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Urinary incontinence

Pathophysiology and management outline

Background
Urinary incontinence is common in the community and may impact significantly on quality of life; yet only one-third of sufferers seek medical attention. There are many treatment options for patients suffering with urinary incontinence.

Objective
This article aims to aid general practitioners in the managment urinary incontinence. We outline the pathophysiology of urinary incontinence in women and provide a primary care treatment paradigm. Suggestions for when specialist referral would be of benefit are also discussed.

Discussion
Most urinary incontinence can be evaluated and treated in the primary care setting after careful history and simple clinical assessment. Initial treatment, for both urge urinary incontinence and stress urinary incontinence, is lifestyle modification and pelvic floor muscle treatment. Urinary urgency responds to bladder training and pharmacotherapy with anticholinergic medication. Pharmacotherapy has a limited place in stress incontinence. If there is complex symptomatology or primary management fails, then referral to a specialist is suggested.

The International Continence Society (ICS) defines urinary incontinence (UI) as the complaint of any involuntary leakage of urine.[1] It is a distressing and debilitating condition that is becoming more prevalent as our population ages. It significantly impacts on quality of life, both physically and psychosocially and has major economic ramifications. The incidence of UI is higher in women and is currently estimated to affect 4 million Australians.[2] While UI is common, only one-third of sufferers seek medical attention, possibly due to social stigma or ignorance regarding available treatments.[3] Furthermore, one in 5 women with UI also experiences some degree of faecal incontinence. By effectively identifying and treating incontinence it is possible to significantly improve patients' quality of life.

It is essential that general practitioners understand the manifestations of this condition and its treatments to be able to broach this sensitive subject with their patients.[4]

Continence mechanisms in women

Continence is maintained by a coordinated effort between the bladder, urethra, pelvic muscles and the surrounding connective tissue. The function of the lower urinary tract is to either store (storage phase) or expel (voiding phase) urine. This is dependent upon a bladder that is able to expand while maintaining a relatively constant low pressure in the absence of involuntary contractions. The body of the bladder is innervated by parasympathetic nerves while the bladder neck receives sympathetic innervation (*Figure 1*). Normal urine storage is dependent on a closed outlet and a relaxed bladder.[5]

Outlet closure is dependent on the bladder neck and urethral smooth muscle with a skeletal muscle rhabdosphincter, which is under voluntary control (somatic). The outlet remains closed during urine storage and the rhabdosphincter and pelvic floor respond to rises in intra-abdominal pressure. Intact urethral mucosal is also important for a watertight seal. Continence is maintained while the urethral pressure exceeds intravesical pressure.

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The voiding phase starts with relaxation of the outlet with a sustained detrusor contraction. The micturition reflex is normally under voluntary control and coordinated by the pontine micturition centre with various relays in the spinal cord (*Figure 1*). Details of voiding and storage phases are listed in *Table 1*.

Pathophysiology and terminology

Urinary incontinence occurs when there is dysfunction in either the storage function or occasionally, in the emptying function of the lower urinary tract. Urethral sphincter dysfunction and bladder dysfunction can co-exist and various components of the continence mechanism may compensate one another. For example, women may experience anatomical or neuromuscular injury during childbirth but remain asymptomatic until there is a loss of urethral sphincter function due to aging. The ICS has defined nomenclature for UI as: stress (SUI), urge (UUI) and mixed (MUI) (*Table 2*).

Stress urinary incontinence

Stress urinary incontinence occurs when vesical pressure exceeds urethral pressure in the setting of sudden increases in intra-abdominal pressure. This can be due to weakness of the pelvic floor or sphincter. Loss of bladder neck support is referred to as bladder neck hypermobility and treatments target the restoration of that support. Sphincter dysfunction is referred to as intrinsic sphincter deficiency. It is believed that most patients have elements of both disorders in varying degrees. Risk factors for SUI include childbirth, postmenopausal involution of the urethra, or as a complication of pelvic surgery or trauma.

Urge urinary incontinence

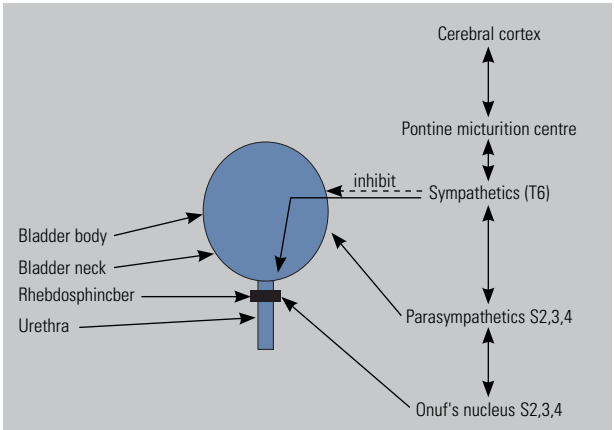
Urge urinary incontinence can be caused by detrusor overactivity or low compliance. Detrusor overactivity is a urodynamic observation characterised by involuntary detrusor contractions during the filling phase. Detrusor overactivity may be neurogenic or idiopathic.

Detrusor overactivity can originate from the bladder epithelium or detrusor muscle itself. The myogenic theory[6] suggests that age related changes in smooth muscle lead to hyperexcitability. The neurogenic theory[7] suggests that detrusor overactivity can be due to denervation at the spinal or cortical level, leading to hyperactive voiding secondary to the spinal micturition reflexes. Parkinson disease or stroke may cause a loss of inhibitory neurons, leading to neurogenic detrusor overactivity.

History and examination

As up to two-thirds of UI sufferers may not seek medical treatment for their symptoms, it is important to broach the subject with women and give them the opportunity to discuss their symptoms. The type of UI can most often be diagnosed by history alone. Storage symptoms include frequency, nocturia, urgency and incontinence. Voiding symptoms include hesitancy, poor or interrupted stream, straining and terminal dribbling. Frequency of incontinent episodes, pad usage, degree of bother, circumstances of loss, time of day (or night) and relationship to drug treatments (eg. diuretics, alpha blockers), voiding habits and fluid intake need to be evaluated. A history of urinary tract infection or poorly controlled diabetes can also impact on lower urinary tract function and constipation needs to be excluded. Haematuria (microscopic or macroscopic) or irritative symptoms require assessment to exclude malignancy.

Figure 1. Anatomy and innervation of the bladder



A bladder diary or frequency and volume chart is simple and useful for initial assessment and quality of life evaluation.[8,9]

Examinations are focused on organ systems that could be implicated in UI. Initial assessment includes general observation for mobility, cognitive status, peripheral oedema and body habitus; abdominal examination for pelvic masses and focused neurological examination for upper motor neuron lesions (eg. multiple sclerosis, Parkinson disease) or lower motor neuron lesions (eg. sacral nerve root lesion) if suspected. Vaginal examination is performed to assess oestrogen status, the presence of pelvic organ prolapse, urethral meatal abnormality, pelvic floor muscle tone and leakage during coughing or Valsalva manoeuvre. The strength of the pelvic floor muscles can be assessed during the bimanual examination by asking patients to contract the muscles around the fingers of examining physician.

Urine microscopy and culture is required to exclude infection as well as postvoid residual volume by ultrasound. In patients with suspected voiding difficulties or neuropathy, previous failed treatment or when considering surgical treatment, cystometry or urodynamic studies, can be performed.[10,11] In cases where central nervous system pathology is suspected, the opinion of a neurologist may be required.

Assessment of UI is discussed in further detail in the article 'Urinary incontinence – assessment in women: stress, urge or both?' by Karen McKertich in this issue of *AFP*.

Table 1. Summary of the functions of the autonomic and somatic nervous systems

	Storage phase	Voiding phase
Somatic (Onuf's nucleus)	Contracted rhabdosphincter	Relaxed rhabdosphincter
Sympathetic	Contracts bladder neck and inhibits contraction of bladder body	Relaxes bladder neck
Parasympathetic	No action	Contracts bladder
Sensory (afferent) fibres run with the autonomic nerves		

Treatment

The aims of treatment are to reduce symptoms and improve quality of life through nonsurgical and surgical therapies.

Nonsurgical therapy

Lifestyle intervention

Weight loss and exercise in morbidly obese patients reduces SUI, and to a certain extent, UUI.[12,13] Fluid and caffeine restriction may also reduce UI,[14] while the effect of smoking on UI is unclear.[15,16] Constipation and straining may increase the risk of pelvic organ prolapse and SUI.

Pelvic floor exercise

Pelvic floor muscle training (PFMT) involves strengthening the pelvic floor muscles. It needs to be continued for 3–4 months before determining its success. It should be done with three sets of 8–12 slow maximal contractions sustained for 6–8 seconds and repeated 3–4 times per week. A 2001 Cochrane review has shown that women undergoing PFMT were seven times more likely to be cured and 23 times more likely to show improvement.[17] It can be combined with biofeedback equipment such as intravaginal resistance devices or weighted vaginal cones. However, these have not been shown to improve the efficacy of PFMT.[17,18]

Bladder training

Bladder training is the initial treatment for UUI, being noninvasive, inexpensive and easy. This includes PFMT, a scheduled voiding program with gradual increases in the duration between voids, and urge suppression techniques with distraction or relaxation. A Cochrane review suggests bladder training may be more effective than placebo, however, there was not enough data to determine whether bladder training was a useful supplement to other therapies.[19] The World Health Organization and the International Consultation on Incontinence recommended that initial bladder training involves a voiding interval of 1 hour during waking hours with a gradual increase by 15–30 minutes per week until a 2–3 hour voiding interval is reached.[20]

Pelvic floor muscle training and bladder training are best undertaken with the assistance of a continence therapist (see *Resources*). The role of the physiotherapist in PFMT and bladder training is discussed in the article 'Physiotherapy for urinary incontinence' by Patricia Neumann and Shan Morrison in this issue of *AFP*.

Table 2. International Continence Society definition of the symptoms of urinary incontinence[1,26]

Stress urinary incontinence (SUI)	The complaint of involuntary leakage on effort or exertion, or on sneezing or coughing
Urge urinary incontinence (UUI)	The complaint of involuntary leakage accompanied by, or immediately preceded by, urgency. (Urgency is a sudden compelling desire to pass urine, which is difficult to defer)
Mixed urinary incontinence (MUI)	The complaint of involuntary leakage associated with urgency and also with exertion, effort, sneezing or coughing

Pharmacotherapy

Anticholinergic medications form the basis of pharmacological treatment in UUI by reducing involuntary detrusor contractions mediated by acetylcholine. A Cochrane review showed anticholinergic medications are effective in reducing symptoms of urgency and improve quality of life and symptoms during treatment when compared with, or combined with, bladder training alone.[21,22] In Australia, the most commonly used anticholinergic drug is oxybutynin. Newer uroselective anticholinergic medications including tolterodine, solifenacin and darefenacin have similar efficacy to oxybutynin but an improved side effect profile. As yet they are not available on the Pharmaceutical Benefits Scheme (*Table 3*).

Medical treatments are less effective for SUI. However, there is evidence that serotonin noradrenaline re-uptake inhibitors (SNRIs) may be effective in SUI.[23] Duloxetine, a SNRI, relaxes the bladder and increases outlet resistance. However, adverse effects are common including nausea, fatigue, dry mouth and constipation.[24]

Other medications

Imipramine, a tricyclic antidepressant, may reduce detrusor contractility and increases outlet resistance and can be used in conjunction with anticholinergics. There is little evidence to support the use of oestrogen replacement for UI.[25]

Surgical therapy

If symptomatology is complex or pharmacotherapy unsuccessful, then specialist referral is warranted for further assessment and treatment.

For detrusor overactivity refractory to oral medications, intravesical botulinum toxin A injections or neuromodulation can be performed, usually in specialist units. More invasive options include detrusor myomectomy or bladder augmentation, which reduce the efficacy of detrusor contraction and thus improve continence. As a last resort, ileal conduit urinary diversion may be performed.

Table 3. Anticholinergic medications for urge urinary incontinence

Medication/formulation	Uroselective	Usual dosage	Comments
Oxybutynin (Ditropan)	No	2.5–5 mg orally 2–4 times per day (geriatric dose 2.5 mg)	• Effective and inexpensive • Side effects include constipation, dry mouth, blurred vision • May precipitate acute urinary retention • In the elderly may cause confusion and sedation • Available on the PBS
Oxybutynin transdermal patch (Oxytrol)	No	39 cm^2 patch 2 times/week (3.9 mg/day	• Side effects of oxybutynin are due to metabolites which may be reduced by newer transdermal delivery system • Not available on the PBS
Tolterodine (Detrol)	Yes	2–4 mg orally per day	• Comparable efficacy to oxybutynin • Improved side effect profile • No PBS listing as yet
Darifenacin hydrobromide (Enablex)	Yes	7.5–15 mg orally once per day	• Comparable efficacy to oxybutynin • Improved side effect profile • No PBS listing as yet
Solifenacin (Vesicare)	Yes	5 mg/day orally	• Comparable efficacy to oxybutynin • Improved side effect profile • No PBS listing as yet

Figure 2. Demonstration of midurethral sling placed via a retropubic approach

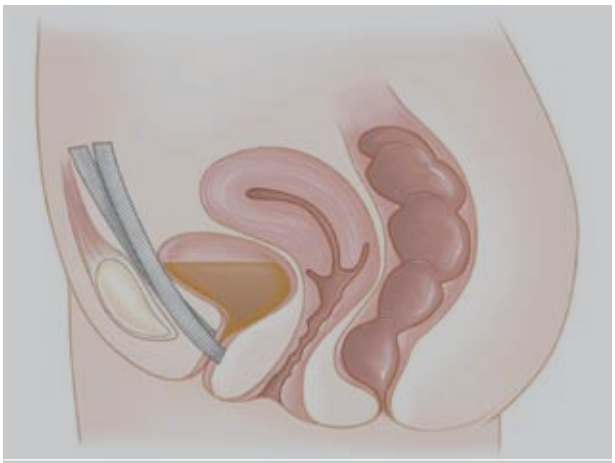


Image courtesy American Medical Systems

Stress urinary incontinence can be treated with colposuspension, pubovaginal sling with rectus fascia or midurethral tapes. There are various midurethral tape kits available using a retropubic or transobturator route. These slings aim to reduce bladder neck hypermobility (*Figure 2*). Periuretheral bulking agents may also be used and work well for some patients.

Further details of these procedures for treating both SUI and UUI are available in the article 'Urinary incontinence: procedural and surgical treatments for women' by Karen McKertich in this issue of *AFP*.

Conclusion

Urinary incontinence is common in women but it is under-reported and undertreated. Generally UI is caused by aging, childbirth, pelvic surgery or neurological disorders. Most UI can be evaluated and treated in the primary care setting after careful history and simple clinical assessment. This can include lifestyle modification, PFMT, bladder training and/or pharmacotherapy. If there is complex symptomatology or primary management fails, then referral to a specialist is suggested. Urinary incontinence can be very distressing both physically and psychologically and impacts on quality of life and health. As primary care providers, it is essential that GPs evaluate, treat and refer high risk patients.

Resource

Continence Foundation of Australia
Freecall 1800 33 00 66 www.continence.org.au.

Conflict of interest: none declared.

References

1. Abrams P, Cardozo L, Fall M, et al. The standardisation of terminology of lower urinary tract function: report from the Standardisation Sub-committee of the International Continence Society. Am J Obstet Gynecol 2002;187:116–26.
2. Continence Foundation of Australia. Available at www.continence.org.au.
3. Holst K, Wilson PD. The prevalence of female urinary incontinence and reasons for not seeking treatment. N Z Med J 1988;101:756–8.
4. O'Donnell M, Viktrup L, Hunskaar S. The role of general practitioners in the initial management of women with urinary incontinence in France, Germany, Spain and the UK. Eur J Gen Pract 2007;13:20–6.
5. Morrison A, Levy R. Fraction of nursing home admissions attributable to urinary incontinence. Value in Health 2006;9:272–4.
6. Brading AF. A myogenic basis for the overactive bladder. Urology 1997;50(6A Suppl):57–67; discussion 8–73.
7. de Groat WC. A neurologic basis for the overactive bladder. Urology 1997;50(6A Suppl):36–52; discussion 3–6.
8. van der Vaart CH, de Leeuw JR, Roovers JP, Heintz AP. The effect of urinary incontinence and overactive bladder symptoms on quality of life in young women. BJU Int 2002;90:544–9.
9. Coyne KS, Zhou Z, Thompson C, Versi E. The impact on health-related quality of life of stress, urge and mixed urinary incontinence. BJU Int 2003;92:731–5.

10. Artibani W, Cerruto MA. The role of imaging in urinary incontinence. BJU Int 2005;95:699–703.
11. Homma Y. The clinical significance of the urodynamic investigation in incontinence. BJU Int 2002;90:489–97.
12. Subak LL, Whitcomb E, Shen H, Saxton J, Vittinghoff E, Brown JS. Weight loss: a novel and effective treatment for urinary incontinence. J Urol 2005;174:190–5.
13. Bo K, Borgen JS. Prevalence of stress and urge urinary incontinence in elite athletes and controls. Med Sci Sports Exerc 2001;33:1797–802.
14. Swithinbank L, Hashim H, Abrams P. The effect of fluid intake on urinary symptoms in women. J Urol 2005;174:187–9.
15. Bump RC, McClish DM. Cigarette smoking and pure genuine stress incontinence of urine: a comparison of risk factors and determinants between smokers and nonsmokers. Am J Obstet Gynecol 1994;170:579–82.
16. Hannestad YS, Rortveit G, Daltveit AK, Hunskaar S. Are smoking and other lifestyle factors associated with female urinary incontinence? The Norwegian EPINCONT Study. BJOG 2003;110:247–54.
17. Hay-Smith EJ, Bo Berghmans LC, Hendriks HJ, de Bie RA, van Waalwijk van Doorn ES. Pelvic floor muscle training for urinary incontinence in women. Cochrane Database Syst Rev 2001;(1):CD001407.
18. Aksac B, Aki S, Karan A, Yalcin O, Isikoglu M, Eskiyurt N. Biofeedback and pelvic floor exercises for the rehabilitation of urinary stress incontinence. Gynecol Obstet Invest 2003;56:23–7.
19. Wallace SA, Roe B, Williams K, Palmer M. Bladder training for urinary incontinence in adults. Update of Cochrane Database Syst Rev. 2000;(2):CD001308; PMID: 10796768. Cochrane Database Syst Rev 2004;(1):CD001308.
20. Wilson P. Incontinence. Plymbridge Distributors; 1999, p. 579–634.
21. Nabi G, Cody JD, Ellis G, Herbison P, Hay-Smith J. Anticholinergic drugs versus placebo for overactive bladder syndrome in adults. Cochrane Database Syst Rev 2006; Issue 4. Art. No.: CD003781.
22. Alhasso AA, McKinlay J, Patrick K, Stewart L. Anticholinergic drugs versus non-drug active therapies for overactive bladder syndrome in adults. Cochrane Database Syst Rev 2006; Issue 4. Art. No.: CD003193.
23. Hashim H, Abrams P. Pharmacological management of women with mixed urinary incontinence. Drugs 2006;66:591–606.
24. Mariappan P, Alhasso A, Ballantyne Z, Grant A, N'Dow J. Duloxetine, a serotonin and noradrenaline reuptake inhibitor (SNRI) for the treatment of stress urinary incontinence: a systematic review. Eur Urol 2007;51:67–74.
25. Grady D, Brown JS, Vittinghoff E, et al. Postmenopausal hormones and incontinence: the Heart and Estrogen/Progestin Replacement Study. Obstet Gynecol 2001;97:116–20.
26. Abrams P, Cardozo L, Fall M, et al. The standardisation of terminology in lower urinary tract function: report from the standardisation sub-committee of the International Continence Society. Urology 2003;61:37–49.

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