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Urinary Incontinence in the Elderly
Drug Treatment Options

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Contents

Abstract

Urinary incontinence is commonly seen in the elderly. It has multiple complications and is often the reason an elderly person is forced to abandon independent living and enter a nursing home.

There are multiple causes of urinary incontinence: it is not a single entity or a specific diagnosis. In most patients, the incontinence can be placed into 1 of the following 4 categories: detrusor overactivity (urge incontinence), overflow incontinence, stress incontinence (outlet incompetence) or functional incontinence.

To understand the pathophysiology of urinary incontinence, some knowledge of the urinary tract anatomy and physiology is required. It is also important to know how the anatomy and physiology changes in response to the aging process. Successful treatment depends on the specific cause of the incontinence. If incorrectly diagnosed, various treatments may actually worsen the incontinence or cause other problems. Since most elderly patients do not volunteer a problem of urinary incontinence, questions regarding the presence of symptoms must be asked. In most patients, the specific type of incontinence can be diagnosed with a thorough medical history and physical examination. Several simple and widely available laboratory tests may also be useful in the evaluation. Occasionally, urological consultation or urodynamic testing should be performed. Once correctly diagnosed, there are a large number of pharmacological as well as non-pharmacological treatments (behavioural, surgical) available.

This article concentrates on the pharmacological therapies for patients with various types of urinary incontinence. Since most patients with urinary incontinence are elderly, they are more susceptible to the effects as well as the adverse effects of medications. This must be taken into account before any pharmacological therapy is initiated. Although many elderly patients believe their symptoms of urinary incontinence to be a part of growing old, urinary incontinence is never

a normal change of aging. With proper evaluation and treatment, the majority of patients with urinary incontinence can be helped or cured.

Urinary incontinence is extremely common in the elderly and becomes even more common with advancing age and increasing functional dependence.[1] (i.e. people who require assistance from others for basic day to day activities such as feeding and dressing). An estimated 15% of community-dwelling elderly individuals and 50% of the institutionalised elderly have significant urinary incontinence.[2-5] It is felt to be frequently unrecognised, and these numbers probably underestimate the true prevalence since the topic of urinary incontinence is rarely discussed with the primary physician.

Urinary incontinence has several complications, both medical and social.[6,7] It can lead to skin breakdown and contribute to the production of skin ulceration in those who are chronically wet. It is also a common reason for nursing home placement. The care of an incontinent nursing home resident is significantly more expensive than one who is continent, reflecting frequent linen and clothing changes as well as increased nursing time.

1. The Pathophysiology of Urinary Incontinence

Urinary incontinence can often be treated quite successfully with behavioural, pharmacological or occasionally surgical therapy.[8] Effective treatment requires a good understanding of the pathophysiology of urinary incontinence.[9] The neurological control of urination is especially important and involves the CNS, spinal cord and peripheral nerves. Sensory receptors are located within the bladder wall and respond to stretching of the detrusor muscle as urine fills the bladder. The information regarding bladder fullness is transmitted up the spinal cord via the spinothalamic tracts to the brain. Inhibitory signals are sent to the bladder when detrusor relaxation (bladder filling) is desired, and excitatory signals for detrusor contraction (bladder emptying). This information is transmitted from the brain down the spinal cord via the dorsal columns and corticospinal tracts to the urinary bladder.

The urinary bladder has somatic, parasympathetic and sympathetic innervation.[10,11] The pudendal nerve represents the somatic component of bladder innervation and innervates the external urinary sphincter. When stimulated, the sphincter contracts. This occurs with transient increases in intra-abdominal pressure such as coughing, sneezing and laughing. The external sphincter does not play a major role in maintaining continence, since it fatigues rapidly and is only able to remain tightly contracted for a short period of time. A normally functioning internal urinary sphincter is extremely important in maintaining continence; it is innervated by the sympathetic nervous system.[12,13] These nerves originate from the lower thoracic and upper lumbar segments of the spinal cord. When the sympathetic nerves are stimulated, the internal sphincter contracts, preventing urinary flow and facilitating bladder filling. The parasympathetic nerve fibres arise from the second to the fourth segments of the sacral spinal cord, and innervate the detrusor muscle.[10,12,13] When an individual wants to urinate, stimulation of the parasympathetic nerves occurs. The detrusor then contracts, resulting in an elevated intravesical pressure.

The physiology of micturition is complex, but a basic understanding is necessary to appreciate the aetiology and subsequent treatment of urinary incontinence.[14] As the bladder fills with urine, stretch receptors within the bladder wall are stimulated, giving the brain information regarding the amount of urine present within the bladder. Although there is considerable variability from one individual to another, approximately 100 to 200ml of urine is necessary in the average healthy elderly patient before the intravesical pressure rises enough for the brain to recognise a sense of bladder fullness.

With low bladder volumes, stimulation of the sympathetic nervous system and inhibition of the

parasympathetic system results in internal sphincter contraction and detrusor relaxation, facilitating bladder filling. When the bladder is full and micturition is desired, the inhibitory signals from the brain are replaced by impulses which stimulate the parasympathetic system producing detrusor contraction; inhibition of the sympathetic system results in internal sphincter relaxation. The intravesical pressure eventually rises to a point at which it exceeds the resistance within the urethra, and urinary flow occurs. Once the bladder has emptied, the brain again initiates signals resulting in parasympathetic inhibition and sympathetic stimulation producing detrusor relaxation and internal sphincter contraction. The urinary bladder is again ready to be filled with urine.

1.1 Diagnosis and Types of Urinary Incontinence

The treatment for urinary incontinence requires an accurate diagnosis of the type of incontinence present. If an incorrect diagnosis is made, the treatment may actually worsen the incontinence. The history combined with a limited physical examination and selected laboratory tests will usually result in a correct diagnosis. An accurate medical history is the most important component of an evaluation for urinary incontinence. Occasionally, more elaborate testing or a urological consultation will be required.

When urinary incontinence is of recent onset, there is a greater likelihood of cure. Established urinary incontinence is usually more difficult to treat although there is still significant potential to help these individuals. Established incontinence can usually be divided into one of 4 types; detrusor overactivity (urge incontinence), overflow incontinence, outlet incompetence (stress incontinence) or functional incontinence.

2. Detrusor Overactivity/Urge Incontinence

Detrusor overactivity is a common cause of urinary incontinence in the elderly, occurring in 40 to 70% of those who present to the physician with complaints of incontinence.[12,15] This type of incontinence is also known as detrusor instability, detrusor hyperreflexia or uninhibited bladder. Patients with detrusor overactivity have early, forceful detrusor contractions, which occur well before the bladder is full. This creates symptoms of urinary urgency and frequency. Patients with detrusor overactivity describe frequent losses of small to moderate volumes of urine. Symptoms are present during the daytime as well as the night and nocturia every 1 to 2 hours is often described.

Detrusor overactivity can be found in conditions of defective CNS 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 (tumour, aneurysm, haemorrhage), demyelinating disease (multiple sclerosis), Alzheimer's disease and Parkinson's disease.[12,16] Increased afferent stimulation from the bladder can result from lower urinary tract infections (cystitis), atrophic urethritis, fecal impaction or uterine prolapse. Benign prostatic hyperplasia (BPH) is a very common cause of detrusor overactivity in men. It can also produce symptoms of urinary outflow obstruction.

Patients with detrusor overactivity often respond to behavioural therapy, and this should be attempted before pharmacological treatment. Various types of bladder retraining can be used. Noninstitutionalised patients must be motivated to try behavioural therapy and need to be relatively cognitively intact.[17-20] Biofeedback has been shown to be useful in some patients with detrusor overactivity. Transvaginal electrical stimulation has also been used and approximately half of the patients with urge incontinence will have symptomatic improvement. [21] Instructions are given for timed or scheduled voiding, gradually increasing the duration between voidings. It is important for patients to maintain a stable fluid intake. Over half of those treated with behavioural therapy show improvement or cure with this technique. Institutionalised patients, even those with cognitive impairment, can benefit from behavioural training

using scheduled toileting or prompted voiding. For behavioural therapy to be successful, a dedicated and consistent nursing staff is required.[22-24]

Detrusor overactivity often responds well to various pharmaceutical agents. Since acetylcholine is the neurotransmitter which mediates detrusor contraction, medications with anticholinergic effects are often used to suppress the premature detrusor contractions.[25-28] Commonly used medications include oxybutynin[12,15] and flavoxate.[9] Propantheline bromide[15] or dicycloverine (dicyclomine)[29] can also be given. The tricyclic antidepressants imipramine or doxepin are also useful in the management of detrusor overactivity, again due to their anticholinergic activity.[9,12,30] Nortriptyline is often better tolerated in the elderly as it has less anticholinergic activity than other tricyclic antidepressants.

When initiating therapy with these drugs in elderly patients, the starting dosage should be low and gradually increased to a therapeutic level to minimise adverse anticholinergic effects such as dry mouth, dry eyes, constipation, orthostatic hypotension and tachycardia. Drugs with anticholinergic activity also have potential to produce confusion, especially in elderly patients with pre-existing dementia.[9,16,18] They should not be used in those who have narrow-angle glaucoma. Special care must also be taken when using these medications in patients who may have urinary outflow obstruction, especially older men with BPH. Anticholinergic drugs can create difficulty with bladder emptying and precipitate urinary retention.[9,15] Patients taking anticholinergic drugs who develop a urinary tract infection or symptoms of outflow obstruction should have their post-void residual urine volume checked.

Since detrusor overactivity can be caused by atrophic urethritis, topical application of estrogen vaginal cream should be considered in women. Although not approved specifically for use in incontinence, there is evidence that calcium channel antagonists may also be useful in the management of detrusor overactivity due to their direct effect on smooth muscle relaxation.[31] Surgical treatment of BPH will produce resolution of detrusor overactivity symptoms in the majority of patients; however, symptomatic improvement may not occur for several months following the surgical procedure.

3. Overflow Incontinence

Overflow incontinence is much less common than detrusor overactivity. Patients with overflow incontinence commonly present with symptoms of a markedly reduced urinary stream, incomplete voiding and frequent or even continuous urinary dribbling.[9,13,16] Overflow incontinence is generally due to a bladder with contractile dysfunction (hypotonic/atonic bladder) or obstructed urinary outflow.[9,16] In a few individuals, it can result from a dyssynergistic contraction of the bladder and external urethral sphincter.[9] In all cases, the large bladder volumes eventually result in the intravesical pressure exceeding the intraurethral resistance, resulting in urine loss.

Overflow incontinence secondary to a hypotonic or atonic bladder occasionally occurs following general or regional anaesthesia, after bladder instrumentation or with the use of various medications such as narcotics or muscle relaxants. These patients often benefit from bladder drainage by catheterisation for a few days, after which normal bladder function usually returns. Contractile dysfunction of the bladder can be caused by disease of the peripheral nerves, such as peripheral neuropathy secondary to diabetes mellitus or vitamin B_{12} deficiency.[10,18] Damage to sacral nerve roots or the cauda equina can also cause defective innervation to the bladder. Bladder outlet obstruction in men is usually secondary to an enlarged prostate from BPH. It may also be caused by pelvic neoplasm or fecal impaction.

Patients with overflow incontinence secondary to outflow obstruction due to BPH may benefit from medications with peripheral sympathetic antagonist activity. The neurotransmitter to the internal urinary sphincter is noradrenaline (norepinephrine). Blockade of this neurotransmitter will decrease the force of contraction of the internal

sphincter. α-Adrenergic antagonists include prazosin, terazosin[32] or doxazosin. Tamsulosin is a new α-adrenergic antagonist with greater α_1 specificity. The prostate has a higher percentage of α_{1A}, α_{1L} and α_{1D} receptors. These receptors seem to be responsible for urethral contraction. Tamsulosin is the first α-adrenergic antagonist which specifically targets the α_{1A} and α_{1D} receptors.[33] These agents must be used cautiously in the elderly because of their propensity to produce orthostatic hypotension. Many elderly people have age-related baroreceptor dysfunction and an increased susceptibility to orthostatic hypotension. Initial doses of these medications should be low, then gradually increased as tolerated. Patients should be warned about possible lightheadedness or syncope.

Finasteride may also be of benefit in men with urinary outflow obstruction secondary to BPH. The medication works directly on the prostatic tissue and can gradually reduce the size of the prostate gland such that improvement in urinary flow may occur – however, an immediate effect should not be anticipated. Finasteride inhibits the enzyme 5α-reductase. This inhibition deprives the prostate of the major androgen dihydrotestosterone, which reverses the progression of BPH.[34] Although the benefit of finasteride in the treatment of BPH has recently been questioned,[35,36] its use has been shown to produce a 57% decrease in the incidence of acute urinary retention over a 2-year period. It has also been shown to produce a 34% reduction in surgical intervention for BPH.[37] The benefit of finasteride in the treatment of urinary incontinence is unclear.

Patients with overflow incontinence resulting from a hypotonic or atonic bladder of recent onset, precipitated by anaesthesia or surgery, should initially be treated with bladder catheterisation.[12] The goal is to avoid overdistension of the bladder. Intermittent catheterisations are performed, their frequency determined from the residual urine volume. Some of these patients may also benefit from the temporary use of pharmacological agents. The majority will regain their normal bladder function with time. Since the neurotransmitter to the detrusor muscle mediating detrusor contraction is acetylcholine, benefit from medications with cholinergic agonist activity, such as bethanechol chloride, can be anticipated. Adverse effects are common, especially in the elderly, and include abdominal cramps and diarrhoea. Cholinergic agonists should be avoided in those with heart disease or asthma.[9,18] While pharmacological therapy for overflow incontinence may be of benefit in the short term, there is little evidence to suggest that long term success can be expected. Postoperative patients with no prior history of bladder dysfunction are those most likely to benefit from short term pharmacological therapy.

Patients with overflow incontinence can also be instructed in assistive voiding techniques (e.g. abdominal strain, Crede manoeuvre). The Crede manoeuvre involves placing external pressure on the lower abdomen to expel urine and is typically used in patients with neurogenic bladders. However, in instances when the sphincter is not denervated, the pressure can transmit urine to the upper tract. This is especially true in children with vesicoureteral reflux.[38] Patients with BPH who do not respond to medical therapy should be considered for surgical correction [transurethral resection of the prostate (TURP)] unless their bladder is atonic. Surgery may also produce a resolution of symptoms in those with significant uterine prolapse.

4. Stress Incontinence/Outlet Incompetence

Urinary stress incontinence, also known as outlet incompetence, is the most common cause of incontinence in women.[8] Although this type of incontinence can occur in men, it is usually limited to those who have had internal sphincter damage from various urological procedures. Patients describe losses of small volumes of urine with activities producing transiently increased intra-abdominal pressure (e.g. coughing, sneezing, running, laughing). It is not uncommon to have a combination or 'mixed type' of urinary stress incontinence and detrusor overactivity.[8] The loss of

small amounts of urine from stress incontinence can trigger a detrusor contraction.

In women, the aetiology of urinary stress incontinence is usually pelvic relaxation resulting from multiple childbirths combined with the aging process.[15-18] These changes become more pronounced following menopause as estrogen deficiency allows atrophy of the genitourinary tissues.[8,9] Pelvic relaxation allows 'funnelling' of the bladder neck, increasing the normal urethrovesical angle beyond 100°. This predisposes the urethral sphincter to greater difficulty in preventing urine flow with increased intravesical pressure. Loss of urine then results. A second, less common cause of urinary stress incontinence is an abnormally low urethral closure pressure called intrinsic sphincter deficiency.[8] It is often a result of operative trauma. In some women, intrinsic sphincter deficiency occurs with urethral atrophy which is superimposed on normal, age-related decreases in urethral pressure.[39] Despite this natural reduction in pressure and pelvic muscle relaxation, stress incontinence is a pathological process and is not a normal part of aging. Finally, cauda equina lesions or peripheral neuropathy can also, rarely, result in stress incontinence. Physical examination may reveal evidence of pelvic relaxation, such as cystocele, rectocele and/or uterine prolapse. Atrophic urethritis can be inferred if there is evidence of atrophic vaginitis. Urine loss can often be demonstrated with coughing while the patient is in the standing or supine position.

Pelvic floor exercises (e.g. Kegel exercises) can be effective in the treatment of urinary stress incontinence in motivated individuals.[40,41] These exercises strengthen both the periurethral and pelvic floor muscles and as a result can increase resistance within the urethra.[16,18] They are easy to perform, but must be performed frequently throughout the day and continued for an extended period of time in order to achieve a long term effect. Patients can be taught to identify the pelvic floor muscles by attempting interruption of voiding or by digital palpation during contraction. Most authors recommend 10 to 20 pelvic floor contractions for 10 seconds each, 3 times daily. An adequate period of relaxation between contractions is important. It may take 6 to 8 weeks before any beneficial effect is noted. Approximately 40 to 50% of women will have a >50% improvement in incontinent episodes using pelvic floor exercises.[42] These results compare favourably with pharmacological treatment. Continued benefit after improvement with pelvic floor strengthening depends on the patient's motivation and ability to continue practising the pelvic floor exercises. Another option for treatment of urinary stress incontinence is a pessary. These have been shown to be effective in up to 80% of women; however, they often cause local vaginal irritation.[43]

A goal in the treatment of urinary stress incontinence is to produce an increase in the internal sphincter tone. Since the neurotransmitter to the internal urinary sphincter is noradrenaline, α-adrenergic agonists such as phenylpropanolamine,[9,16,44] pseudoephedrine or imipramine can increase the internal sphincter tone and bladder outflow resistance. Long term use of these medications has not been proven to be beneficial, although intermittent use (e.g. before planned activities) may provide limited improvement in symptoms. These drugs should be used cautiously in patients with hypertension or a history of cardiac arrhythmias. Imipramine also has significant anticholinergic activity which can cause problems in the elderly.[9,12,18]

Improvement of genitourinary atrophy with the use of estrogen replacement therapy can be very helpful in improving periurethral and vaginal tissue thickness and quality.[18,45] Topical, oral or transdermal estrogen preparations are all effective.[16] Since the problem is a local one, topical hormonal therapy is generally adequate, unless other systemic estrogen effects are desired. Systemic estrogen is associated with breast tenderness and headaches. They can also produce vaginal bleeding in those with an intact uterus and need to be given with a progestin to prevent an increased risk of endometrial cancer. Combining hormonal therapy with an α-agonist can provide benefits greater than either product alone.[46]

There are several surgical procedures which may also prove helpful for stress incontinence due to pelvic relaxation or internal sphincter insufficiency.[47-49] In women, correction of pelvic relaxation and re-establishment of the normal urethrovesical angle with bladder neck suspension surgery can improve urinary stress incontinence. The surgery re-establishes the normal posterior urethrovesical angle of 90 to 100°. Placement of an artificial sphincter (usually as a last resort) may also be beneficial in women or men who have developed complete sphincter insufficiency.[50,51] Which specific surgical procedure for stress incontinence is best is currently under debate. Retropubic suspensions and slings appear to be the most efficacious procedures for long term care. These procedures also carry a slightly higher complication rate. Transvaginal suspensions and anterior repairs are also acceptable procedures in many instances.[52] Collagen injection therapy is often used as a means to improve stress incontinence. Teflon, autologous fat, silicone or glutaraldehyde cross-linked bovine collagen is injected close to the urethral lumen, compressing or narrowing the lumen. Using this procedure, 74% of patients from recent studies showed a significant improvement.[53]

5. Functional Incontinence

Patients with functional incontinence are those who would otherwise be continent but who, as a result of some (usually external) factor, become incontinent. Patients with functional incontinence often have symptoms of recent onset. When this is the case, there is a much better prognosis for cure.[8,16,18,32] If the cause can be identified and corrected, the incontinence usually resolves. Common causes of recent onset (functional) urinary incontinence include the following:

Change in mental status: A transient deterioration in mental status (e.g. delirium) can result in urinary incontinence. Correcting the cause of the delirium usually results in resolution of the incontinence.[16]

Urinary tract infection: Asymptomatic bacteriuria is common in the elderly and is not associated with urinary incontinence.[54-56] An attempt at treatment is reasonable when bacteriuria is found in an individual who has recently developed urinary incontinence since acute urinary tract infections can result in the development of urinary incontinence.

Atrophic urethritis: This can be treated with hormonal therapy (topical or systemic).[18,44]

Medications: Some medications can contribute to the development of urinary incontinence (table I), either as a result of increased urine production (diuretics), or by direct effects on the detrusor (anticholinergics) or internal sphincter (sympathetic agonists/antagonists).[16] Potent diuretics such as furosemide (frusemide) or bumetanide can produce urine at a rapid rate resulting in symptoms of urinary frequency.[16] Medications which have anticholinergic effects such as tricyclic antidepressants, antispasmodics or antihistamines can decrease the contraction of the detrusor, potentially leading to urinary retention.[16] This is also true for medications which have direct effects on the bladder muscle, including opioids and calcium channel antagonists which can impair the contractility of the detrusor.[15]

Medications with sympathetic activity can have an effect on the internal urinary sphincter. Sympathetic agonists such as pseudoephedrine can increase the tone of the internal sphincter, a desired effect in patients with outlet incompetence and urinary stress incontinence. However, in elderly men with urinary outflow obstruction, medications with sympathetic agonist activity can result in incomplete bladder emptying.[16] Similarly, sympathetic

Table I. Medications which can contribute to the development of urinary incontinence

Drug class	Effect
Diuretics	Can result in brisk filling of bladder
Anticholinergics	Impair detrusor contraction
Sedative/hypnotics	Can produce confusion
Opioids	Impair detrusor contraction
α-Adrenergic agonists	Increase tone of internal sphincter
α-Adrenergic antagonists	Decrease tone of internal sphincter
Calcium channel blockers	Decrease detrusor contraction

antagonists such as peripheral α-antagonists (prazosin, terazosin, doxazosin, tamsulosin) can produce relaxation of the internal urinary sphincter and potentially worsen urinary stress incontinence in women with outflow incompetence.[18] Angiotensin converting enzyme (ACE) inhibitors can produce a chronic nonproductive cough which can induce or worsen the symptoms of urinary stress incontinence.[16]

Depression: Although atypical, depression in the elderly can present with the development of urinary incontinence.[10]

Excessive urine production: This may be secondary to potent diuretics or metabolic disorders which result in polyuria (e.g. hyperglycaemia, hyperalcaemia).[57] Medical conditions associated with excessive fluid accumulation such as peripheral oedema from venous insufficiency or congestive heart failure can produce nocturnal incontinence due to fluid shifts and increased urinary flow which occurs when the individual assumes a recumbent position.

Restricted mobility: Problems with mobility from arthritis, muscular weakness or previous stroke, especially when combined with the use of restraints, can result in an incontinent patient. In some, the distance from the toilet facility can decide whether a patient is continent or incontinent.

Fecal impaction: This may cause a mechanical obstruction to urinary outflow, or produce increased stimulation of the bladder resulting in premature detrusor contractions.[12]

6. Conclusion

A mnemonic ('DIAPPERS') has been developed to help the practitioner remember the causes of recent onset of urinary incontinence.[58]

D delirium
I infection
A atrophic urethritis
P pharmaceuticals
P psychological
E excessive urine output
R restricted mobility
S stool impaction

An evaluation of and attempt to treat urinary incontinence can be a very rewarding experience. Urinary incontinence can be devastating to patients, often resulting in social withdrawal which can lead to an increase in functional dependence and depression. It is a common reason for nursing home placement.[16] Yet, in most cases, incontinence can be treated very effectively. Since patients rarely voluntarily initiate a discussion of their incontinence, they need to be asked about any potential symptoms. The evaluation is neither difficult, nor commonly requires elaborate testing or special equipment. Using a combination of behavioural, pharmacological and, in some cases, surgical therapy, most patients with urinary incontinence can achieve substantial improvement or complete resolution of their symptoms.[8]

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