) should also be discussed. Objective assessment of the patient's incontinence using a bladder diary is recommended. We routinely ask patients to keep a two-day record of fluid intake, voids, and incontinent episodes with precipitating events prior to their first visit. This is subsequently reviewed with the patient in order to assess if the patient will benefit from behavioral or dietary modification, such as increased fluid intake, change in voiding pattern, or dietary restriction. A detailed medical and surgical history should be obtained to rule out diabetes, thyroid disease, spinal cord injury, cerebral vascular accidents, urethral sphincter damage, or fistula conditions. As incontinence increases with advanced age, a detailed drug history is particularly important as many of these elderly patients are on multiple medications. A variety of medications can induce urinary incontinence directly by acting on the bladder and/or urethra or indirectly by inducing cough or pelvic muscle relaxation mechanisms. Pharmacotherapy may induce any of the subtypes of urinary incontinence previously discussed. (*See Table 2.*) In women, estrogen status should be determined as hypoestrogenism can contribute to recurrent cystitis, detrusor instability, and stress incontinence. Patients should also be questioned about a history of recurrent urinary tract infection, kidney stones, bladder pain, or hematuria.[8]

Following a general physical exam and mental status assessment, clinical evaluation of the lower urinary tract should begin with a detailed neurologic exam of the perineum and lower extremities. Normal sensation in the perineal dermatomes and the back of the leg confirms intact sensory enervation of the lower urinary tract. Sacral reflex activity is tested via two reflexes. In the anal reflex, stroking the skin adjacent to the anus causes reflex contraction of the external anal sphincter while the sacral (bulbocavernosus) reflex involves contraction of the bulbocavernosus and ischiocavernosus muscles in response to tapping or squeezing of the clitoris. Pelvic floor muscle tone can be assessed by voluntary contraction of the anal sphincter and vagina during a bimanual exam.

In women, a pelvic exam should be performed to assess the external genitalia, perineal sensation, presence of pelvic organ prolapse (cystocele, enterocele, rectocele, uterine prolapse), estrogen status, and pelvic muscle strength. During inspection, particular attention should be given to the assessement and grading of pelvic organ prolapse to rule out urethral hypermobility and potential stress incontinence. A bimanual exam with

Table 2. Medications Causing Incontinence

ACE Inhibitors
- Enalapril

Anti-Parkinsonism Agents
- Benzotropine
- Trihexyphenidyl

Benzodiazepines
- Valium

Bethanocol

Cisapride

Diuretics
- Furosemide
- Hydrochlorothiazide

Diospyramide

Alcohol

Calcium-Channel Blockers
- Verapamil

Anticholinergics
- Hyoscyamine
- Oxybutinin

Antihypertensives
- Prazosin
- Terazosin
- Alpha-methyl dopa
- Reserpine

Beta-blockers
- Pindolol

Neuroleptics
- Thioridazine
- Chloropromazine
- Haloperidol
- Clozapine

Over-the-counter Cough Preparations

rectovaginal exam should be done to rule out pelvic masses compressing the bladder. In men, a rectal exam should be performed to test for perineal sensation or rectal mass and to evaluate the consistency and size of the prostate.

The patient should be examined with a full bladder so that observation of urine loss can be performed by having the individual cough vigorously, either in the standing or supine position. If instantaneous leakage occurs with cough, stress urinary incontinence is likely while urge incontinence (detrusor instability) should be considered with delayed or sustained leakage. In women, urethral hypermobility due to loss of bladder neck support can most easily be assessed using a simple cotton swab test. A sterile, lubricated cotton swab is inserted transurethrally into the bladder and then withdrawn slowly until definite resistance is felt, indicating that the cotton swab is at the bladder neck. The resting angle of the cotton swab in relation to the horizontal is measured, and the patient is then asked to cough or perform a valsalva maneuver; the maximum straining angle from the horizontal is then measured. Although no standardized data is available to differentiate abnormal from normal measurements, most clinicians have adopted a 30° deflection as a cutoff for urethral hypermobility.

Initial diagnostic testing should include a post-void residual to rule out overflow incontinence and incomplete bladder emptying, and a urinalysis and/or urine culture to rule out urinary tract infection. After a normal void, a post-void residual urine volume is determined using a catheterization or bladder scan. Although values for normal bladder emptying may vary with age, a post-void residual should be less than 25% of the total bladder volume and below 100 cc. Patients with high post-void residual measurement may experience overflow incontinence. A catheterized sample of urine should be obtained for urinalysis or urine culture. We have found that clean catch urine specimens are routinely contaminated and do not provide accurate data with regard to urinary tract infection. Cystitis is the leading cause of acute urinary incontinence in younger women. Based on initial evaluation and testing, a preliminary diagnosis can be made. Further office testing, such as simple cystometry, can be performed at the time of the initial exam or be reserved for subsequent visits. We find that it is an inexpensive test that is easy to perform and provides important initial data; we recommend it be done at the initial evaluation. In addition, it allows simultaneous measurement of a post-void residual and collection of a catheterized urine specimen.

Cystometry is used to measure the pressure volume relationship of the bladder as it distends and contracts and determine abnormalities of the bladder with respect to detrusor activity, sensation, capacity, and compliance. Complex cystometry (multichannel urodynamics) uses specialized equipment with pressure catheters to record abdominal, bladder, and urethral pressures and determines specific detrusor activity through subtracted calculations. (*See Figure 3.*) In contrast, simple office cystometry can readily be performed in the office and requires a stopwatch, red rubber catheter, 50 mL syringe, and sterile water or saline. The patient should be initially evaluated with a full bladder. The patient is allowed to void normally in a private setting, and the time to void and amount of urine void are recorded. (This process is called uroflow.) The patient then returns to the examination room where a transurethral catheter is inserted into the bladder lumen to check a post-void residual and to obtain a sterile urine specimen for urinalysis or culture. With the transurethral catheter in place, a 50 mL catheter tip syringe with its bulb removed is attached to the catheter and held approximately 15 cm above the pubic symphysis. With the patient in the sitting or standing position, the bladder is filled by pouring sterile water or saline into the syringe at a medium fill rate, attempting to keep the water level in the syringe constant. (*See Figure 4.*) During bladder filling, the patient is asked to report first bladder sensation, initial urge to void, and maximum bladder capacity. Normal values are 100-200 cc, 200-400 cc, and 400-600 cc, respectively. Decreased bladder capacity is suggestive of urgency-frequency syndromes and urge incontinence.

The water level is closely monitored during filling, as a rise with or without associated urgency or urinary leakage may be indicative of an uninhibited bladder contraction, suggesting detrusor instability. Unfortunately, rises in intravesical pressure may result from a detrusor contraction or valsalva maneuver and, therefore, are not diagnostic of detrusor overactivity. It may be helpful to ask the patient to inspire during a noted rise in intravesical pressure, as women can rarely increase their intra-abdominal pressure during inspiration. Once the bladder is filled to maximum capacity, the transurethral catheter is removed and the patient is examined in the supine or lithotomy

position and standing position. The patient is asked to perform provocative maneuvers, such as coughing, heel jumping, and valsalva. Urethral hypermobility and urinary leakage are evaluated. Loss of small amounts of urine simultaneous with cough suggests a diagnosis of stress incontinence. Prolonged loss of urine leaking 5-10 seconds after cough or no urine loss with provocation indicates that other causes of incontinence, especially detrusor instability, may be present.[9] Patients with reduced bladder capacity with co-existing urge-related complaints most likely have underlying detrusor instability and should be treated accordingly.

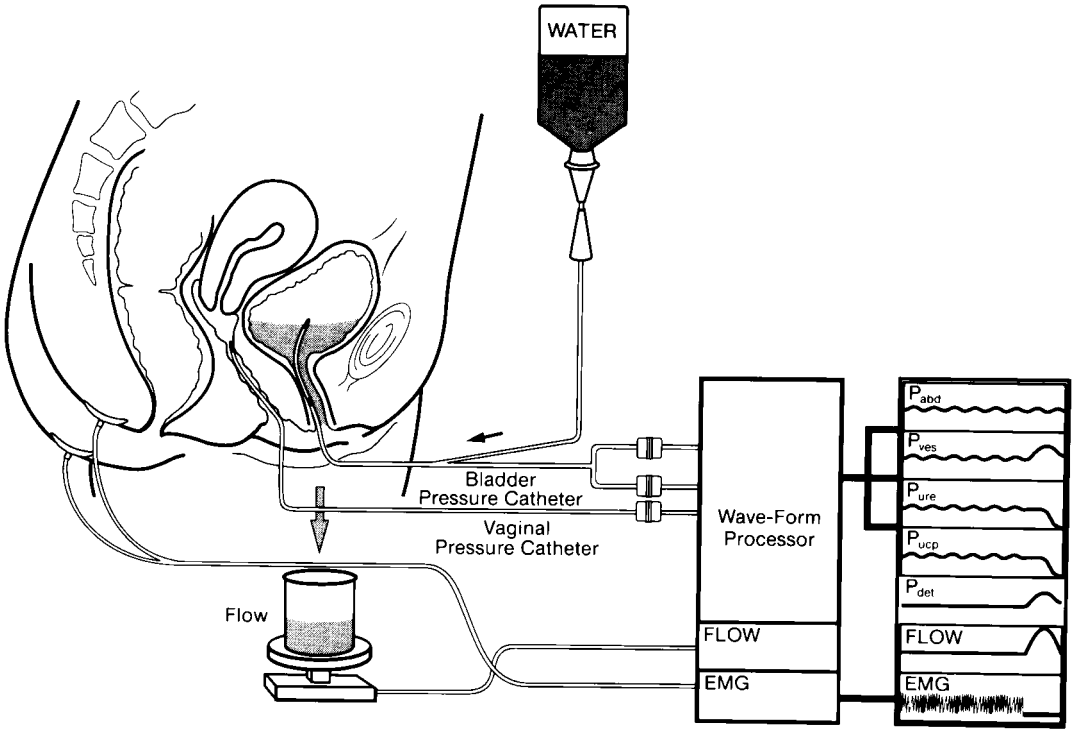
Additional evaluation, including blood testing, multichannel urodynamic testing, and radiographic studies, should be considered on an individualized basis. Blood testing, including BUN, creatinine, glucose, and calcium, is recommended if compromised renal function is suspected or if polyuria is present. Urine cytology and cystoscopy are not necessary in the routine initial evaluation of the incontinent patient but may be helpful for patients with persistent symptoms or coexisting hematuria.

After a presumptive diagnosis has been made, the patient should be treated appropriately and re-evaluated in 6-8 weeks. Referral to a subspecialist will depend on various factors, such as the clinician's experience and comfort level with the diagnosis and management of urinary incontinence, access to urodynamic testing equipment, and healthcare referral patterns. However, patients with significant pelvic prolapse and concurrent urinary tract symptoms, recurrent incontinence, anatomic abnormalities, voiding dysfunction, and co-existing neurologic disorders may benefit from a urogynecologic or urologic consultation. (*See Table 3.*) The role of the subspecialist should include comprehensive urodynamic testing, interpretation of these findings, and further management including surgery as appropriate.

Treatment of Urinary Incontinence

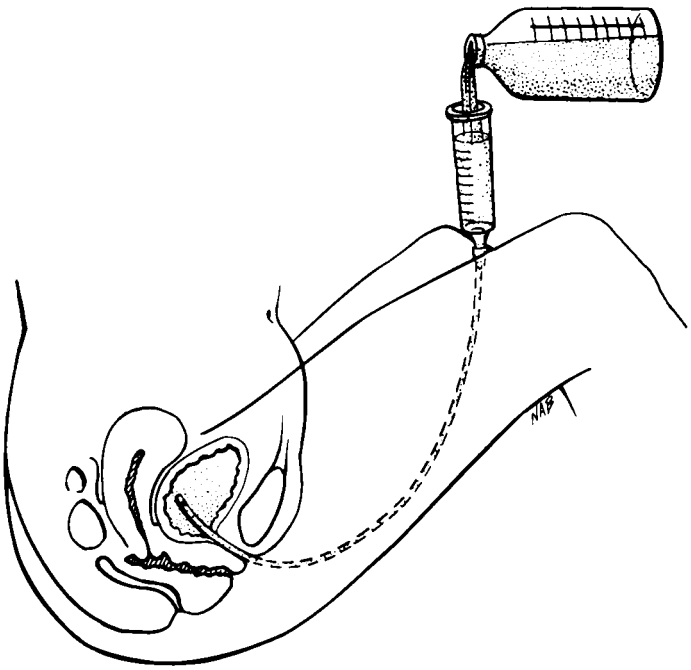
The appropriate treatment of urinary incontinence primarily depends on accurate diagnosis of the underlying etiology. Patients with isolated urge incontinence (detrusor instability) are best treated with conservative treatment options, including bladder retraining and pharmacologic therapy, with the primary goal being to expand bladder capacity to a functional level with resulting inhibition of spontaneous bladder spasms. Patients with detrusor instability refractory to first-line therapy may be candidates for polypharmacotherapy, biofeedback, psychotherapy, or functional electrical stimulation. Surgical treatment of refractory detrusor instability includes bladder distention, sacral neurectomy, and augmentation cystoplasty, but these procedures are associated with significant complications and have produced mixed results. Patients with isolated genuine stress incontinence, due to urethral hypermobility or intrinsic sphincter deficiency, may be candidates for conservative therapy including Kegel exercises, pharmacotherapy, physical therapy with biofeedback, electrical stimulation, or

Figure 3. Complex Multichannel Urodynamics with Pressure Catheters



Complex multichannel urodynamics with pressure catheters allows precise determination of intra-abdominal, intraurethral, and detrusor/urethral pressures. Reprinted with permission from Mosby Year-Book Inc. Walters MD, Karram MM. *Clinical Urogynecology* 1993;1:65.

Figure 4. Simple Office Cystometry



Simple office cystometry is performed with a catheter, syringe, and fluid. Reprinted with permission from Mosby Year-Book Inc. Walters MD, Karram MM. *Clinical Urogynecology* 1993;1:55.

medical devices. We recommend an initial course of conservative therapy for all patients with follow-up assessment in 2-3 months. Surgery using traditional anti-incontinence procedures should be considered for those patients who experience significantly persistent urinary leakage or those who desire surgical correction. Patients with mixed incontinence should be initially offered a trial of conservative treatment consisting of Kegel exercises, bladder training, and pharmacotherapy, as improvement in the urge component of their incontinence may be sufficient to provide the patient with symptomatic relief and alleviate the need for further intervention, including surgery. Patients with a predominant stress component or persistence of their symptoms after a course of conservative therapy may warrant surgical correction. However, it should be clearly explained to these patients that the course of their detrusor instability is unpredictable following surgery and may worsen postoperatively.[10] Patients with secondary causes of incontinence (reflex, overflow, functional, anatomic) should be treated on an individual basis. Patients with significant voiding dysfunction, anatomic abnormalities, and recurrent incontinence pose a challenging problem to the primary care provider. These patients will often require additional testing or complex procedures and urogynecologic or urologic consultation is recommended.

Behavioral Modification

Behavioral modification is useful in the treatment of many types of urinary incontinence and may include dietary restriction, toileting assistance, bladder retraining, and pelvic muscle rehabilitation. Patients with urge incontinence may benefit from dietary restriction of caffeine, alcohol, chocolate, and spicy food, as these can all cause bladder irritation. Routine or scheduled toileting should be offered to incontinent patients on a consistent schedule and is recommended in the treatment of functional incontinence. The mainstay of treatment for urge and mixed incontinence is bladder training with pharmacotherapy. However, several reports demonstrate that bladder training is effective in reducing episodes of stress incontinence, although the exact etiology of this effect is unclear.[11] Bladder training (timed voiding) helps to progressively distend the bladder and allows the patient to regain critical control over voiding patterns. The patient is instructed to void at pre-assigned times during the waking hours. The initial voiding interval is set at less than the patient's current voiding interval, and this is gradually increased on a weekly basis over an eight-week period. Patients are encouraged to try to suppress the sudden urge to void in between these designated intervals. Regular monitoring and positive reinforcement by review of the bladder diary provides continued feedback to the patient.

Pelvic muscle rehabilitation, or Kegel's exercise, can reduce the severity of incontinent episodes by strengthening the pelvic muscles and re-establishing support to the bladder neck continence mechanism.[12] Although pelvic muscle exercises are used predominantly in the treatment of stress urinary incontinence in women with poor pelvic supports and in men post-prostatectomy, there is evidence that it is also useful in patients with urge or mixed incontinence. Unfortunately, only 30% of women can perform pelvic floor exercises correctly following verbal instruction. We recommend simple biofeedback or referral to a physical therapist.[13] Simple biofeedback can be performed in the office at the time of the bimanual exam with the clinician asking the patient to squeeze her levator muscles while two fingers are placed in the vagina. Care must be given to discourage the patient from performing a Valsalva maneuver or tightening her gluteus muscles. Pelvic floor exercises can be enhanced using biofeedback with intravaginally pressure probes or mechanical devices, such as graduated vaginal cone weights. A typical regimen of pelvic floor exercises is based on sets of short and long contractions performed 2-4 times daily. Under this regimen, patients with mild-to-moderate incontinence can expect 60-70% improvement in their symptoms.[14]

Electrical stimulation has been shown to be effective in the treatment of stress, urge, and mixed incontinence. This treatment modality involves the use of non-implantable vaginal or anal sensors or surface electrodes to stimulate a reflex arc in the sacral micturition center and produce a contraction of the pelvic musculature and urethral sphincter with an accompanying reflex inhibition of the detrusor muscle. This treatment modality is especially useful in the patient who is unable to properly perform pelvic muscle exercises or in patients with an acontractile levator muscle. Electrical stimulation is usually given twice daily for 15 minutes and can be performed in the office or at home using a portable generator. Treatment should be continued 8-12 weeks depending on the underlying etiology and on symptomatic improvement. Several studies addressing long-

Table 3. Complex Conditions Requiring Further Consultation or Testing

- Complicated history
- Office cystometry inconclusive
- Frequency, urgency, and pain syndromes unresponsive to conservative therapy
- Stress incontinence before surgical correction
- Recurrent urinary loss after previous surgery for stress incontinence
- Urge-related symptoms with gross or microscopic hematuria
- Nocturnal enuresis unresponsive to previous therapy
- Complaints of stress incontinence with absence of urethral hypermobility
- Advanced pelvic prolapse before surgical correction
- Co-existing neurologic disorders or diabetes mellitus
- Urge incontinence unresponsive to previous therapy
- Age older than 65
- Continuous urinary leakage
- Lower urinary tract dysfunction after pelvic radiation or radical pelvic surgery

term follow-up after pelvic floor electrical stimulation have reported cure rates ranging from 54-77%.[15]

Pharmacotherapy

Pharmacotherapy is the mainstay for the treatment of urge incontinence but can also be used for stress urinary incontinence as an adjunct to other nonsurgical modalities or in patients who do not desire surgical correction. Medications used in the treatment of urinary incontinence function by either relaxing the overactive detrusor muscle in women with urge incontinence or increasing urethral sphincter tone in patients with stress incontinence. Anticholinergic and antispasmodic agents are recommended as the first-line pharmacologic therapy for patients with detrusor instability. These medications mediate the parasympathetic control of the bladder and treat detrusor instability by producing bladder relaxation. Commonly used medications are listed in Table 4. Oxybutynin has long been considered the primary anticholinergic agent of choice. It also has a smooth muscle relaxant and local anesthetic properties. The recommended dose is 2.5-5 mg orally tid-qid, but we find that many elderly patients experience significant side effects at this dosage. We routinely start patients on 2.5 mg/po/bid and then titrate up based on improvement of symptoms and occurrence of side effects. The primary side effects of anticholinergic medications include dry mouth, constipation, blurred vision, change in mental status, and nausea. These medications are contraindicated in patients with narrow angle glaucoma. Other anticholinergic agents, including propantheline, dicyclomine hydrochloride, and flavoxate, may be used as second-line agents in patients with a poor response to oxybutinin. Recently, tolterodine has been introduced as a bladder selective anticholinergic agent that is associated with improved symptoms and reduced side effects. Initial results with its use are encouraging as it is well tolerated in most patients.

Imipramine, a tricyclic antidepressant, has been shown to be effective in the treatment of both stress and urge incontinence. Although the exact mechanism of action is incompletely understood, it seems to work by increasing urethral contractility and suppressing involuntary bladder contractions via its anticholinergic properties. The recommended dosage is 25-100 mg daily. Side effects include orthostatic hypertension, dry mouth, nausea, and hepatic dysfunction. It is contraindicated in patients taking MAO inhibitors.

Calcium channel blockers, such as nifedipine, verapamil, and terodiline, work by blocking the influx of extra-cellular calcium, which is important for detrusor muscle contraction. They have been used extensively in the treatment of detrusor instability in Europe but are still currently under investigation for use in the United States. Side effects include dry mouth, blurred vision, headache, and cardiac arrhythmia. At the present time, these agents are not recommended for general use in the treatment of urinary incontinence.

Alpha-adrenergic agonists, phenylpropanolamine (PPA) and pseudoephedrine, have little effect on the detrusor muscle but can significantly increase urethral pressure by inducing contraction of the urethral sphincter. These agents are useful in the treatment of genuine stress incontinence particularly due to intrinsic sphincter deficiency. Recommended dosage for PPA is 25-100 mg orally in a sustained release form (bid) and for pseudoephedrine, 15-30 mg orally tid. Patients often report improvement in their symptoms within one week. Side effects include drowsiness, dry mouth, and hypertension.

Estrogen replacement, either oral or vaginal, should be used as an adjuctive pharmacologic agent for postmenopausal women with stress urinary incontinence or mixed incontinence. Although the exact mechanisms by which estrogen therapy improves incontinence symptoms is unknown, various theories have been proposed. Estrogen has been shown to alter the vaginal pH and decrease the frequency of urinary tract infections. In addition, estrogen-induced cytologic changes in the urethral mucosa may lead to improve coaptation and re-creation of the mucosal seal. Estrogen may also augment periurethral vascularity with improved function of the smooth and striated periurethral muscles.[16] Finally, the combination of alpha-agonists and estrogen may have synergistic effects. Conjugated estrogen can be administered either orally or vaginally and progestin should be added in patients who have an intact uterus. Side effects include irregular vaginal bleeding, breast tenderness, weight gain, and nausea. Estrogen is contraindicated in patients with a history of breast or gynecologic malignancy.[17]

Medical Devices

Medical devices for the management of urinary incontinence include absorbent products, vaginal support devices, and urethral products.[18] The widespread use of absorbant pads for the symptomatic control of urinary incontinence has prolonged research and technological advances in the treatment of this

socially debilitating condition. Often marketed as "adult diapers" or "absorbant undergarments," these products provide palliative control and often dissuade the patient from seeking medical attention for incontinence that is usually readily treatable. Given the variety of surgical and nonsurgical management options today, current use of absorbant products should be reserved for long-term care of patients with chronic, intractable urinary incontinence.

Medical devices, classified as obstructive or supportive, are designed primarily for the treatment of stress urinary incontinence or mild mixed incontinence due to urethral hypermobility or intrinsic sphincter deficiency. (*See Figure 5.*) Urethral plugs and patches are placed in or over the urethra to occlude the lumen and prevent urinary leakage with increases in abdominal pressure. Complications include urinary tract infection, hematuria, migration of the device into the bladder, and persistent incontinence. Vaginal devices include diaphragms, pessaries, and tampons. These devices function by providing intravaginal support to the bladder neck during episodes of increased abdominal pressure. Complications related to intravaginal devices are less common and include vaginal discharge and pelvic discomfort. Success rates from medical devices have been found to vary depending on the severity of incontinence. Vierhout and Lose have reported subjective success rates ranging from 40-60% depending on the type of device.[19]

Surgical Correction

Although a comprehensive discussion of the various surgical procedures used in the treatment of incontinence is beyond the scope of this paper, it may be helpful to briefly review the major classes of surgery to assist the primary care clinician in providing preliminary information to the patient regarding his/her surgical options. Surgery is used primarily in the treatment of genuine stress urinary incontinence due to urethral hypermobility or intrinsic sphincter deficiency.

In female patients with urethral hypermobility, the goal of the surgical procedure is to restore normal anatomic bladder neck support and prevent descent during increased abdominal pressure. This can be done by a retropubic urethropexy, a transvaginal needle suspension procedure, or a suburethral sling. The retropubic urethropexy is performed through a small suprapubic laparotomy incision, which provides access to the retropubic space. Vaginal tissue underneath the urethra is then suspended to the pubic symphysis (Marshall-Marchetti-Krantz urethropexy) or Cooper's ligament (Burch colposuspension). Recently, the retropubic procedures have been performed laparoscopically with equivalent cure rates on short-term follow-up. The transvaginal needle suspension procedure is mainly performed via the vaginal route with a small abdominal incision. The retropubic space is entered vaginally and support tissue on either side of the urethra is grasped and sutured in a helical stitch. These sutures are then transferred above the anterior rectus fascia to the suprapubic incision via a long needle carrier. Tie down over the rectus fascia results in elevation and stabilization of the bladder neck. The suburethral sling is performed in a similar manner but uses a synthetic or natural graft underneath the urethra rather than incorporating the periurethral tissue in a helical stitch. Risks of these procedures

Table 4. Pharmacologic Agents for the Treatment of Urinary Incontinence

Medication (brand name)	Dosage	Mechanism of Action	Indication
Oxybutynin (Ditropan)	2.5 mg bid - 5mg tid	Anticholinergic/ Spasmolytic	Urge incontinence
Hyoscyamine (Levsin, Cystospaz)	0.15 mg tid to qid 0.375 mg bid to tid (extended release)	Anticholinergic	Urge incontinence
Flavoxilate (Urispas)	100 -200 mg tid to qid	Anticholinergic/ Spasmolytic	Urge incontinence
Tolterodine (Detrol)	2 mg bid	Anticholinergic	Urge incontinence
Propantheline bromide (Pro-Banthine)	7.5 mg tid	Anticholinergic	Urge incontinence
Phenylpropanolamine (Entex)	5 mg bid	Alpha-adrenergic stimulation	Stress incontinence
Psuedoephedrine (Sudafed)	60 mg qid	Alpha-adrenergic stimulation	Stress incontinence
Imipramine (Tofranil)	25-75 mg daily	Anticholinergic and alpha - adrenergic stimulation	Urge incontinence Stress incontinence
Estrogen (Premarin)	0.625 mg po or vaginally, daily	Beneficial effects on urethral mucosa and sphincter	Urge incontinence Stress incontinence

include bleeding, infection, damage to the lower urinary tract, recurrent incontinence, and postoperative urinary retention. In a meta-analysis performed by Jarvis, the long-term success rates for the retropubic urethropexy, transvaginal needle suspension, and suburethral sling have been reported to be 80%, 70%, and 85%, respectively.[20]

In patients with intrinsic sphincter deficiency, the goal of the surgical procedure is to restore the normal continence mechanism by compressing the urethra at the bladder neck. This can be done by a suburethral sling, an artificial urinary sphincter, or periurethral collagen injections. The suburethral sling has been previously described. The artificial urinary sphincter involves placement of a mechanical prosthesis around the bladder neck which can inflate and deflate depending on the need for continence or voiding. Periurethral collagen injections involve injection of bulk enhancing agents, most commonly cross-linked bovine dermal collagen, around the urethra until closure of the internal urethral meatus occurs. This procedure is commonly performed under local anesthesia in the office setting. Risks of these procedures include infection, recurrent incontinence, and postoperative urinary retention.

Newer Techniques

Recently, a number of medical and surgical advances have been made in the treatment of urinary incontinence. A variety of new medications for the treatment of stress and urge incontinence are expected to be released over the next several years. These include new, long-acting delivery systems as well as new pharmacologic agents that should improve treatment outcomes and reduce the incidence of side effects.

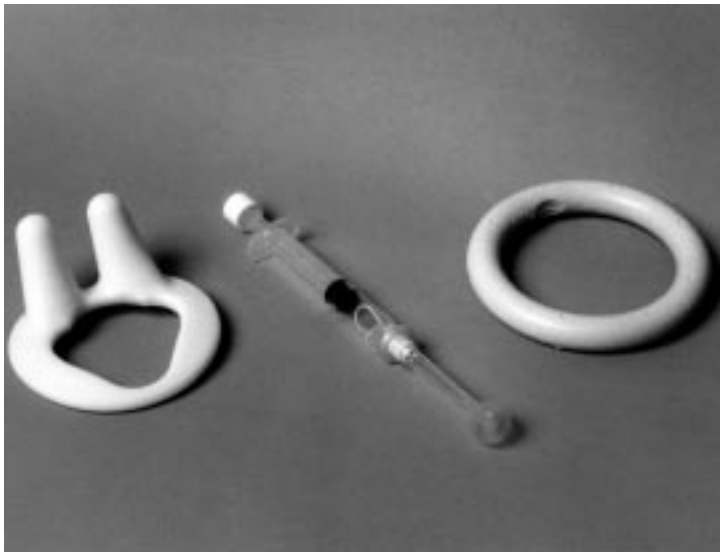
Although pharmacotherapy and behavioral modification are still the mainstay of treatment for detrusor instability, patients with persistent urge incontinence may be candidates for newly introduced implantable nerve root stimulators. The implantable electrode is designed to stimulate the dorsal nerve root of S2, S3, and S4, re-establishing a neurologic equilibrium with relief of persistent urge incontinence. Patients initially undergo placement of a subcutaneous test stimulator on an outpatient basis. Those patients who experience at least a 50% reduction in symptoms are candidates for subsequent placement of a permanent implantable device. Initial results have been encouraging, especially since surgical alternatives for refractory urge incontinence are associated with poor outcomes.

For patients with stress incontinence, new laparoscopic procedures are providing good surgical outcomes with fewer complications and reduced hospital stay. New substances for periurethral injection in the treatment of intrinsic sphincter deficiency are being developed to provide long-lasting results with minimal risk. With increased understanding of this condition and greater media attention, it is expected that more treatment options will be available in the future.

Conclusion

Urinary incontinence is a condition that affects more than 13 million Americans in this country. It is expected that this number will continue to grow as our country continues to age.

Figure 5. Medical Devices for Incontinence



Medical Devices for Incontinence include pessary devices with and without support (left and right) and urethral plugs (center)

The diagnosis and treatment of this disorder will begin with the primary care provider. A basic understanding of the pathophysiology of the lower urinary tract is vital in diagnosing and treating this condition. Initial diagnosis can usually be made on the first visit after history and physical exam. Many patients may require basic additional testing, which can easily be performed in the office setting. Preliminary treatment modalities will either treat or significantly improve the majority of patients. The small subset of patients who have a complex presentation or require surgery will need further consultation and complex urodynamic testing.

The primary care provider serves as the gatekeeper for this common problem. A step-by-step algorithm with an understanding of the available treatment options will allow the clinician to provide care for this condition and identify those patients who require subspecialist referral. In either case, this care will probably make a long-lasting effect in the medical, social, and psychological well-being of patients suffering from this debilitating condition.

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Attention CME Subsribers

Due to an oversight, a mistake has been made with the CME numbering. The numbering in the November 16 issue of *Primary Care Reports*, Volume 4, No. 23, should have read 46-50, instead of 1-5. On your Scantron form inserted with this issue, please follow these instructions. We regret any confusion this may have caused.

Physician CME Questions

51. Which of the following may be a cause of urinary incontinence?
    a. Stool impaction
    b. Medications
    c. Urinary tract infection
    d. Estrogen deficiency
    e. All of the above

52. The factor responsible for maintaining high-resting urethral pressure is:
    a. parasympathetic stimulation of muscarinic receptors.
    b. sympathetic stimulation of alpha-adrenergic receptors.
    c. sympathetic stimulation of beta-adrenergic receptors.
    d. inhibition of alpha-adrenergic receptors.
    e. parasympathetic stimulation of nicotinic receptors.

53. The evaluation of all patients with incontinence should include:
    a. cystoscopy.
    b. urine cytology.
    c. measurement of post-void residual volumes.
    d. intravenous pyelography.
    e. comprehensive urodynamic testing.

54. The first-line medication for the treatment of detrusor instability is:
    a. oxybutinin.
    b. phenylpropanolamine.
    c. flavoxate.
    d. terodiline.
    e. propantheline bromide.

55. Pelvic muscle exercises:
    a. are performed correctly by 90% of women.
    b. should never require physical therapy referral.
    c. may be enhanced by pressure probes or vaginal cones.
    d. are similar to the Valsalva maneuver.
    e. should be discouraged as ineffective in mild cases.

In Future Issues:

Aspirin—New Tricks for an Old Drug
—Steven M. Weisman, PhD, and Carolyn S. Rabe, PhD