Urinary Incontinence and Menopause: Update on Evidence-Based Treatment CME

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Overview

Urinary incontinence is a common but poorly understood problem. The International Continence Society defines incontinence as involuntary loss of urine that is objectively demonstrable and is a social or hygienic problem.[1] In 1998, Thom[2] reviewed published population-based studies of prevalence to determine the estimated prevalence of incontinence stratified by frequency, age, and sex and found rates that varied from 14% to 35%. The Agency for Healthcare Research and Quality (AHRQ) estimates that 13 million Americans are incontinent; 11 million are women.[3]

The Economic Costs of Urinary Incontinence

The economic costs of urinary incontinence can be substantial, with direct costs from diagnosis, treatment, and continuing care, including purchase of products for protection, as well as indirect costs from loss of freedom and independent living. Wagner and Hu[4] reported that incontinence in individuals 65 years and older in the United States in 1995 cost $24.3 billion, or $3565 per individual with incontinence. The AHRQ calculates that in the United States $16.4 billion is spent every year on incontinence-related care: $11.2 billion for community-based programs and at home and $5.2 billion in long-term care facilities. As well, $1.1 billion is spent every year on disposable products for adults. A more recent study by Wilson and colleagues[5] corroborates these estimates. They calculated an annual direct cost of urinary incontinence in the United States of $16.3 billion in 1995 dollars; the cost for women was 3-fold higher than for men, twice as high for women older than 65 years compared with women younger than 65 years, and more than 2-fold higher for community-dwelling women than for institutionalized women.

The Relationship Between Menopause and Urinary Incontinence

The relationship between menopause and urinary incontinence is unknown. Many experts quote menopause as a major risk factor for both stress and urge incontinence. It is theorized that the lack of estrogen in menopause can result in bladder muscle weakness and thinning of the lining of the urethra, which causes improper closure. The combination of a thin, injury-prone urethra and weak bladder muscles can cause the urethra to open unexpectedly during physical activity, leading to stress incontinence.

This article reviews the etiology, diagnosis, and treatment of urinary incontinence in postmenopausal women.

Types and Causes of Urinary Incontinence

Established urinary incontinence can usually be divided into 4 major types: stress incontinence, urge incontinence (detrusor overactivity or instability), mixed incontinence, and overflow incontinence. These disorders often have classic histories or typical physical findings. Neurogenic incontinence may be related to defects in the nervous system, which conducts urination signals between the bladder and the brain. Since neurogenic incontinence is not related to menopause, it will not be covered in this article.

Stress Incontinence

Stress urinary incontinence is diagnosed when the pressure inside the bladder exceeds the pressure in the urethra, in the absence of a detrusor contraction. Patients typically describe losses of small volumes of urine with activities that produce transiently increased intra-abdominal pressure (eg, coughing, sneezing, running, laughing). Urine loss can also result from a reduced muscle tone of the internal and external urinary sphincter. This kind of incontinence occurs mostly in women younger than 60 years. In women, the etiology of urinary stress incontinence is usually presumed to be relaxation of the supporting structures in the pelvis that results from childbirth and the aging process. Atrophy of the genitourinary tissues that results from estrogen deficiency may or may not correlate with symptoms. Pelvic relaxation, including uterine prolapse and cystocele, with resultant urethral hypermobility allows descent of the normal urethrovesical angle. This anatomic distortion renders the urethral sphincter more vulnerable to increased intravesical pressure from any activity. Loss of urine then results. Other less common causes are cauda equina lesions or peripheral neuropathy. Physical examination may reveal evidence of pelvic relaxation, such as cystocele, rectocele, and/or uterine prolapse. Urine loss can usually be demonstrated with coughing while the patient is in the supine or standing position.

Urge Incontinence

Urge incontinence is diagnosed when the detrusor muscle contracts, spontaneously or on provocation, during the filling phase of the bladder, while an individual is attempting to inhibit micturition.[1] Urge incontinence is more common in older adults. This type of incontinence is also known as detrusor overactivity, detrusor instability, detrusor hyperreflexia, uninhibited bladder, or overactive bladder. Patients with detrusor overactivity have early, forceful detrusor contractions well before the bladder is full, which creates a sensation of urinary urgency and frequency. Patients with detrusor overactivity tend to lose small-to-moderate volumes of urine. When the detrusor contraction is strong enough to overcome urethral resistance, incontinence occurs. Detrusor overactivity can be found in conditions of defective central nervous system inhibition or increased afferent sensory stimulation from the bladder. Examples of disorders that impair the ability of the central nervous system to send inhibitory signals include strokes, masses (tumor, aneurysm, hematoma), demyelinating disease (multiple sclerosis), and Parkinson's disease. Increased

afferent stimulation from the bladder can result from lower urinary tract infections, atrophic urethritis, fecal impaction, or uterine prolapse. The diagnosis of detrusor overactivity is made primarily by history and confirmed with urodynamic testing. There are no pathognomonic findings on physical examination, although a careful pelvic and rectal examination and neurologic screening can occasionally reveal anatomic abnormalities (eg, uterine prolapse, fecal impaction) or evidence of neurologic disease.

Mixed Incontinence

Mixed incontinence is a combination of both stress and urge incontinence and is most common in older women.

Overflow Incontinence

Overflow incontinence, in which the bladder becomes too full because it can't be fully emptied, is rare and is the result of bladder obstruction or injury. Those with overflow incontinence commonly present with symptoms of markedly reduced urinary stream, incomplete or unsuccessful voiding, and frequent or even continuous urinary dribbling. Overflow incontinence is generally caused by bladder contractile dysfunction (hypotonic/atonic bladder) or obstructed urinary outflow. In either case, large bladder volumes result in the intravesicular pressure exceeding intraurethral resistance and symptoms of urinary dribbling. Overflow incontinence secondary to an atonic bladder is often transient and occurs after general or regional anesthesia or bladder instrumentation or with the use of various medications, such as narcotics. Contractile dysfunction of the bladder can be caused by disease of the peripheral nerves, for example, diabetic peripheral neuropathy, or sacral nerve roots; it may also be caused by pelvic neoplasm or fecal impaction. Physical examination often reveals a distended bladder and measurement of urine volume after voiding reveals an elevated postvoid residual volume. Patients also demonstrate low urinary flow rates on urodynamic tests.

Other factors can cause incontinence, such as decreased mobility, cognitive impairment, or medications (Table 1).

Table 1. Medications That Can Cause Urinary Incontinence*

Drug	Adverse Effect
Antidepressants, antipsychotics, sedatives/hypnotics	Sedation, retention (overflow)
Diuretics	Frequency, urgency (OAB)
Caffeine	Frequency, urgency (OAB)
Anticholinergics	Retention (overflow)
Alcohol	Sedation, frequency (OAB)
Narcotics	Retention, constipation, sedation (OAB and overflow)
Alpha-adrenergic blockers	Decreased urethral tone (stress incontinence)
Alpha-adrenergic agonists	Increased urethral tone, retention (overflow)
Beta-adrenergic agonists	Inhibited detrusor function, retention (overflow)
Calcium channel blockers	Retention (overflow)
ACE inhibitors	Cough (stress incontinence)

SSRIs**Urinary incontinence (not defined)

*OAB indicates overactive bladder; ACE, angiotensin-converting enzyme.
**SSRIs indicates selective serotonin reuptake inhibitors. A recent retrospective study has found that exposure to SSRIs increases the risk of urinary incontinence.[6]

Evaluation

Evaluation and treatment for incontinence depends on the type of incontinence and the person's age, medical history, and desires for therapy. The assessment for incontinence should include a history, physical examination, and mental, functional, and environmental assessments.
The characteristics of the incontinence should be noted, including the onset, frequency, and severity as determined through the patient's description of the problem and the pattern of incontinence behavior. Voiding diaries can be very helpful in identifying the timing and pattern of voiding. Key questions to be asked include the following: Do you lose (leak) urine unexpectedly? For example, do you lose (leak) urine when you laugh, cough or sneeze, lift something heavy, walk, sleep? Do you lose your urine on the way to the bathroom or toilet? Do you wet the bed at night? Do you go to the bathroom frequently because you are afraid of wetting yourself? Do you use sanitary pads or absorbent products to collect the urine?

Urinary symptoms provide clues to possible causes of the problem, and when combined with the information obtained from a history and physical examination, a provisional diagnosis can often be made. Urinary retention (incomplete bladder emptying) may be the presenting sign of a serious underlying disorder. With urinary retention, the patient may complain of lower abdominal pain with an intense desire to urinate and a feeling of incomplete emptying of her/his bladder. However, people with neurologic injury (ie, spinal cord injury) may experience minimal or no pain. Additional questions to ask the patient to determine voiding dysfunction secondary to outlet obstruction include the following: Do you have dribbling after voiding? Do you void in small quantities? Do you have difficulty initiating urination? How often are you urinating? How many times at night do you awaken to go to the bathroom?

The patient should be thoroughly questioned about related urinary symptoms and habits. Symptoms can be classified as obstructive or irritative. Obstructive symptoms include hesitancy, dribbling, intermittency, impaired trajectory, and sensation of incomplete emptying. Irritative symptoms include nocturia, frequency, urgency, and dysuria. Obstructive symptoms often require referral to a specialist, whereas irritative symptoms can often be controlled by behavioral and pharmaceutical interventions. A urinalysis should be obtained to rule out infection.

A physical examination should be performed of the external genitalia, vagina, and pelvic organs. Women with irritative symptoms may have urethral or vulvar erythema. Evidence of pelvic organ prolapse, especially cystocele, may be associated with incontinence. With Valsalva, women with stress or overflow incontinence may leak urine.

Obtaining a recent medical history can identify acute or reversible causes. Significant medical history includes the number of births, recurrent urinary tract infections, bladder repair surgical procedures, and pelvic radiation. The history should include an assessment of memory

impairment and environmental barriers. A mental status assessment should be performed if the patient has memory loss. Certain environmental barriers, such as the location of the toilet, may be contributing to the incontinence. This is especially true in older persons. In these cases, incontinence may improve with the use of catheters or other urine assistive or collective devices.

Urodynamics

Urodynamic assessment includes a group of tests that measure bladder function. Multichannel urodynamic studies include uroflow, cystometrogram, urethral pressure profiles, and electromyogram. Uroflowmetry studies urinary flow rate and can detect possible outlet obstruction or deficient detrusor contractility. An obstruction may be identified if there is difficulty putting in the catheter. The cystometrogram is a test that compares volume and intravesical pressure and is useful in determining bladder capacity, the compliance of the bladder wall, the stability of the detrusor muscle, and sensations of filling. A urethral pressure profile produces a graphic representation of sphincter closure pressure, functional length, and continuous response to a specific provocative maneuver such as a cough. An electromyogram is a test that measures the activity of the pelvic musculature. Many physicians combine the above 3 studies and perform a voiding pressure study, which provides a detailed assessment of micturition.

Today, multichannel urodynamic studies to document bladder pressure and capacity, muscle contractibility, urethral length, and sphincter control are performed under the auspices of a gynecologist who specializes in disorders of the pelvic floor or a urologist. These studies should be performed if surgery is being considered on the pelvic floor for urinary incontinence.

Treatment

Treatment for incontinence depends on the type of incontinence, its causes, and the capabilities of the patient. The evidence on the effects of clinical interventions will be reviewed below.

Pelvic Muscle Rehabilitation

Pelvic muscle rehabilitation is used to improve pelvic muscle tone and prevent leakage.

Pelvic Floor Exercises

Kegel exercises. Regular, daily exercising of pelvic muscles can improve and even prevent urinary incontinence. This is particularly helpful for younger women. Kegel exercises should be performed 30-80 times daily for at least 8 weeks.

Biofeedback. Used in conjunction with Kegel exercises, biofeedback helps people gain awareness and control of their pelvic muscles.

One review identified 15 randomized, controlled trials (RCTs) on therapy for urge urinary incontinence, of which 8 were considered of sufficient quality and were included in a further analysis.[7] Each RCT included at least 50 women in each group, had appropriate blinding, had a less than 10% dropout rate, and included outcome measures. Women performing pelvic floor muscle exercises in comparison with no treatment were more likely to be dry or mildly incontinent than the no treatment group (61% vs 3%). After 3 months, incontinent episodes were significantly reduced in the treatment group. There was a greater rate of "cure or almost cure" for high-intensity vs low-intensity home-based pelvic floor muscle exercise (60% vs 17%). As well, it found improved social and urodynamic parameters. Biofeedback was compared with pelvic floor muscle exercise in 5 RCTs. One trial found biofeedback significantly improved urge urinary incontinence, whereas the other 4 found no difference.

A separate meta-analysis of the 5 trials for stress urinary incontinence[8] identified in the systematic review of urge incontinence[7] was also performed. The odds ratio for biofeedback combined with pelvic floor muscle exercises, compared with pelvic floor muscle exercises alone, leading to cure was 2.1 (95% confidence interval, 0.99-4.4).[8] The authors concluded that biofeedback may be an important adjunct to pelvic floor muscle exercises alone in the treatment of female genuine stress urinary incontinence.

In addition, an RCT compared pelvic floor muscle training with bladder training or the 2 treatments combined for general female urinary incontinence (ie, regardless of urodynamic diagnosis) and showed that combination therapy was most effective immediately.[9] However, each of the 3 interventions had similar effects 3 months after treatment.

Vaginal Weight Training

Small weights are held within the vagina by tightening the vaginal muscles. This exercise should be performed for 15 minutes, twice daily, for 4-6 weeks.

The systematic review described above identified 3 RCTs that compared pelvic floor muscle exercise alone or in combination with an intravaginal resistance device (1 RCT) or biofeedback (2 RCTs).[7] There was no significant difference in the frequency of incontinent episodes per week. One RCT compared the effect of pelvic floor exercises, electrical stimulation, vaginal cones, and no treatment for genuine stress incontinence. The investigators in this study found training of the pelvic floor muscles was superior to electrical stimulation and vaginal cones.[10]

Pelvic Floor Electrical Stimulation

Mild electrical pulses stimulate muscle contractions. This exercise should be performed in conjunction with Kegel exercises.

Two systematic reviews of RCTs have found conflicting evidence on the effects of electrical stimulation of the pelvic floor in women with stress incontinence.[7,9] RCTs have found it to be less effective than pelvic floor muscle exercises.

Behavioral Therapies

Behavioral therapies are used to assist in regaining control of bladder function.

Bladder Training
Bladder training teaches an individual to resist the urge to void and gradually expand the intervals between voiding. Biofeedback and muscle conditioning, known as bladder training, can alter the bladder's schedule for storing and emptying urine. These techniques are effective for urge and overflow incontinence. The evidence on biofeedback was reviewed above.

Toileting Assistance
Toileting assistance uses routine or scheduled toileting, habit training schedules, and prompted voiding to empty the bladder regularly to prevent leaking. Timed voiding (urinating) and bladder training are techniques that use biofeedback. In timed voiding, the patient fills in a chart of voiding and leaking. From the patterns that appear in her chart, she can plan to empty her bladder before she would otherwise leak.

Pharmacologic Therapies

Alpha-Adrenergic Agonists
Alpha-adrenergic agonists are drugs that stimulate sites in the nervous system that respond to norepinephrine. Therefore, patients with forms of incontinence that require increased muscle tone and urethral resistance may benefit from treatment with these agents.

Phenylpropanolamine Hydrochloride
Phenylpropanolamine hydrochloride was the prototype agent in this class previously found in many prescription and nonprescription cough/cold preparations and antihistamines (antiallergy drugs) before being removed from the US market in 2000. The results of the Hemorrhagic Stroke Project suggested that the use of phenylpropanolamine in appetite suppressants, and possibly cold and cough remedies, is an independent risk factor for hemorrhagic stroke in women.[11] This study provided important data for an assessment of risks associated with the use of this common medication. One systematic review identified a single RCT on phenylpropanolamine,[7] which found no significant difference between pelvic floor muscle exercise and phenylpropanolamine for the treatment of urge urinary incontinence. There are new alpha-adrenergic agents with tissue selectivity in development, namely oxymetolazine and methoxamine.

Muscarinic Receptor Antagonists
Tolterodine tartrate (Detrol) is a muscarinic receptor antagonist; that is, it blocks nerve receptors that respond to muscarine. Both bladder contraction and formation of saliva are controlled by muscarinic receptors. By blocking muscarinic nerve receptors, tolterodine tartrate can reduce symptoms of urinary frequency or urgency and reduce or resolve bladder overactivity and urge incontinence.

Two RCTs showed that tolterodine administration resulted in a significant decrease in the frequency of voiding and improved voided volume, and troublesome or severe side effects were rare.[12,13] Two other RCTs have been performed comparing tolterodine and oxybutynin. One study compared the efficacy and safety of tolterodine given at 1 or 2 mg twice daily vs placebo.[14] At week 4, a statistically significant increase in the volume at first

contraction (P = .03) and maximal cystometric capacity (P = .03) occurred only in the tolterodine, 2 mg twice daily, group. The other RCT studied the clinical efficacy (determined from micturition diaries) and safety of 12-week treatment with either tolterodine, 2 mg twice daily; oxybutynin, 5 mg 3 times daily; or placebo in 277 patients with an overactive bladder.[15] Both tolterodine and oxybutynin significantly increased volume voided/micturition compared with placebo. Both treatment groups evoked greater decreases in micturitions per 24 hours and incontinence episodes per 24 hours compared with placebo; however, only tolterodine was significantly better than placebo in reducing micturition frequency. An extended-release formulation of tolterodine (Detrol LA) has since been developed that requires once-daily administration. A multicenter RCT has shown that the 4-mg once-daily formulation was well tolerated, safe, and even more effective in reducing urge incontinence episodes compared with the twice-daily formulation. In addition, dry mouth was reported with lower frequency.[16]

Anticholinergic Medications

Oxybutynin (Ditropan) prevents urge incontinence by relaxing sphincter muscle. There is one RCT showing the benefit of oxybutynin in reducing the episodes of incontinence.[17] A once-daily formulation (Ditropan XL) has been shown to reduce the number of incontinence episodes with fewer adverse effects than the short-acting formulation.[18-20] After establishing a dose that produced continence or the best balance between continence and adverse effects in each participant, it was shown that maximum benefit was achieved by week 4 and was sustained through 12 weeks of maintenance therapy. Oxybutynin and tolterodine are equivalent in their effectiveness. However, 1 RCT of biofeedback, medication, and placebo showed behavioral treatment was significantly more effective than drug treatment, and both were more effective than the placebo.[21]

Estrogen Therapy

Until relatively recently, estrogen, either oral or vaginal, had been thought to improve incontinent episodes either alone or in conjunction with other treatments for postmenopausal women with incontinence. Both the urethra and trigone of the bladder are covered by nonkeratinized squamous epithelium similar to the vagina.[22] These tissues contain estrogen receptors[23,24] and respond to estrogen.[25,26] In the baboon model, estrogen therapy increased urethral closure pressures, suggesting that estrogen therapy might be effective treatment for incontinence.[27]

There has been one systematic review and 17 uncontrolled trials of estrogen on the treatment of incontinence in women.[28] Although the uncontrolled trials showed subjective improvement of incontinence, the 3 RCTs found no objective improvement in measures of urine loss. Two subsequent RCTs found no significant difference between treatment and control groups in the number of incontinent episodes at 3 and 6 months of follow-up.[29,30] Several large observational studies have shown an increased risk of urinary incontinence in older women undergoing hormone therapy.[31-33] In addition, the well-known RCT, the Heart Estrogen/progestin Replacement Study, found use of menopausal hormone therapy to be associated with worsening of urinary incontinence.[34] Subgroup analysis included 1525 participants who had reported at least once-weekly episodes of incontinence. Within 4 months, the group of women taking the standard combination of conjugated equine estrogen and medroxyprogesterone acetate experienced an overall increase of incontinence episodes compared with those taking placebo (0.7 vs -0.1 episodes, P = .001) The investigators concluded that estrogen could not be recommended as treatment for urinary incontinence in older postmenopausal women.

Combined Estrogen and Alpha-Adrenergic Agonist Therapy

Because estrogen therapy seems to heighten the response of nerve receptors in the urethra (that is, the alpha-adrenergic receptors, which increase the tone of striated and smooth muscle), it is believed that a combination of estrogen and alpha-adrenergic agonists (drugs specific for the alpha-adrenergic receptors) may be beneficial in women who have undergone menopause and who lose bladder control because of insufficiency (malfunction) of the urinary sphincter muscles. Two trials of combination therapy have been performed that concluded that frequency and nocturia improved more with combined treatment than with estrogen alone.[22] However, these studies were performed using prescription and over-the-counter cough and cold preparations, which have since been removed from the US market. When the new alpha adrenergic agents are available, future studies will be needed to determine whether combination therapy will be more effective than single-agent therapy.

Selective Estrogen Receptor Modulators

There is only one published study to date on selective estrogen receptor modulators (SERMs) that bears relevance to postmenopausal urinary incontinence.[35] Goldstein and colleagues analyzed data from 3 RCTs that evaluated the safety of raloxifene, a SERM used to prevent and treat osteoporosis, in 6926 postmenopausal women to determine the frequency of surgery for pelvic floor relaxation. The analysis revealed that raloxifene significantly reduced the frequency of pelvic floor repair (odds ratio, 0.5; 95% confidence interval, 0.31-0.51). The study suggests that raloxifene would not increase urinary incontinence in postmenopausal women and may have a protective effect on the pelvic floor. The side effect profiles of estrogen and raloxifene, which are notably different, are thus further distinguished by this effect.[36]

Inhibitors of Serotonin (5-HT) and Norepinephrine Reuptake: Imipramine and Duloxetine

Imipramine has been found to be effective in treating stress and urge incontinence, although its mechanism of action has not been clarified and there are no published RCTs evaluating this common antidepressant for urinary incontinence.[37-39] Duloxetine has been evaluated in one recently published RCT.[40] The study involved 553 women 18-65 years of age having stress urinary incontinence as a predominant symptom. Subjects were randomized to placebo (n = 138) or 1 of 3 dosages of duloxetine (20 mg/d, n = 138; 40 mg/d, n = 137; or 80 mg/d, n = 140) for 12 weeks of therapy. Incontinence episode frequency recorded in a real-time diary and responses to the Patient Global Impression of Improvement scale and the Incontinence Quality of Life questionnaire were used to determine outcome. The median incontinence episode frequency decreased significantly with the 2 higher doses compared with placebo: duloxetine, 80 mg/d, 64% (P < .001); duloxetine, 40 mg/d, 59% (P = .002), placebo 41%. With the highest dosage, half the subjects had a >/= 64% reduction in incontinence episode frequency (P < .001 vs placebo); 67% had >/= 50% reduction (P = .001 vs placebo). However, significant concurrent dose-dependent increases in the average voiding interval occurred in the duloxetine groups compared with the placebo group. In a subgroup of 163 subjects who had more severe stress urinary incontinence (>/= 14 incontinence episode frequency per week), duloxetine reduced incontinence episode frequency 49% to 64% compared with 30% with placebo. The dropout rates for adverse events were 5% for placebo and 12% and 15% for duloxetine 40 and 80 mg per day, respectively (P = .04). Discontinuation was most commonly attributed to nausea. Duloxetine is not yet on the market.

Table 2. Common Medications Used to Treat Urinary Incontinence

Drug	Dosage
Stress incontinence	
Vaginal estrogen ring (Estring)	Insert into vagina every 3 months
Vaginal estrogen cream	0.5-1 g, apply in vagina every night
Overactive bladder	
Oxybutynin ER (Ditropan XL)	5-15 mg, every morning
Generic oxybutynin	2.5-10 mg, 2- 4 times daily
Tolterodine (Detrol)	
Tolterodine (Detrol LA)	1-2 mg, 2 times daily 4 mg once daily
Imipramine (Tofranil)	10-75 mg, every night
Dicyclomine (Bentyl)	10-20 mg, 4 times daily
Hyoscyamine (Cystospaz)	0.375 mg, 2 times daily

Surgical Therapies

Colposuspension, Anterior Colporrhaphy, Needle Suspension, Slings, and Implantable Devices

There have been 2 systematic reviews of surgery for stress incontinence in women. The first review included 11 RCTs and 20 nonrandomized trials/prospective cohort studies and 45 retrospective studies.[41] This review evaluated limited data and found colposuspension to be the most successful surgical procedure for stress incontinence. There are conflicting data on sling vs colposuspension; however, they appear to have similar cure rates. The review also identified 3 RCTs and 5 nonrandomized studies, which found no difference in effectiveness between anterior colporrhaphy and needle suspensions. There was one RCT showing that tension-free vaginal tape had similar effectiveness as colposuspension at 6 months.[42] The 3-year follow-up has been reported, which shows cure rates similar to those of the 6-month trial.[43] One RCT found urethropexy more effective than colposuspension.[44] Newer treatments, such as artificial sphincters and injectable devices, have not been adequately evaluated in RCTs. In the second review,[45] 7 trials were identified, which included 252 women who underwent an anterior vaginal repair and 517 who received comparison interventions. A single small trial provided insufficient evidence to assess anterior repair in comparison with physical therapy. The performance of anterior repair in comparison with needle suspension appeared similar, but clinically important differences could not be assuredly ruled out. No trials compared anterior repair with suburethral sling operations or laparoscopic retropubic suspensions. In addition, no trials compared alternative vaginal operations. Anterior repair was less effective than open abdominal retropubic suspension based on patient-reported cure rates in 6 trials both in the short term (failure rate within first year after anterior repair; 24% vs 11%; relative risk [RR], 2.15) and long term (after first year; 45% vs 18%; RR, 2.63). Two of these trials used subsequent operations for incontinence as evidence of failure (19% vs 2.9%; RR, 7.12). These findings were regardless of whether prolapse or pelvic relaxation was present. A subsequent prolapse operation appeared to be equally common after vaginal (3.6%) or abdominal (3.6%) operation. For type of open abdominal retropubic suspension, most studies reported comparisons of anterior repair with Burch colposuspension. The few data describing comparison of anterior repair with the Marshall-Marchetti-Krantz procedure were consistent with those for Burch colposuspension.

The reviewers concluded that there were not enough data to allow comparison of anterior vaginal repair with physical therapy or needle suspension for primary urinary stress incontinence in women. Open abdominal retropubic suspension appeared to be better than anterior vaginal repair judged on subjective cure rates in 6 trials, even in women who had prolapse in addition to stress incontinence (4 trials). The need for additional incontinence surgery was also less after the abdominal operation. However, there was not enough information about postoperative complications and morbidity.

Bulking Injections

Newer tissue-bulking agents are primarily used in women with stress incontinence or intrinsic sphincter deficiency to support the urethra. A RCT on periurethral injection of collagen in women with genuine stress incontinence followed up for 5 or more years after injection found no evidence to support the use of periurethral collagen injections in women with intrinsic sphincter deficiency.[46] These women had a higher failure rate than those with hypermobility. A case series of 63 consecutive women with sphincteric incontinence confirmed by urodynamics who underwent a total of 131 transurethral collagen injections were found to have a low short-term cure rate.[47]Durasphere is a water-based gel of sandy material with small carbon-coated beads that is injected through the urethra.[48] Collagen has a rapid absorption rate necessitating repeat injections every 4-12 months. Durasphere has a slower absorption rate so that the supportive effects last longer.

Treatment Recommendations for the Chronically Incontinent

Although treatment can alleviate incontinence in many women, some will never become completely dry. They may need to take medications that cause incontinent episodes or have cognitive or physical impairments that keep them from being able to perform pelvic muscle exercises or retrain their bladders. Many will be cared for in long-term care facilities or at home. The AHRQ guideline update makes the following recommendations to help caregivers keep chronically incontinent patients drier and reduce their cost of care:

Scheduled toileting -- take people to the toilet every 2-4 hours or according to their toilet habits.

Prompted voiding -- check for dryness and encourage use of the toilet.

Improved access to toilets -- use equipment such as canes, walkers, wheelchairs, and devices that raise the seating level of toilets to make toileting easier.

Managing fluids and diet -- eliminate dietary caffeine (for those with urge incontinence) and encourage adequate fiber in the diet.

Disposable absorbent garments -- use to keep people dry and protect clothing.

Education

The AHRQ guideline recommends that patients and professionals learn about the different treatment options for incontinence. Patients and their families should know that incontinence is not inevitable or shameful but is treatable or at least manageable. All management alternatives should be explained. Professional education about incontinence evaluation and treatment should be included in the basic curricula of undergraduate and graduate training programs of all healthcare providers and continuing education programs.