The two functions of the bladder are storing and voiding urine. Dysfunction of either can cause incontinence.

Urgency incontinence is a storage dysfunction most often associated with detrusor overactivity. It can be managed by bladder training and antimuscarinic drugs.

Stress incontinence is a storage dysfunction most often associated with poor ligamentous support of the pelvic floor or sphincter deficiency, or both. Drugs have a very limited role in treatment. Surgery is often needed.

Voiding dysfunction can be caused by bladder outlet obstruction, such as benign prostatic hyperplasia. Drug treatment may include alpha blockers.

Drugs for incontinence only have modest efficacy. They can have adverse effects away from the urinary tract, which may be particularly problematic in older people.

Incontinence from storage dysfunction

Urgency and stress incontinence are the most common symptoms of bladder storage dysfunction.

Bladder factor – urgency incontinence

Apart from urinary tract infection the most common cause of urgency incontinence is the overactive bladder syndrome. This syndrome has urgency as its pivotal symptom, although it may also be associated with frequency and nocturia.

There are many causes of overactive bladder syndrome and they can be divided into neurogenic and non-neurogenic causes. Neurogenic overactive bladder can be associated with spinal cord injury, Parkinson's disease, multiple sclerosis and diabetes.

In non-neurogenic overactive bladder syndrome, urinary tract infection and bladder malignancy must be ruled out along with foreign bodies such as bladder stones. Age-related overactive bladder and idiopathic detrusor overactivity should be considered. Also look for outlet factors such as benign prostatic hypertrophy or atrophic vaginitis.

The initial assessment requires a thorough history and examination. The assessment focuses on the duration, severity and inconvenience of the overactive bladder syndrome, the presence of other lower urinary tract symptoms especially haematuria, as well as past urological, neurological, and obstetric or gynaecological history. Feel for a palpable bladder and examine the prostate or vagina and test for stress incontinence with coughing and for pelvic organ prolapse. Examination of the S2-S4 nerve segment, for example by testing the bulbocavernosal reflex and anal tone, is vital to exclude occult neuropathology.
A midstream urinalysis and culture, and measurement of the post-void residual urine volume is mandatory. A shorter onset of symptoms (for example less than three months), haematuria, or a history of heavy smoking may suggest urinary tract malignancy and will require further tests such as urine cytology, imaging and a referral to a urologist.

Treatment

There is little difference in drug therapy for neurogenic or non-neurogenic overactive bladder syndrome, with the mainstay being antimuscarinic drugs. When there is a low index of suspicion for cancer, empirical drug treatment with an antimuscarinic drug is reasonable. In idiopathic overactive bladder syndrome, the addition of bladder training to antimuscarinics is more effective than either treatment alone. Bladder training involves modifying lifestyle and fluid intake, pelvic floor muscle training, and postponement and distraction techniques. Lifestyle modification includes losing weight, regular exercise and avoiding bladder irritants such as cigarettes and caffeine. A continence nurse specialist and physiotherapist should have the necessary expertise to assist with this training. The Continence Foundation of Australia also provides brochures free of charge to patients and healthcare professionals (www.continence.org.au). Psychogenic factors and their drug treatment may be associated with lower urinary tract symptoms and incontinence. If diagnosed, some patients may respond to cognitive behavioural therapy. There are five subtypes of muscarinic receptors. The detrusor and urothelium contain mainly M2 and M3 receptors. Although M2 receptors account for 80% of the receptors in the urinary tract, M3 receptors are primarily responsible for bladder contraction.

Antimuscarinic drugs can be classified as M3-selective or non-selective. Most of the current drugs produce varying degrees of common antimuscarinic adverse effects such as dry mouth, blurred vision, confusion, constipation and rarely tachycardia. As the drugs have modest efficacy the patient needs to decide if the benefits outweigh these adverse effects. One less micturition may not be relevant to a patient who is having 12 episodes every day.

Non-selective antimuscarinics

Muscarinic receptors are found in gut and salivary glands, so non-selective drugs act on them as well as on the bladder. Oxybutynin

Oxybutynin is available in both oral and transdermal patch formulations. It can cross the blood–brain barrier leading to adverse effects such as dizziness and cognitive dysfunction.

Tolterodine

Tolterodine has greater specificity for bladder receptors than for salivary glands. This has contributed to reports that it causes significantly less dry mouth than oxybutynin. When tolterodine was compared to oxybutynin it had better tolerability, but there was no statistically significant difference in quality of life.

M3-selective antimuscarinics

These drugs mainly act on bladder muscle. Solifenacin

As solifenacin blocks M3 receptors, there is a lower rate of dry mouth and constipation than with non-selective antimuscarinic drugs. An initial dose of 5 mg daily can be increased to 10 mg daily. In clinical trials, solifenacin reduced the daily mean number of voids compared to placebo (2.2 vs 1.2 episodes). The reduction in the number of urgency episodes is 2.8 with 5 mg solifenacin, and 1.4 with placebo, while the urgency incontinence episodes reduced by 1.4 per day compared to 0.5 per day with placebo. While frequency and urgency were less commonly reported at a dose of 10 mg, there was an increased risk of dry mouth at 4–12 weeks. Solifenacin is relatively safe in patients over 65 years old.

Darifenacin

Darifenacin 7.5 mg significantly reduces the number of incontinence episodes per week, with a median reduction of two episodes compared to placebo. The most common adverse events in placebo-controlled 12-week trials were dry mouth and constipation. As darifenacin is metabolised by the liver it is not recommended for patients with severe liver impairment.

Botulinum toxin

Randomised controlled trials show that an injection of botulinum toxin type A into the bladder wall is effective in both drug-refractory non-neurogenic and neurogenic overactive bladder syndrome. The toxin inhibits the release of acetylcholine into the neuromuscular junction, thus dampening detrusor contractility. It reduces the frequency of micturition and the number of episodes of urgency incontinence, and increases functional bladder capacity and quality of life. The main potential adverse effects are temporary urinary retention that may require catheterisation, and urinary tract infection. Botulinum toxin injections are given into the detrusor muscle during cystoscopy. Patients do require
Management of urinary incontinence in adults

repeated injections after a mean of 6–9 months due to loss of efficacy.

Recent approval was granted in the USA for the use of the toxin in both drug-refractory neurogenic and non-neurogenic overactive bladder syndrome. In Australia botulinum toxin A is approved by the Therapeutic Goods Administration for both neurogenic and non-neurogenic overactive bladder, with the neurogenic indication now listed on the Pharmaceutical Benefits Scheme.

Outlet factor – stress incontinence

The most common storage dysfunction related to an outlet factor is stress incontinence. It is due to intra-abdominal pressure exceeding urethral closure pressure, causing involuntary loss of urine. If pelvic floor muscular training fails, the mainstay of treatment is surgery, although lifestyle modifications (for example weight loss) and controlling comorbidities which put chronic strain on the pelvis (for example chronic obstructive pulmonary disease) may have a supportive role.

Effective surgical options for stress incontinence include the synthetic mid-urethral sling and autologous fascial slings in women, and the transobturator bulbo-urethral sling in men. Implantation of an artificial urinary sphincter can be tried if sling surgery fails.

The role for drug therapy in stress incontinence is very limited. Duloxetine, which is a serotonin and noradrenaline reuptake inhibitor, has some effects on increasing bladder outlet resistance. It has been effective in controlling mild urinary stress incontinence in women, but it is not approved for this indication in Australia.

Incontinence from voiding dysfunction

The detrusor muscle is relaxed when urine is stored. It contracts to overcome the resistance of the bladder outlet during voiding.

Overflow incontinence

Leakage of urine can be associated with urinary retention. This may be caused by poor detrusor contractility or be secondary to chronic bladder outlet obstruction.

Incontinence associated with benign prostatic hyperplasia (outlet factor)

Chronic bladder outlet obstruction leads to functional changes, such as decreased bladder compliance and detrusor overactivity. In turn, this may result in frequency, urgency and urgency incontinence. Detrusor overactivity, mediated by M2 and M3 muscarinic receptors, contributes to lower urinary tract symptoms in approximately 15% of men.

In patients with overactive bladder syndrome secondary to bladder outlet obstruction, treatment varies from watchful waiting to drug therapy and various surgical options depending on the severity of symptoms and indications for intervention. The mainstay of drug treatment includes alpha adrenergic receptor blockers and 5-alpha-reductase inhibitors. If the overactive bladder syndrome is secondary to bladder outlet obstruction there may be a role for combinations of these drugs. Although there is a risk of acute urinary retention with alpha adrenergic receptor blockers and antimuscarinic drugs in combination, the rate is low. With 5-alpha-reductase inhibitors, common adverse effects include fatigue, loss of libido and ejaculatory and/or erectile dysfunction. Long-term use at the end of four years shows an absolute reduction in the overall risk of developing prostate cancer. The alpha blockers can cause hypotension. As the elderly are more susceptible to orthostatic hypotension, they may have an increased risk of falls.

Incontinence in the elderly

Using drugs to manage incontinence in the elderly follows the basic prescribing principle of ‘start low, go slow’. As well as the dose, polypharmacy and coexisting medical comorbidities must be considered.

Antimuscarinics such as oxybutynin and tolterodine are the mainstay for bladder overactivity, but should be used with care. The risk of urinary retention is a concern, as the ageing bladder is often associated with impaired emptying. This necessitates a slow escalation of the dose, frequent review of the response and monitoring of urine output and post-void residual volume.

Newer drugs such as darifenacin and solifenacin are more M3-selective and cross the blood–brain barrier less readily than non-selective drugs. There is some evidence that non-selective drugs are more likely to cause cognitive dysfunction. The most recent randomised trial (2013 SENIOR trial) reports that both oxybutynin and solifenacin are well-tolerated in the elderly, but oxybutynin is associated with a reduction in attention compared to placebo.

Detrusor hyperactivity with impaired contractility is a common but lesser known cause of incontinence in older people. Detrusor dysfunction results in both storage and the emptying dysfunction. The clinical diagnosis is difficult and the management of this condition may require urological referral and urodynamic evaluation. Combination therapy with an anticholinergic and alpha, blocker can be efficacious by targeting both components of detrusor hyperactivity and impaired contractility.
Conclusion

Understanding the biphasic storage and voiding functions of the bladder helps diagnose and treat overactive bladder with or without incontinence. The mainstay of overactive bladder management is pharmacological and is evolving. Newer treatments such as intravesical botulinum toxin injections are being used in neurogenic and non-neurogenic overactive bladders. Treatment is best delivered by a multidisciplinary approach via medical, nursing and physiotherapy personnel.

Vincent Tse is a consultant for Astellas.

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REFERENCES


FURTHER READING


Patient Support Organisation

Continence Foundation of Australia

The Continence Foundation promotes bladder and bowel health and the management of incontinence. It provides support and information about incontinence products and treatment for men, women and children. Its website has links to:

• resources to help manage incontinence, such as pelvic floor safe exercises, information on continence aids and financial assistance, bladder/bowel diaries, surgical options, the roles of health professionals including nurses and physiotherapists, a national public toilets map, leaflets designed specifically for indigenous people, and other information for carers and health professionals
• an online support forum for sharing information and ideas
• Bridge magazine, free for consumers
• the Australian Continence Exchange resources for health professionals at www.continenceexchange.org.au

• the Continence Aids Payment Scheme (CAPS), a government payment to help eligible people with permanent and severe incontinence purchase incontinence products from suppliers.

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