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Urinary incontinence in women

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Abstract

Urinary incontinence symptoms are highly prevalent among women, have a substantial effect on health-related quality of life and are associated with considerable personal and societal expenditure. Two main types are described: stress urinary incontinence, in which urine leaks in association with physical exertion, and urgency urinary incontinence, in which urine leaks in association with a sudden compelling desire to void. Women who experience both symptoms are considered as having mixed urinary incontinence. Research has revealed overlapping potential causes of incontinence, including dysfunction of the detrusor muscle or muscles of the pelvic floor, dysfunction of the neural controls of storage and voiding, and perturbation of the local environment within the bladder. A full diagnostic evaluation of urinary incontinence requires a medical history, physical examination, urinalysis, assessment of quality of life and, when initial treatments fail, invasive urodynamics. Interventions can include non-surgical options (such as lifestyle modifications, pelvic floor muscle training and drugs) and surgical options to support the urethra or increase bladder capacity. Future directions in research may increasingly target primary prevention through understanding of environmental and genetic risks for incontinence.

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Urinary incontinence is the complaint of involuntary loss (leakage) of urine[1]. The condition occurs in both sexes, but is much more frequent in women. Although some overlap in pathophysiology is evident between sexes, incontinence in men is often a consequence of prostatic enlargement or from damage to continence mechanisms during surgery or radiotherapy for prostate cancer. By contrast, incontinence in women is typically related to dysfunction of the bladder or pelvic floor muscles, with such dysfunction often arising during pregnancy or childbirth, or at the time of menopause. This Primer focuses on female urinary incontinence because of its higher prevalence and unique pathophysiology.

There are two main subtypes of urinary incontinence: stress incontinence and urgency incontinence. According to the International Urogynecological Association (IUGA) and the International Continence Society (ICS) standard definition, stress incontinence is the complaint of urine leakage in association with coughing, sneezing or physical exertion, whereas urgency incontinence is the complaint of urine leakage associated with a sudden compelling desire to void that is difficult to defer[1]. These two subtypes are so common that they often coexist, as a combination of symptoms termed mixed incontinence. Most women with urgency incontinence also receive a diagnosis of overactive bladder syndrome (BOX 1), of which urgency incontinence forms one possible component.

Rarer subtypes of incontinence in women include postural incontinence, which is the loss of urine with a change of body position (often when standing up or bending over); nocturnal enuresis, which is the leakage of urine during sleep; continuous incontinence, of which the common causes include vesical fistulae; and coital incontinence, the loss of urine during sexual intercourse[1]. The term ‘functional incontinence’ can be used to refer to incontinence in the setting of physical or cognitive impairment, such as hip fracture or dementia, that limits mobility or the ability to process information about bladder fullness[2].

Incontinence symptoms are highly prevalent, have a substantial impact on health-related quality of life[3] and are associated with huge personal[4] and societal[5,6] expenditure. All types of incontinence are more common with age and obesity[7–10], and so the public health burden of these conditions is likely to increase with current demographic trends. The burden on individuals and populations of these conditions[11] is quite disproportionate to the attention they receive in the press, or the levels at which incontinence research is funded. Policy makers, medical professionals and the general public are largely unaware that urinary incontinence is a disease[12], despite its International Classification of Diseases (ICD) classification[13]. Part of this lack of awareness is rooted in the widespread misperception that incontinence represents a normal part of ageing, or is a natural consequence of childbirth[14]. Furthermore, women often delay or entirely defer presentation to their health care practitioners[15]; even among women who receive a diagnosis, only a minority receive effective therapy[16,17].

This Primer summarizes the current state of understanding of urinary incontinence in women, with a focus, in particular, on stress incontinence and urgency incontinence. Both these areas have witnessed considerable innovations in practice over the past decade.

Epidemiology

Urinary incontinence is considered a stigmatizing condition in most populations[18], which contributes to low rates of presentation for care and creates a high risk for respondent bias in observational studies[19,20]. The best prevalence estimates, therefore, come from general health surveys not focused on incontinence among representative samples using validated, symptom-based questionnaires[21].

Such robust prevalence studies, using validated measures, exist for the United States and many developed European and Asian countries; population-level prevalence data for developing countries are less readily available. However, most early epidemiological studies did not differentiate between stress and urgency urinary incontinence. The range of reported prevalence for urinary incontinence of any subtype in adult women is broad (5–72%), with studies converging on a prevalence of approximately 30%[22–27]. This enormous variation between studies is observed both within and between countries. If true prevalence rates vary between countries, it is obscured by cultural differences in the perception of urinary incontinence and willingness to report urinary incontinence, as well as methodological differences[28], including the wording of questionnaire items, the method of administration of questionnaires and — perhaps most importantly — the differences in case definitions used[25,29]. These differences in case definitions relate to the time period over which symptoms are ascertained and whether symptom frequency, severity and bother are assessed. Indeed, studies specifically measuring severe urinary incontinence, defined as urine leakage several times per week, have a more consistent reported prevalence of 6–10% in Europe and the United States[26,30].

The association of urinary incontinence with age is well characterized. Across all available studies, the age-specific incidence is <2 per 1,000 person-years in women <40 years of age, but it increases with age[31]. For example, the EPINCONT study, a longitudinal study of women in Norway surveyed in 1995–1997 and again in 2006–2008, showed a 16% increase in the prevalence of urinary incontinence between the two time periods, with an incidence rate of 18.7% and a remission rate of 34.1%[26]. For the population as a whole, stress incontinence is more common than either urgency or mixed incontinence in most studies[22–24,26]. However, the prevalence of stress incontinence peaks in the fifth decade of life, and thereafter the prevalence of mixed and urgency incontinence continues to increase (FIG. 1). Studies project that the prevalence of urinary incontinence and other pelvic floor disorders, such as pelvic organ prolapse and faecal incontinence, will increase as the global population ages[32]. It is estimated that the number of women in the United States with urinary incontinence will rise from 18.3 million in 2010 to 28.4 million in 2050 (REF 32).

Data regarding the association of urinary incontinence with ethnicity are conflicting. Some studies have shown a higher prevalence of urinary incontinence in white women, whereas others have shown similar prevalence across ethnicities[19,25,33–35]. In several US studies, stress urinary incontinence is more common in white women than in African-American or Asian- American women[19,34,35]. Other factors associated with urinary incontinence in multiple, large, population-based studies[25,36–38] include parity[25,26,36], obesity[25,26,36,38],

previous hysterectomy or pelvic surgery[25,36,38], pulmonary disease[36,38], diabetes mellitus[22,25,26,33,37,38] and nursing home admission or dementia[1,3,4].

Mechanisms/pathophysiology

Bladder structure and function

The bladder, urethra and urinary sphincters work in concert to store urine at low pressure and to void voluntarily at socially convenient or appropriate times. The detrusor muscle and internal urethral sphincter are predominantly smooth muscle, whereas the external urethral sphincter and pelvic floor muscles are predominantly striated muscle. The bladder lumen is lined with epithelial cells (called urothelium) and the basement membrane (mucosal layer) that protect the underlying detrusor muscle from toxins contained in the urine and enable communication with neural cells that coordinate storage and voiding phases (FIG. 2).

The sympathetic nervous system predominates during the storage phase and maintains continence through the paravertebral ganglia, the hypogastric nerves and hypogastric plexus. The parasympathetic system coordinates the voiding phase, through the sacral plexus and pelvic nerves (S2–S4)[39]. Afferent signals from the urothelium and bladder wall are transmitted through to the thalamus; the balance between storage and voiding is maintained by the central pontine micturition centre (FIG. 3). The neurotransmitters responsible for execution of these commands are acetylcholine and noradrenaline.

Voiding up to 7 times per day in the waking hours is considered normal[1], with a micturition volume of 250–300 ml per void (although the volume is typically higher with the first morning void). A healthy adult bladder has a limit of comfortable tolerance of approximately 500 ml, and can accommodate this relatively large volume of urine with little, if any, increase in intravesical pressure, owing to the viscoelastic compliance of the bladder. Although the anatomy of the bladder, urethra and urinary sphincters are well understood, the physiology underlying incontinence — particularly urgency incontinence — remains surprisingly controversial. Several factors have been implicated in the mechanisms underlying stress and urgency incontinence. These include damage to the endopelvic fascia and pelvic floor muscles that support the urethra, decreased function of the striated urinary sphincter, changes in the compliance and innervation of the detrusor muscle, changes in the urothelium, changes in urine composition and changes in the central nervous system.

Stress urinary incontinence

Two common, often overlapping, mechanisms for stress urinary incontinence have been described: urethral hypermobility resulting from loss of support of the bladder neck and urethra (such that they move during peaks of abdominal pressure), and weakness of the urinary sphincter itself. If the urinary sphincter mechanism is damaged, a specific subtype of stress urinary incontinence ensues, sometimes referred to as intrinsic sphincter deficiency. Weakness of the urinary sphincter can result from trauma, repeated urogynaecological surgeries, neurological disease, ageing or diseases leading to systemic muscular atrophy. Although all contemporary treatments are used for both subtypes of stress urinary

incontinence, in general, treatments are more successful for patients with some degree of urethral hypermobility than for isolated weakness of the urinary sphincter[40].

The hammock hypothesis is widely accepted as the pathophysiological explanation of stress urinary incontinence associated with urethral hypermobility[41]. The hypothesis states that the urethra is supported by the endopelvic fascia, which is the fibromuscular connective tissue ofthe vagina (FIG. 4). The endopelvic fascia creates a 'hammock' against which the urethra is compressed during rest and activity. This compression, combined with 'intrinsic' urethral sphincter pressure and mucosal coaptation, effectively closes the urethral lumen and prevents the involuntary loss of urine even when the intravesical pressure increases. Damage to the arcus tendineus fasciae pelvis or paravaginal tissue as a result of excess loading from obesity, chronic cough, constipation, parturition or menopause can decrease the anatomical support of the bladder neck and the urethra. This loss of support results in hypermobility of the urethra, such that rather than being compressed at times of increased intra-abdominal pressure, the urethra moves downward without being compressed — resulting in lower pressure in the urethra than in the bladder, with consequent urine leakage.

Urgency urinary incontinence

Unlike the physical changes associated with stress urinary incontinence, urgency urinary incontinence involves physiological perturbations to bladder function. There are three main aetiologies intrinsic to the bladder that lead to urgency incontinence: detrusor overactivity, poor detrusor compliance and bladder hypersensitivity.

Detrusor overactivity—Most current drugs for urgency incontinence block muscarinic receptors that mediate contraction within the detrusor muscle, or stimulate β3 receptors that promote relaxation. Many patients with urgency incontinence have measurable spontaneous uninhibited contractions of the detrusor muscle during bladder filling, called detrusor overactivity. These contractions can coincide with perceived urinary urgency, and urgency incontinence can result if the pressure generated overcomes the resistance of the urinary sphincter.

Early hypotheses of the pathophysiology of detrusor overactivity were anatomically centred on the spine and parasympathetic motor supply to the bladder — the so-called neurogenic hypothesis[42]. Detrusor overactivity is common after spinal injuries, with substantial spinal disease from multiple sclerosis or other lesions of the central nervous system. However, in the general population, neurogenic causes of detrusor overactivity are uncommonly identified[42]; that is, detrusor overactivity in most women is labelled as 'idiopathic' without finding a single pathophysiological cause.

Much research in the early 2000s focused instead on the detrusor muscle as the origin of detrusor overactivity — the so-called myogenic hypothesis. This hypothesis is based on the recognition that both strips of bladder muscle and individual detrusor cells from patients with detrusor overactivity show heightened contractile responses *in vitro.* Both myogenic and neurogenic mechanisms can coexist, so detrusor overactivity can be viewed as a multifactorial condition, akin to irritable bowel syndrome[43]. Although detrusor overactivity has been a major focus for research, it is only observed, at the time of urodynamics, in just

over half of women presenting with urgency incontinence, but may occur in at least half of elderly asymptomatic adults[44].

Poor detrusor compliance—In low compliance, the bladder fails to stretch, which causes increased pressure, discomfort during filling and a limited capacity. This pattern is typical after pelvic radiotherapy, or can result from prolonged periods of catheterization.

Bladder hypersensitivity—Afferent sensory information from the bladder has recently been established as a key factor in overactive bladder syndrome and, accordingly, attention has focused on the sensory role of the urothelium in this condition (FIG. 5). The urothelium is not merely a barrier, but is a responsive structure that is able to detect thermal, mechanical and chemical stimuli. Indeed, this is observed in both rodent and porcine models, with the mucosal layer of the bladder augmenting detrusor function, either through the release of various neurotransmitters or through its own spontaneous electrical activity[45,46].

With the recognition of the urothelium as a pivotal mediator of bladder function, much attention has been concentrated on the role of urothelial inflammation and infection in the aetiology of overactive bladder syndrome. The balance of urinary microbiota[47] is now believed to substantially alter bladder sensation and, perhaps, function. Historically, the bladder and urine have been considered as sterile, and urinary tract infection (UTI) in women was thought to result from ascending spread of uropathogenic organisms colonizing the vagina. However, the introduction of expanded quantitative urine culture and 16S rRNA gene sequencing for bacterial identification has transformed our understanding of the microbial ecology of the bladder. Sequencing of urine samples collected using specific techniques to avoid vaginal contaminants, from patients and healthy volunteers, has revealed a wide range of colonizing organisms that might be implicated in lower urinary tract dysfunction[48]. Evidence of exacerbation of lower urinary tract symptoms (LUTS) in association with both planktonic bacteria in the urine and urothelial colonization by bacteria has been reported[49–51], but it remains unclear which species of bacteria may be responsible.

The vaginal microbiota can be classified into five community-state types, of which four are dominated by different *Lactobacillus* species (*L. iners, L. crispatus, L. gasseri* and *L. jensenii*), whereas the fifth community is more diverse[52]. Most urinary bacterial communities are also dominated by lactobacilli[53]. Recent work exploring the urinary microbiota have identified bacteria that would be considered as either commensal or pathogenic at other body sites[54]. As our understanding of the urinary microbiota increases, we should expect to be able to differentiate common commensal organisms from bacterial colonizers that are likely to have a negative effect on urothelial function, and potentially lead to bladder hypersensitivity.

Diagnosis, screening and prevention

The presenting symptom of urinary incontinence is, by itself, not necessarily diagnostic of the subtype of urinary incontinence or its underlying cause. A comprehensive assessment is needed to determine the exacerbating factors, the effect on the woman's quality of life and her desire for treatment[55]. The typical diagnostic work-up involves medical history, physical

examination, urinalysis (see below), assessment of post-void residual volume and exclusion of conditions that require specialist referral (FIG. 6). After these assessments, a provisional diagnosis of stress, urgency or mixed urinary incontinence can be made in the majority of patients[56]. Most women present with a degree of mixed urinary incontinence, and establishing the predominant symptom can assist in directing the appropriate treatment. Depending on the severity of the individual components of their overall incontinence, patients often benefit from initial treatment that is focused on urgency urinary incontinence.

Symptoms and risk factors

A comprehensive patient history includes the onset, duration and timing of urinary incontinence, and associated LUTS and voiding symptoms (BOX 2), recognizing that the reported symptoms often relate to the patient's normal bladder function and expectations. Other risk factors or conditions that can exacerbate urinary incontinence should also be assessed and include age, obstetric history (parity and mode of delivery), gynaecological status (the presence of pelvic organ prolapse, defecatory dysfunction or anal incontinence, sexual dysfunction and urogenital syndrome of menopause), medical status (the presence of a UTI, dementia, delirium, diabetes mellitus or diabetes insipidus, cardiorespiratory disorders, chronic cough, obesity and obstructive sleep apnoea) and pharmacological status (the use of hormonal replacement therapy, α-adrenergic agonists and antagonists, calcium channel blockers, diuretics, lithium therapies, opioid analgesics, anticholinergics and angiotensin-converting enzyme inhibitors; BOX 3)[57]. Patients with mild cognitive impairment are 30% more likely to have urinary incontinence[58]. In addition, functional and lifestyle factors, such as smoking status, mobility and frequency of heavy lifting, should be considered during assessment.

Some patients can present with a history that suggests alternative lower urinary tract pathologies that also cause urinary incontinence, but require specialist referral. These symptoms include recurrent UTI; sterile pyuria (leukocytes in the urine); concurrent pelvic organ prolapse, bladder pain, haematuria and continuous leakage suggestive of fistula; urinary retention or obstruction; or neurological symptoms. Similarly, a history of urogynaecological malignancy, pelvic irradiation, urogenital tract abnormalities or surgery should prompt a specialist referral[59].

In a patient who is frail, or those >65 years of age, a targeted history of reversible causes of urinary incontinence includes an assessment of delirium, infection, pharmaceuticals, psychological morbidity, excess fluid intake, restricted mobility and stool impaction (the DIPPERS assessment[60]) (FIG. 6).

Physical examination

Physical examination should include a functional assessment observing the mental status and mobility as well as body mass index of the patient. Abdominal examination should assess for pelvic masses, a palpable bladder and costovertebral angle tenderness. The urogenital examination might reveal vaginal atrophy and incontinence-associated dermatitis (that is, damage to the skin with exposure to urine).

A positive cough stress test (whereby observed urethral leakage is provoked by a series of forceful coughs in the supine or standing position with a comfortably full (~300 ml) bladder volume) has a high sensitivity and specificity for diagnosing stress urinary incontinence[61]. After emptying the bladder, a positive supine empty stress test can be suggestive of intrinsic sphincter deficiency[62], but invasive urodynamic studies are required to confirm the diagnosis[63]. The findings should be related back to the patient's predominant reported type of urinary incontinence to ensure prioritization of management for the symptom of most bother.

Evidence of pelvic organ prolapse should be observed during a Valsalva manoeuvre (that is, forced attempted exhalation against a closed airway) over ≥6 seconds[64] in whichever position maximum protrusion is best demonstrated (supine, left lateral or standing). The validated staging of pelvic organ prolapse is described by Pelvic Organ Prolapse Quantification (POP-Q)[65], but a simplified description (S-POP-Q) has also been validated for use in clinical practice[66], in which staging is based on assessment after the bladder is emptied. This staging system only defines anatomical descent and does not define the normal range, with up to 50% of women having stage 2 descent. Only a weak-to-moderate correlation between anatomical descent and urinary symptoms has been shown, with common prolapse symptoms being a vaginal bulge, sensation of heaviness or difficulty in voiding[67].

For women with stage 2-4 pelvic organ prolapse, the anatomical distortion may kink the urethra, sometimes resulting in a false-negative cough stress test. Thus, reducing the prolapse digitally without distorting the bladder neck while performing a cough stress test might be of value. However, limited evidence has been reported on how to optimally reduce urethral kinking for the test[68].

The relationship between urethral mobility and stress urinary incontinence has been assessed using a range of techniques, including the POP-Q point Aa descent[69], urethral Q-tip[70], vaginal cotton swab[71], visual evaluation[72], ultrasonography[72] and lateral chain urethrocystography[73]. However, none of these is recommended and there is no consensus definition for clinically relevant urethral hypermobility. The advantage of hypermobility assessment lies in finding the absence of urethral descent, which is associated with a twofold higher rate of surgical failure[74].

A digital examination palpating the pelvic floor for muscle tone, contraction technique and strength is advised. Standardized terminology has been developed to describe pelvic floor function, but no standardized method or normative value for measurement or classification is available[75]. Other abnormalities such as urethral diverticulae (out-pouching of the urethral mucosa, which creates a midline anterior wall cystic mass) and pelvic masses can also be identified when assessing for pelvic organ prolapse.

Finally, a speculum examination can assist in assessing each vaginal compartment as well as assessing for any extra-urethral loss of urine that may suggest a fistula. Any bowel dysfunction or neurological symptoms should prompt a rectal examination assessing for tone

and sphincter squeeze. In addition, bowel dysfunction should prompt a general neurological examination, including testing of the S2-S4 nerve distribution[63].

Urological tests

Diagnosis might also require objective and semi-objective measures, such as voiding diaries, pad tests, urinalysis and urodynamic tests.

Urinalysis—Urinalysis using a colorimetric reagent test strip evaluates urine for a range of chemical parameters including pH, protein, glucose, ketones, occult blood, bilirubin, urobilinogen, nitrite, leukocyte esterase and specific gravity. Urinalysis is recommended as a screening tool for UTIs and other associated conditions in the assessment of urinary incontinence[76]. Indeed, the criteria for diagnosing overactive bladder syndrome and detrusor overactivity require the exclusion of UTIs. Urine reagent dipsticks have a low sensitivity and high specificity for the exclusion and identification of UTIs[77,78], respectively. Formal microscopy, culture and antibiotic sensitivity (MCS) analyses of a clean-catch urine specimen or catheter urine specimen are recommended[79] in women who have symptoms of UTI or have a positive dipstick[63].

The microbiological criteria for UTI are defined as bacteriuria of >100,000 colony-forming units (CFU) per ml on voided specimen or >1,000 CFU per ml on catheterized specimen with pyuria (defined as >10 white blood cells per ml). This diagnostic threshold is important given the association between low-CFU bacteriuria count (<1,000 CFU per ml), urinary incontinence[50] and refractory LUTS. In those with a history of recurrent UTIs, requesting the results of the previous microscopy, culture and antibiotic sensitivity analyses can assist in confirming the diagnosis, determining the correct treatment based on sensitivities and determining whether the UTIs are recurrent or persistent[1]. In addition to screening for infection, urine dipsticks detect the presence of blood, glucose and protein, and have a role in screening for malignancy, diabetes mellitus and renal impairment.

Elderly patients have a high prevalence of asymptomatic bacteriuria. Thus, care should be taken in interpreting urinalysis findings in relation to the symptoms to avoid overuse of antibiotics that might be of little benefit or even harm[80]. The revised McGreer criteria for diagnosing UTIs are recommended for use in this population; these criteria require both acute localizing genitourinary signs and symptoms and a positive urine culture for the diagnosis of symptomatic UTI in individuals without indwelling catheters[81].

Post-void residual volume assessment—The post-void residual (PVR) volume is determined by measuring the volume remaining in the bladder immediately after voiding; it is a measure of the completeness of bladder emptying. Thus, it is recommended when evaluating those with voiding symptoms, symptomatic pelvic organ prolapse or palpable bladder overdistension[82]. Measuring the PVR volume can be achieved using ultrasonography, with either a bladder scanner or formal renal tract imaging, or an in–out catheter. The technique has acceptable sensitivity and specificity and, importantly, greater patient acceptability and lower adverse outcomes than the gold standard of catheterization[76,83]. An increased PVR volume when assessed immediately post-void is defined as >30 ml, but may be up to 100 ml if assessed 10 minutes post-void and depends on

the method of measurement[1]. However, there is wide variation in what clinicians consider to be a clinically significant PVR volume. In those with a PVR volume of ≥100 ml, there may be benefit in repeating the measurement in the event of a false-positive diagnosis of increased PVR volume[84].

Although large and persistent PVR volumes might be related to UTIs, there is little evidence for the effect of increased PVR volume on urinary incontinence and storage symptoms. However, evidence of voiding dysfunction is still important to determine because it can alter the choice of urinary incontinence treatment options, as some treatments can further impair voiding (for example, intradetrusor onabotulinumtoxinA (commonly known as Botox (Allergan)) and surgical procedures for stress urinary incontinence).

Voiding diaries—As an objective measure of mean voided volume, frequency and urinary incontinence frequency, 3- to 7-day voiding diaries are widely used as a reliable tool that is sensitive to small changes[83]. Three types of urinary diary can be used: a micturition chart to record the timing of each void, a frequency–volume diary to record the volume voided with the time and a bladder diary to record additional information on urinary incontinence episodes, pad usage, fluid intake, fluid type and sensation of urgency[1]. In addition, the 24-hour urine production can provide an assessment of daytime and nocturnal polyuria, which may be particularly useful in older women with nocturia (BOX 1). Some women might be unable to complete the bladder diary for functional reasons, such as cognitive impairment. The value of the diary may also be limited in those with either very severe or unconscious urinary incontinence, whereby leakage can be difficult to accurately record. Under these circumstances, other methods, such as pad tests, are of more value.

Pad testing—Pad testing uses an absorbent perineal pad worn by the patient to detect the presence of urinary incontinence and to measure the volume lost, and can be a useful correlate with symptoms[83]. The pad is worn for up to 24 hours while conducting a range of normal activities designed to replicate the usual provocations of urinary incontinence, such as walking and exercising[1]. Some increase in pad weight can occur through perspiration; accordingly, a positive pad test is defined as a weight increase of >1 g over a 1 -hour test or >4 g for a 24-hour test. These thresholds are also recommended as an objective measure of treatment outcome[76].

Pelvic floor imaging—Ultrasonography and other radiological modalities have been used to investigate urinary incontinence by visualizing the morphology, movement and function of structures, including the levator ani, pelvic organs, bladder, bladder neck, urethral sphincter and urethra[85,86]. Although abnormalities observed can be associated with urinary incontinence, such imaging is not predictive or diagnostic of the cause of the incontinence[87,88]. Ultrasonography might have a role in confirming the findings of the patient history and clinical examination, assessing postoperative complications[89] or as a means of providing biofeedback to help women to identify effective pelvic floor contraction during pelvic floor muscle training (PFMT)[90].

Urodynamic studies—Urodynamic studies constitute a series of investigations assessing lower urinary tract function that include uroflowmetry, voiding cystometry, filling

cystometry, urethral function and provocative manoeuvres to demonstrate urinary incontinence (FIG. 7). Auxiliary tests using fluoroscopy or ambulatory equipment might also be of value[91]. Depending on the clinical or research context, such studies can provide a range of information that includes identification of factors that contribute to LUTS and their relative importance, assessment of other aspects of lower urinary tract function, prediction of the consequences of lower urinary tract dysfunction on the upper tracts (particularly in patients with neurogenic causes of incontinence), prediction of the consequences and outcomes of treatment, investigation of reasons for treatment failure and investigation of the effects of interventions[55].

When used in clinical practice, the urodynamic studies should be of suitable technical quality[92] and the process should replicate the patient's symptoms to provide results relevant to the clinical problem[83]. To achieve the quality required, the studies should be conducted in a standardized manner with parameters reported according to published reference ranges[92]. In the research setting, standardization enables comparison of study cohorts and outcomes. In the clinical setting, standardization enables the clinician to provide evidence-based recommendations relevant to the patient's clinical problem.

Conducting a urodynamic study is associated with risks that include procedure-related discomfort and post-procedure transient dysuria (painful urination; 47% of patients), haematuria (14%), bacteriuria (12%) and symptomatic UTI (28%), with no significant reduction in UTI when prophylactic antibiotics are given[93]. For those classified as having overactive bladder syndrome, only around half demonstrate detrusor overactivity, and only one-quarter of those with uninhibited detrusor contractions have overactive bladder symptoms[94]. Accordingly, urodynamic studies are not recommended before non-surgical management of uncomplicated stress, urgency or mixed urinary incontinence[76,82,83]. Similarly, in patients with symptomatically predominant, demonstrable stress urinary incontinence, no pelvic organ prolapse and a PVR volume of <150 ml, treatment outcomes are not improved in those undergoing urodynamic studies before primary surgical treatment[95], even when the urodynamic results are discordant with the symptoms of the patient[96].

Given these limitations, urodynamic studies are of value if the incontinence diagnosis remains uncertain after the initial assessment, when symptoms do not correlate with physical findings or after failed previous treatment[55,82]. Similarly, urodynamic studies should be performed in patients who are considered for invasive, morbid or irreversible overactive bladder or detrusor overactivity treatments, and in those with neurogenic or obstructive voiding conditions. In those with urinary incontinence and an associated neurological condition (for example, neurogenic urinary incontinence), the addition of radiological imaging at the time of invasive urodynamics for the detection of urinary tract anomalies and vesico-ureteric reflux is regarded as the gold-standard investigation[97]. Other specialist uro-neurophysiological tests include electromyography to assess muscular responses to bladder stimuli, pudendal nerve conduction testing and reflex latency measurements[98].

Screening and prevention

Several populations might benefit from proactive screening for symptoms of urinary incontinence, including older women who are frail, for whom there is a high prevalence of urinary incontinence and related morbidity[55], including falls and fractures. Other highrisk groups include pregnant patients, for whom urinary incontinence preventive measures such as fluid management, PFMT and bladder retraining can be offered. Women presenting with other pelvic floor disorders (such as pelvic organ prolapse) might also benefit from screening, as the prevalence of urinary incontinence in this population is >60%[98].

Management

The management of urinary incontinence in adult women is an iterative process. For some affected women, urinary incontinence causes sufficient bother and intrusion to warrant consideration of treatment. The options range from lifestyle modification to more-invasive surgical interventions[99]. Otherwise healthy women might prioritize resolution of their urinary incontinence by actively engaging in pelvic floor rehabilitation, lifestyle changes (including fluid optimization), pharmacological treatment or surgery to resolve persistent symptoms. Alternatively, women with other serious health conditions might consider their urinary incontinence as a chronic condition, with the emphasis on decreasing symptom impact rather than complete resolution. This spectrum of engagement in treatment of urinary incontinence changes over a woman's lifetime and reflects her changing health priorities and preferences. Thus, the goals might vary between the ideal of independent continence with minimal symptoms and the compromise of dependent continence (in which the patient remains dry through regular toileting) or contained incontinence (in which the patient remains dry through the use of aids)[100]. Goals and preferences are likely to change with time and should be periodically reconsidered[101,102].

Role of the multidisciplinary team

Multidisciplinary care for women with urinary incontinence is regarded as important[76]. Such disciplines include physiotherapy, specialist nursing, occupational therapy, gynaecology, urology and geriatrics, with each providing a different service within their scope of practice. Although little evidence supports the comparative effectiveness of any discipline in particular, the data on outcomes of patients treated with multidisciplinary expertise are promising[103].

Non-surgical interventions

Women with mixed urinary incontinence often experience symptom reduction as they embark on behavioural changes that may include weight loss, timed voiding or bladder retraining and fluid optimization[104]. These interventions can be initiated without exhaustive diagnostic work-up, unless signs or symptoms suggest a need for specialist referral. Women with urinary incontinence vary in their willingness and ability to engage in these first-line interventions; however, for women who are able to work closely with their clinical team, significant improvements in symptoms can be achieved[105]. Patients with mixed urinary incontinence benefit from interventions that are designed to optimize fluid intake, medication use and pelvic muscle function[106].

Fluid optimization—Important clues to potential management strategies are often detected during evaluation and the initial diagnosis. For example, if a voiding diary reveals a fluid intake pattern that exacerbates symptoms — such as excessive total fluid intake, large fluid intake before sleep and a pattern of infrequent but large fluid bolus intake — changes might be warranted. Similarly, the intake of caffeinated, alcoholic and carbonated drinks can be temporarily modified to determine whether incontinence symptoms improve. Some medications (BOX 3) are associated with an increase in incontinence symptoms; in such cases, mindful adjustments to fluid intake and optimizing medication timing can facilitate symptom reduction and decrease withdrawal from social interactions.

Weight loss—Weight reduction in women who are overweight or obese can substantially improve symptoms and associated bother[107,108]. Clinicians and patients alike are aware of the challenge this poses for most individuals who are overweight. However, one study showed that, for those enrolled in a weight loss programme and losing at least 3–5% of their baseline weight, a 47% reduction in stress incontinence episodes[109] was achieved, with concomitant reduction in urge incontinence episodes. Similarly dramatic improvements have been demonstrated with bariatric surgery and other weight loss modalities[110].

PFMT—PFMT aims to improve pelvic floor muscle function. The strongest evidence of benefit is for supervised PFMT in women with stress incontinence, with less efficacy in those with urgency incontinence[111]. Emerging evidence supports unsupervised delivery of PFMT[112], which could be cost-effectively delivered through e-training[113]. Women are taught to consciously contract their pelvic floor muscles before and during any increase in abdominal pressure, such as coughing, to avert leakage, and simultaneously to build up the support of the pelvic floor through regular muscle strength training[114]. These exercises are often supplemented by bladder retraining advice (systematically increasing voiding interval), techniques to avert urinary urgency[115] and to avoid imminent stress-induced leakage[116]. In those who are unable to contract the pelvic floor, biofeedback techniques might be useful[90]. Although short-term efficacy is good, with no harmful effects, evidence of long-term benefit is lacking[117–119].

Incontinence pessaries and intravaginal devices—Women seeking further treatment for stress urinary incontinence who wish to avoid or defer surgery, and are unable to adhere to behavioural therapy, can use vaginal continence pessaries[120], which aim to compress the urethra. These treatments show the greatest benefit in those with severe stress urinary incontinence[121]. New options are becoming available, including tampon-like devices licensed for over-the-counter sale, but the effectiveness of these devices is yet to be established.

Continence aids and products—Counselling and guidance about the appropriate use of incontinence aids and pads are important for enhancing quality of life and reducing the stigma of incontinence. The range of products includes mobility aids, accessible commodes and containment products, such as absorbent pads or catheters. When choosing the optimal product, many things must be considered. Nurse continence advisers are best placed to counsel patients and provide access to these products, although independent web-based

resources (such as https://www.continenceproductadvisor.org/) are also available for patients and health care providers.

Medical interventions

Should first-line lifestyle, behavioural and physical therapies be ineffective in treating urinary incontinence, a range of pharmacological agents are available, depending on the specific incontinence symptoms. As for any medication, the woman's coexisting conditions, use of other existing medication and risk of adverse effects should be considered before prescribing. For many of these medications, the benefits may not be observed for several weeks, so it is important to reliably assess baseline symptoms to enable comparison with the treatment effects. Women should also be cautioned about this delay in response.

Vaginal oestrogen—Low-dose vaginal oestrogen should be offered when appropriate to women with urogenital atrophic changes, to promote improved blood supply and decrease LUTS. A meta-analysis of 14 randomized controlled trials (RCTs) in postmenopausal women showed that vaginal oestrogen was associated with improved incontinence[122]. Systemic hormone replacement therapy has been associated with worsening incontinence outcomes[123], particularly for combination oestrogen and progesterone. The reasons for this harmful effect remain unclear.

Anticholinergic drugs for urgency incontinence—Anticholinergic drugs (also known as antimuscarinics) are often used in primary care alongside behavioural and lifestyle modifications as part of first-line therapy. Anticholinergic drugs act directly on the detrusor muscle, which leads to reductions in urgency urinary incontinence, with concomitant improvements in urinary urgency, voiding frequency and, to a lesser extent, nocturia[124,125] (BOX 2). Improvement in these key bothersome symptoms is associated with moderate gains in quality of life[126]. The different licensed anticholinergics offer a range of dosing schedules and, in the case of oxybutynin, are available as either oral or topical formulations, with the latter preparation also available over the counter for women in the United States. All available drugs cause typical cholinergic adverse effects, including dry mouth, constipation, blurred vision, somnolence and confusion[127–129] (TABLE 1). Owing to adverse effects, costs and inconvenience, adherence to treatment is often poor[130–132].

Oxybutynin was the first drug with level 1 evidence of efficacy and is sometimes still recommended as a first choice, despite its poor adverse-effect profile[76]. All newer anticholinergic medications for overactive bladder syndrome have been compared in RCTs against both placebo and oxybutynin as a condition of licensing. The overall effect of this class of drugs is very modest, with few long-term treatment responders[133]. Very few head-to-head studies of contemporary anticholinergic medications have been conducted, and when direct comparisons have been made, the evidence of differential efficacy is limited[134]. The long-term effects of anticholinergic incontinence medications (cholinergic load) on cognitive function and incident dementia are emerging[135], particularly in older patients and in those already using other medications with anticholinergic effects. In these patients, alternative medications are recommended[83].

β_3-adrenergic agonists for urgency incontinence—Mirabegron is a β_3-adrenergic receptor agonist that acts directly on the detrusor muscle. In licensing trials, mirabegron was tested against placebo and tolterodine (an antimuscarinic), and subsequent post-marketing trials tested it against, and in combination with, several anticholinergics[136,137]. Meta-analyses have shown similar efficacy to available anticholinergic drugs, but with a much reduced incidence of dry mouth[138,139], and lower efficacy than intravesical onabotulinumtoxinA[140]. Mirabegron has the potential to exacerbate hypertension[131], but is otherwise well tolerated compared with most anticholinergics, with good persistence[141]. However, for most patients, symptom improvement is quite modest. Mirabegron is best used in patients for whom there is a contraindication to, or risk of cognitive or other adverse effects from, anticholinergics.

Serotonin–noradrenaline reuptake inhibitors for stress incontinence—Duloxetine is a serotonin–noradrenaline reuptake inhibitor (SNRI) that is widely used in depression. SNRIs are proposed to increase the levels of serotonin and noradrenaline at the presynaptic neuron in the nucleus of Onuf (FIG. 3) of the sacral spinal cord by inhibiting reuptake and increasing striated urethral sphincter activity and urethral pressures[142]. Duloxetine was licensed for the treatment of stress incontinence in the European Union after RCTs initially suggested efficacy[143], but licensing for the indication of stress incontinence failed in the United States owing to concerns of adverse events, including nausea and suicidal ideation. Accordingly, the drug is not recommended as first-line therapy[59,76], and persistence of use for the indication of incontinence is poor[144]; thus it is not commonly used.

Invasive interventions

Stress incontinence surgery—Although conservative measures should be tried first[59], surgery to treat stress urinary incontinence is highly effective in reducing symptoms (FIG. 8). Women with mixed incontinence are candidates for surgery but are likely to need adjunctive treatment for their urgency incontinence. Synthetic mid-urethral sling placement is currently the first-line surgical procedure, with a cure rate of >80%[145] and low morbidity rate[145]. Both the retropubic and transobturator techniques are based on a strip of polypropylene mesh[146]. Complications of vaginal mesh surgery have become a common cause of litigation, and although the risk of complications is lower for mid-urethral slings than for mesh for prolapse, these procedures need meticulous documentation of the consent process. For women and/or clinicians seeking nonmesh-based procedures, a fascial sling or retropubic urethropexy are viable options[147], with similar overall efficacy[148] and similar complication rates, but additional risks[149]. Surgical risks include bleeding, infection, voiding dysfunction, visceral injury, pain and anaesthetic concerns. Persistent stress urinary incontinence after an initial surgery should prompt an updated diagnostic evaluation; subsequent stress incontinence surgery is a possibility for women whose initial surgery was not successful and whose diagnosis remains stress urinary incontinence, but are generally less successful than the primary procedure[150].

A small number of women develop *de novo* urgency urinary incontinence symptoms following stress incontinence surgery[145], which can be associated with position, tension or the inadvertent introduction of surgical material within the bladder or urethra. Once such

complications are excluded, traditional treatment for urgency urinary incontinence can be initiated. More commonly, women with mixed urinary incontinence undergo surgery to resolve the stress urinary incontinence component of their condition. Although some of these women experience resolution of their urgency urinary incontinence, some have ongoing, bothersome symptoms[151]. Treatments aimed at reducing urgency urinary incontinence symptoms that were not effective before surgery should be tried again, especially if after the surgery the symptoms of stress urinary incontinence are reduced or resolved. Clinician encouragement and management of expectations are especially important during this post-surgical treatment window, as women might feel dejected that incontinence symptoms are present despite complete or near-complete resolution of stress urinary incontinence symptoms.

Neuromodulation—Neuromodulation uses direct electrical stimulation to modify bladder sensation and contraction. Various forms of neuromodulation are also available and might be preferred by women with urgency incontinence who wish to avoid daily oral medication. Implantable neurostimulation uses a programmable stimulator placed subcutaneously that delivers low-amplitude electrical stimulation to the sacral plexus via a lead through the S3 foramen. It has high initial costs and high risks of revision surgery[152]. By contrast, percutaneous tibial nerve stimulation is a less-invasive, office-based technique that offers a reasonable rate of symptom response[153,154]. Neither type of neurostimulation is uniformly effective, and careful patient selection is mandatory for both. These therapies should be considered third line after failure of first-line and second-line therapies.

Intravesical onabotulinumtoxinA injections—Intravesical injection of onabotulinumtoxinA is essentially a form of ‘chemical’ neuromodulation that acts at the detrusor presynaptic neuromuscular junction. It has an efficacy similar to oral medication for urgency urinary incontinence but without the need for a daily medication[155,156]. The administration procedure is usually performed under a general anaesthetic· and is accordingly reserved as a third-line treatment. Treatment injections need to be repeated when symptoms recur (approximately 9–12 months). The risks include transient urinary retention and UTIs, which may require patients to learn to pass a urinary catheter to drain off any residual urine. Despite these risks· the procedure is popular with patients and physicians and offers a real hope of cure for patients with even the most recalcitrant symptoms.

Quality of life

During a woman’s lifetime· various health conditions can challenge bladder function. Urinary incontinence symptoms can worsen or improve· and the desire for management can wax and wane. A woman who has experienced prior success with non-invasive treatments might initiate self-care or request referral for simple management. A woman who underwent successful surgery 15 years ago may now be experiencing a change in bladder symptoms, despite efforts at regular pelvic floor muscle exercises. Those who have lived with urinary incontinence as a chronic condition might feel nihilistic about management recommendations; instead, goals might relate to avoidance of certain threshold events, such as avoiding placement in a nursing home because of bladder control concerns.

All subtypes of urinary incontinence are highly bothersome, and management should begin by assessing the patient's willingness to engage in treatment, determining the level of treatment desired and discussing current evidence and recommendations for specific treatment, including benefits, alternatives, risks and complications. Furthermore, assessment of quality of life has become an integral part of determining the effect of urinary incontinence on the individual and assessing the benefit of treatments.

Patient-reported outcomes (PROs) provide an objective assessment of subjective symptoms that may assist in identifying the primary symptom to be treated, choosing the type of treatment and determining treatment goals and success. In the research setting, PROs provide a standardized set of data to be collected and outcome measures. Although these tools are widely used and recommended, no evidence suggests that the use of PROs alone improves treatment outcomes[83]. Several PRO tools have been developed (TABLE 2), but no single questionnaire has been validated to measure all incontinence-associated outcomes[83] and no single questionnaire has been universally adopted for the assessment of quality oflife[157]. When considering a questionnaire, clinicians and researchers should assess its compatibility with their outcome objectives, suitability in the patient group and consider the time and effort burden of lengthy surveys on patients.

The severity of urinary incontinence has been reported as a risk factor for poor quality of life[158] and has a negative effect on many dimensions of quality of life, mental health and social activities. Although not life-threatening, urinary incontinence can certainly prove to be life-changing[159]. In one Austrian study, 65.7% of women stated that their quality of life was affected by continence status[160]. In another population-based longitudinal study, urinary incontinence predicted the onset of psychological distress among community-dwelling adults (≥50 years of age), especially when associated with condition-specific functional loss, and this distress can prompt the individual to avoid social or religious gatherings, travel, physical activities and other everyday activities such as shopping[161]. Incontinence symptoms have also been associated with sexual dysfunction[162] and negative effects on marital relationships[163] or partnerships. Women with urgency urinary incontinence are generally more bothered than those with stress urinary incontinence, because the leakage is unexpected, sudden and often of large volume. These women show worse scores on quality of life and depression scales, poorer quality of sleep, worse sexual function and lower productivity than matched controls[164].

Urinary incontinence also has negative effects on the psychological burden of family caregivers[165]. The mean total score from the Zarit Burden Interview, one of the most widely used tools to measure caregiver burden, was significantly higher among caregivers of patients who were incontinent than among those of patients who were continent. Male partners of women with urinary incontinence also report lower sexual function and sexual satisfaction[166]. Overall, female urinary incontinence and urgency negatively affect sexual function in almost 50% of affected women and in 20% of their partners[167].

Finally, understanding the crucial link between urinary incontinence and quality of life is pivotal to maximizing the efficacy of routine screening and early intervention[168]. Given the broad extent of the reduction in quality of life from urinary incontinence, measurement of

quality of life should also be considered in the design of almost all clinical studies of urinary incontinence interventions.

Outlook

Advances in incontinence research suggest that we might be at the cusp of a revolution in several research areas, especially in understanding continence physiology and in primary prevention. More than 130 years of research into bladder and urethral sphincter dysfunction[169] have revealed overlapping underlying causes of bladder dysfunction, including the myogenic, neurogenic and urotheliogenic hypotheses[42]. However, the development of new drugs has been hampered by a historical focus on detrusor overactivity as a supposed unifying 'cause' of urinary urgency and urgency incontinence. The relationship between urgency and detrusor overactivity is remarkably complex — whether detrusor overactivity is a cause or a consequence of urgency incontinence is itself unclear.

Despite a lack of evidence linking detrusor overactivity to urgency incontinence, it has often also been used as a surrogate end point in clinical trials of interventions of all modalities. Many current treatment options, including anticholinergic medicines and sacral neuromodulation, reduce detrusor overactivity measurably. However, the presence of detrusor overactivity at baseline has been shown to be a poor predictive factor of treatment outcome for a wide variety of incontinence interventions[44]. There is considerable interest in new biochemical and imaging biomarkers that might replace detrusor overactivity as an objective clinical end point; however, none has progressed to widespread adoption in clinical practice. The most widely tested urinary marker, nerve growth factor, has demonstrated problems with specificity and reproducibility[170]. The most widely investigated imaging marker, bladder wall thickness, also failed to demonstrate prognostic value when tested in a large trial of implementation[171]. At the present time, the best clinical end points are subjective rather than objective: symptom severity and quality-of-life impact as reported by patients on validated instruments.

Recent genome-wide association studies have attempted to shed light on the molecular pathogenesis of incontinence. The discovery of new genetic risk variants for incontinence[172,173] and the interrogation of those variants as known causes of objectively measured bladder dysfunction might provide striking insight into the actual variation in human bladder physiology associated with incontinence. Accordingly, we might identify new subtypes of urgency incontinence at which we can direct targeted care.

In addition to the expanding understanding of bladder physiology, primary prevention of incontinence is a growing area of interest. For example, our increasing understanding of the microbiota of the urinary tract might lead to effective interventions to correct urinary dysbiosis or to modify the resident microbial flora. Topical vaginal oestrogens are already used for postmenopausal women with incontinence, and, in part, act to normalize the vaginal flora[174]. Four currently available bladder vaccines (Urovac (Solco Basel) includes heat-killed uropathogens as an intramuscular injection; OM-89/Uro-Vaxom (Vifor Pharma), an oral preparation of bacterial lysates; and Urvakol (Institute of Microbiology, Prague, Czech Republic) and Urostim (National Center of Infectious and Parasitic Diseases, Sofia,

Bulgaria), which are whole-cell inactivated uropathogens in an oral tablet) target *Escherichia coli* and other uropathogens, and are marketed for the prevention of recurrent UTI with modest evidence of efficacy[175]. Novel vaccine approaches are currently emerging. It seems plausible that similar targeted interventions to modify the vaginal or bladder microbiota might be useful for incontinence.

Successful primary prevention of incontinence will depend on the accurate identification of women at high risk. Cohort studies suggest that persistent childhood incontinence strongly predicts adult symptoms[176,177]. Although it is unclear whether this predisposition to incontinence is predominantly genetic or environmental, the recognition of predisposition offers an opportunity to intervene at appropriate times. In parous women, vaginal delivery is a key modifiable risk factor for adult stress urinary incontinence, with traumatic delivery unmasking incontinence in predisposed women[177].

Ongoing studies, such as UR-CHOICE[178], aim to test the acceptability of providing pregnant women with tailored risk estimates for incontinence, and will provide valuable insights into the efficacy of risk-reducing antenatal and intrapartum interventions that target high-risk women. Such data would provide important information for obstetricians, who must be selective in the use of pre-labour caesarian section, with vaginal birth being only one contributing factor for incontinence[179]. The risks and benefits of elective caesarian should be carefully weighed given the substantial risks as a primary procedure and for future deliveries, including maternal mortality, stillbirth, placenta praevia, uterine rupture, neonatal respiratory morbidity, hysterectomy, and bladder and bowel injuries. In the absence of interventional studies, new research is needed to understand whether genetic information can be additionally used to successfully identify women who would benefit from caesarian delivery, and whether such a strategy would have any sustained benefit to incontinence and any overall impact on health-related quality of life.

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References

1. Haylen BT, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. Neurourol Urodyn. 2009; 29:4–20. A useful document on the terminology of urinary incontinence that is important for clinical practice and research.
2. Shakespeare K, Barradell V, Orme S. Management of urinary incontinence in frail elderly women. Obstet Gynaecol Reprod Med. 2011; 21:281–287.
3. Saarni SI, et al. The impact of 29 chronic conditions on health-related quality of life: a general population survey in Finland using 15D and EQ-5D. Qual Life Res. 2006; 15:1403–1414. [PubMed: 16960751]
4. Subak LL, et al. High costs of urinary incontinence among women electing surgery to treat stress incontinence. Obstet Gynecol. 2008; 111:899–907. [PubMed: 18378749]
5. Ganz ML, et al. Economic costs of overactive bladder in the United States. Urology. 2010; 75:526–532.e18. [PubMed: 20035977]

6. Irwin DE, et al. The economic impact of overactive bladder syndrome in six Western countries. BJU Int. 2009; 103:202–209. [PubMed: 19278532]
7. Hannestad YS, Rortveit G, Sandvik H, Hunskaar S. A community-based epidemiological survey of female urinary incontinence. J Clin Epidemiol. 2000; 53:1150–1157. [PubMed: 11106889]
8. Vaughan CP, et al. Impact of obesity on urinary storage symptoms: results from the FINNO Study. J Urol. 2013; 189:1377–1382. [PubMed: 23103801]
9. Greer WJ, Richter HE, Bartolucci AA, Burgio KL. Obesity and pelvic floor disorders. Obstet Gynecol. 2008; 112:341–349. [PubMed: 18669733]
10. Mishra GD, Hardy R, Cardozo L, Kuh D. Body weight through adult life and risk of urinary incontinence in middle-aged women: results from a British prospective cohort. Int J Obes. 2008; 32:1415–1422.
11. Agarwal A, et al. What is the most bothersome lower urinary tract symptom? Individual- and population-level perspectives for both men and women. Eur Urol. 2014; 65:1211–1217. [PubMed: 24486308]
12. Tikkinen KAO, Leinonen JS, Guyatt GH, Ebrahim S, Järvinen TLN. What is a disease? Perspectives of the public, health professionals and legislators. BMJ Open. 2012; 2:e001632.
13. World Health Organization. International statistical classification of diseases and related health problems. WHO; 2010. 10th revisionhttp://www.who.int/classifications/icd/ICD10Volume2_en_2010.pdf
14. Mitteness LS. Knowledge and beliefs about urinary incontinence in adulthood and old age. J Am Geriatr Soc. 1990; 38:374–378. [PubMed: 2179382]
15. Norton PA, MacDonald LD, Sedgwick PM, Stanton SL. Distress and delay associated with urinary incontinence, frequency, and urgency in women. BMJ. 1988; 297:1187–1189. [PubMed: 3144344]
16. Helfand BT, Evans RM, McVary KT. A comparison of the frequencies of medical therapies for overactive bladder in men and women: analysis of more than 7.2 million aging patients. Eur Urol. 2010; 57:586–591. [PubMed: 20036783]
17. Wu JM, Matthews CA, Conover MM, Pate V, Jonsson Funk M. Lifetime risk of stress urinary incontinence or pelvic organ prolapse surgery. Obstet Gynecol. 2014; 123:1201–1206. [PubMed: 24807341]
18. Elstad EA, Taubenberger SP, Botelho EM, Tennstedt SL. Beyond incontinence: the stigma of other urinary symptoms. J Adv Nurs. 2010; 66:2460–2470. [PubMed: 20735505]
19. Thom DH, et al. Differences in prevalence of urinary incontinence by race/ethnicity. J Urol. 2006; 175:259–264. [PubMed: 16406923]
20. Klovning A, Sandvik H, Hunskaar S. Web-based survey attracted age-biased sample with more severe illness than paper-based survey. J Clin Epidemiol. 2009; 62:1068–1074. [PubMed: 19246177]
21. Bedretdinova D, Fritel X, Panjo H, Ringa V. Prevalence of female urinary incontinence in the general population according to different definitions and study designs. Eur Urol. 2016; 69:256–264. [PubMed: 26259998]
22. Minassian VA, Drutz HP, Al-Badr A. Urinary incontinence as a worldwide problem. Int J Gynecol Obstet. 2003; 82:327–338.
23. Cerruto MA, D'Elia C, Aloisi A, Fabrello M, Artibani W. Prevalence, incidence and obstetric factors' impact on female urinary incontinence in Europe: a systematic review. Urol Int. 2013; 90:1–9. [PubMed: 22868349]
24. Irwin DE, et al. Population-based survey of urinary incontinence, overactive bladder, and other lower urinary tract symptoms in five countries: results of the EPIC Study. Eur Urol. 2006; 50:1306–1315. [PubMed: 17049716]
25. Minassian VA, Stewart WF, Wood GC. Urinary incontinence in women. Obstet Gynecol. 2008; 111:324–331. [PubMed: 18238969]
26. Ebbesen MH, Hunskaar S, Rortveit G, Hannestad YS. Prevalence, incidence and remission of urinary incontinence in women: longitudinal data from the Norwegian HUNT study (EPINCONT). BMC Urol. 2013; 13:27. An important epidemiological study of the natural history of incontinence in women. [PubMed: 23721491]

27. Zhang L, et al. A population-based survey of the prevalence, potential risk factors, and symptom-specific bother of lower urinary tract symptoms in adult Chinese women. Eur Urol. 2015; 68:97–112. [PubMed: 25572826]
28. Botlero R, Urquhart DM, Davis SR, Bell RJ. Prevalence and incidence of urinary incontinence in women: review of the literature and investigation of methodological issues. Int J Urol. 2008; 15:230–234. [PubMed: 18304218]
29. Thom D. Variation in estimates of urinary incontinence prevalence in the community: effects of differences in definition, population characteristics, and study type. J Am Geriatr Soc. 1998; 46:473–480. [PubMed: 9560071]
30. Milsom, I., et al. Incontinence. 5th International Consultation on Incontinence. Abrams, P.Cardozo, L.Khoury, S., Wein, A., editors. ICUD-EAU; 2013. p. 15-107.
31. Stewart WF, et al. Urinary incontinence incidence: quantitative meta-analysis of factors that explain variation. J Urol. 2014; 191:996–1002. [PubMed: 24140547]
32. Wu JM, Hundley AF, Fulton RG, Myers ER. Forecasting the prevalence of pelvic floor disorders in U.S. women: 2010 to 2050. Obstet Gynecol. 2009; 114:1278–1283. [PubMed: 19935030]
33. Goode PS, et al. Population based study of incidence and predictors of urinary incontinence in black and white older adults. J Urol. 2008; 179:1449–1454. [PubMed: 18295279]
34. Mitchell ES, Woods NF. Correlates of urinary incontinence during the menopausal transition and early postmenopause: observations from the Seattle Midlife Women's Health Study. Climacteric. 2013; 16:653–662. [PubMed: 23560943]
35. Fenner DE, et al. Establishing the prevalence of incontinence study: racial differences in women's patterns of urinary incontinence. J Urol. 2008; 179:1455–1460. [PubMed: 18295278]
36. Zhu L, et al. The epidemiological study of women with urinary incontinence and risk factors for stress urinary incontinence in China. Menopause. 2009; 16:831–836. [PubMed: 19240656]
37. Ebbesen MH, Hannestad YS, Midthjell K, Hunskaar S. Diabetes and urinary incontinence — prevalence data from Norway. Acta Obstet Gynecol Scand. 2007; 86:1256–1262. [PubMed: 17851814]
38. Brown JS, et al. Urinary incontinence in older women: who is at risk? Study of Osteoporotic Fractures Research Group. Obstet Gynecol. 1996; 87:715–721. [PubMed: 8677073]
39. Gomez-Amaya SM, Ruggieri MR, Arias Serrato SA, Massicotte VS, Barbe MF. Gross anatomical study of the nerve supply of genitourinary structures in female mongrel hound dogs. Anat Histol Embryol. 2014; 44:118–127. [PubMed: 24730986]
40. Hillary CJ, Osman N, Chapple C. Considerations in the modern management of stress urinary incontinence resulting from intrinsic sphincter deficiency. World J Urol. 2015; 33:1251–1256. [PubMed: 26060138]
41. DeLancey JOL. Structural support of the urethra as it relates to stress urinary incontinence: the hammock hypothesis. Am J Obstet Gynecol. 1994; 170:1713–1723. An explanation of stress urinary incontinence pathology and the hammock hypothesis. [PubMed: 8203431]
42. Roosen A, et al. A refocus on the bladder as the originator of storage lower urinary tract symptoms: a systematic review of the latest literature. Eur Urol. 2009; 56:810–820. [PubMed: 19683859]
43. Enck P, et al. Irritable bowel syndrome. Nat Rev Dis Primers. 2016; 2:16014. [PubMed: 27159638]
44. Cartwright R, Afshan I, Derpapas A, Vijaya G, Khullar V. Novel biomarkers for overactive bladder. Nat Rev Urol. 2011; 8:139–145. [PubMed: 21321572]
45. de Groat, WC., Griffiths, D., Yoshimura, N. Comprehensive Physiology. Wiley-Blackwell; 2014. p. 327-396.
46. Li M, Sun Y, Simard JM, Chai TC. Increased transient receptor potential vanilloid type 1 (TRPV1) signaling in idiopathic overactive bladder urothelial cells. Neurourol Urodyn. 2011; 30:606–611. [PubMed: 21351130]
47. Reid G, Burton JP. Urinary incontinence: making sense of the urinary microbiota in clinical urology. Nat Rev Urol. 2016; 13:567–568. An overview of a paradigm shift: the urinary tract is not sterile and bacteria might have important roles in urinary incontinence. [PubMed: 27644934]
48. Hilt EE, et al. Urine is not sterile: use of enhanced urine culture techniques to detect resident bacterial flora in the adult female bladder. J Clin Microbiol. 2013; 52:871–876. [PubMed: 24371246]

49. Sorrentino F, et al. Associations between individual lower urinary tract symptoms and bacteriuria in random urine samples in women. Neurourol Urodyn. 2014; 34:429–433. [PubMed: 24719249]
50. Walsh CA, Moore KH. Overactive bladder in women: does low-count bacteriuria matter? A review. Neurourol Urodyn. 2010; 30:32–37.
51. Khasriya R, et al. Spectrum of bacterial colonization associated with urothelial cells from patients with chronic lower urinary tract symptoms. J Clin Microbiol. 2013; 51:2054–2062. [PubMed: 23596238]
52. Ravel J, et al. Vaginal microbiome of reproductive-age women. Proc Natl Acad Sci USA. 2010; 108:4680–4687. [PubMed: 20534435]
53. Pearce MM, et al. The female urinary microbiome: a comparison of women with and without urgency urinary incontinence. mBio. 2014; 5:e01283–14. [PubMed: 25006228]
54. Brubaker L, Wolfe AJ. Microbiota in 2016: associating infection and incontinence with the female urinary microbiota. Nat Rev Urol. 2017; 14:72–74. [PubMed: 28050013]
55. Abrams, P., et al. Incontinence. 5th International Consultation on Incontinence. Abrams, P.Cardozo, L.Khoury, S., Wein, A., editors. ICUD-EAU; 2013. p. 1895-1956.
56. Martin JL, Williams KS, Sutton AJ, Abrams KR, Assassa RP. Systematic review and meta-analysis of methods of diagnostic assessment for urinary incontinence. Neurourol Urodyn. 2006; 25:674–683. [PubMed: 17016795]
57. Ruby CM, et al. The effect of medication use on urinary incontinence in community-dwelling elderly women. J Am Geriatr Soc. 2010; 58:1715–1720. [PubMed: 20670377]
58. Rait G, et al. Prevalence of cognitive impairment: results from the MRC trial of assessment and management of older people in the community. Age Ageing. 2005; 34:242–248. [PubMed: 15863409]
59. Lucas MG, et al. EAU guidelines on assessment and nonsurgical management of urinary incontinence. Eur Urol. 2012; 62:1130–1142. [PubMed: 22985745]
60. Wagg A, et al. Urinary incontinence in frail elderly persons: report from the 5th International Consultation on Incontinence. Neurourol Urodyn. 2014; 34:398–406. [PubMed: 24700771]
61. Swift SE, Yoon EA. Test-retest reliability of the cough stress test in the evaluation of urinary incontinence. Obstet Gynecol. 1999; 94:99–102. [PubMed: 10389726]
62. Hsu TH, Rackley RR, Appell RA. The supine stress test: a simple method to detect intrinsic urethral sphincter dysfunction. J Urol. 1999; 162:460–463. [PubMed: 10411057]
63. Ghoniem G, et al. Evaluation and outcome measures in the treatment of female urinary stress incontinence: International Urogynecological Association (IUGA) guidelines for research and clinical practice. Int Urogynecol J. 2007; 19:5–33. An important paper that describes evaluation and outcome measures for the treatment of stress urinary incontinence.
64. Orejuela FJ, Shek KL, Dietz HP. The time factor in the assessment of prolapse and levator ballooning. Int Urogynecol J. 2011; 23:175–178. [PubMed: 21887550]
65. Bump RC, et al. The standardization of terminology of female pelvic organ prolapse and pelvic floor dysfunction. Am J Obstet Gynecol. 1996; 175:10–17. [PubMed: 8694033]
66. Swift S, et al. Validation of a simplified technique for using the POPQ pelvic organ prolapse classification system. Int Urogynecol J. 2006; 17:615–620.
67. Jelovsek JE, Maher C, Barber MD. Pelvic organ prolapse. Lancet. 2007; 369:1027–1038. [PubMed: 17382829]
68. Visco AG, et al. The role of preoperative urodynamic testing in stress-continent women undergoing sacrocolpopexy: the Colpopexy and Urinary Reduction Efforts (CARE) randomized surgical trial. Int Urogynecol J. 2008; 19:607–614.
69. Kim S-O, Chung HS, Hwang EC, Oh KJ, Kwon D. Point Aa of the POP-Q system as a predictive value of urethral mobility in women. Int Urogynecol J. 2010; 22:687–691. [PubMed: 21104403]
70. Swift S, Barnes D, Herron A, Goodnight W. Test–retest reliability of the cotton swab (Q-tip®) test in the evaluation of the incontinent female. Int Urogynecol J. 2010; 21:963–967. [PubMed: 20379698]

71. Meyer I, Szychowski JM, Illston JD, Parden AM, Richter HE. Vaginal swab test compared with the urethral Q-tip test for urethral mobility measurement. Obstet Gynecol. 2016; 127:348–352. [PubMed: 26942364]
72. Schaer G, Koechli O, Schuessler B, Haller U. Perineal ultrasound for evaluating the bladder neck in urinary stress incontinence. Obstet Gynecol. 1995; 85:220–224. [PubMed: 7824234]
73. Troeger C, Gugger M, Holzgreve W, Wight E. Correlation of perineal ultrasound and lateral chain urethrocystography in the anatomical evaluation of the bladder neck. Int Urogynecol J Pelvic Floor Dysfunct. 2003; 14:380–384. [PubMed: 14676997]
74. Richter HE, et al. Demographic and clinical predictors of treatment failure one year after midurethral sling surgery. Obstet Gynecol. 2011; 117:913–921. [PubMed: 21422865]
75. Bo K, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for the conservative and nonpharmacological management of female pelvic floor dysfunction. Int Urogynecol J. 2016; 28:191–213. [PubMed: 27921161]
76. Smith A, Bevan D, Douglas HR, James D. Management of urinary incontinence in women: summary of updated NICE guidance. BMJ. 2013; 347:f5170. A good summary of the current evidence for treatments of urinary incontinence. [PubMed: 24021756]
77. Buchsbaum GM, Albushies DT, Guzick DS. Utility of urine reagent strip in screening women with incontinence for urinary tract infection. Int Urogynecol J. 2004; 15:391–393.
78. Hessdoerfer E, Jundt K, Peschers U. Is a dipstick test sufficient to exclude urinary tract infection in women with overactive bladder? Int Urogynecol J. 2010; 22:229–232. [PubMed: 20838986]
79. Holm A, Aabenhus R. Urine sampling techniques in symptomatic primary-care patients: a diagnostic accuracy review. BMC Fam Pract. 2016; 17:72. [PubMed: 27278078]
80. Cai T, Koves B, Johansen TEB. Asymptomatic bacteriuria, to screen or not to screen — and when to treat? Curr Opin Urol. 2017; 27:107–111. [PubMed: 27906777]
81. Nicolle LE. Urinary tract infections in the older adult. Clin Geriatr Med. 2016; 32:523–538. [PubMed: 27394021]
82. Bettez M, et al. 2012 update: guidelines for adult urinary incontinence collaborative consensus document for the Canadian Urological Association. Can Urol Assoc J. 2012; 6:354–363. [PubMed: 23093627]
83. Thüroff JW, et al. EAU guidelines on urinary incontinence. Eur Urol. 2011; 59:387–400. [PubMed: 21130559]
84. Saaby M-L, Lose G. Repeatability of post-void residual urine ≥100 ml in urogynaecologic patients. Int Urogynecol J. 2012; 23:207–209. [PubMed: 22159563]
85. Ashton-Miller JA, DeLancey JO. Functional anatomy of the female pelvic floor. Ann NY Acad Sci. 2007; 1101:266–296. [PubMed: 17416924]
86. Dietz HP. Pelvic floor ultrasound in prolapse: what's in it for the surgeon? Int Urogynecol J. 2011; 22:1221–1232. [PubMed: 21656320]
87. Dietz HP, Nazemian K, Shek KL, Martin A. Can urodynamic stress incontinence be diagnosed by ultrasound? Int Urogynecol J. 2013; 24:1399–1403. [PubMed: 23314227]
88. Serati M, et al. Ultrasound measurement of bladder wall thickness in different forms of detrusor overactivity. Int Urogynecol J. 2010; 21:1405–1411. [PubMed: 20535449]
89. Bogusiewicz M, et al. Both the middle and distal sections of the urethra may be regarded as optimal targets for 'outside-in' transobturator tape placement. World J Urol. 2014; 32:1605–1611. [PubMed: 24531879]
90. Herderschee R, Hay-Smith EJC, Herbison GP, Roovers JP, Heineman MJ. Feedback or biofeedback to augment pelvic floor muscle training for urinary incontinence in women. Cochrane Database Syst Rev. 2011; 7:CD009252.
91. Versi E. Videourodynamic diagnosis of occult genuine stress incontinence in patients with anterior vaginal wall relaxation. J Soc Gynecol Investig. 1998; 5:327–330.
92. Schäfer W, et al. Good urodynamic practices: uroflowmetry, filling cystometry, and pressure–flow studies. Neurourol Urodyn. 2002; 21:261–274. [PubMed: 11948720]

93. Clement KD, Lapitan MCM, Omar MI, Glazener CMA. Urodynamic studies for management of urinary incontinence in children and adults. Cochrane Database Syst Rev. 2013; 10:CD003195.
94. Digesu GA, Khullar V, Cardozo L, Salvatore S. Overactive bladder symptoms: do we need urodynamics? Neurourol Urodyn. 2003; 22:105–108. [PubMed: 12579626]
95. Nager CW, et al. A randomized trial of urodynamic testing before stress-incontinence surgery. N Engl J Med. 2012; 366:1987–1997. A seminal RCT that demonstrates that, for women with uncomplicated, demonstrable stress urinary incontinence, preoperative office evaluation alone is not inferior to evaluation with urodynamic testing. [PubMed: 22551104]
96. van Leijsen SAL, et al. Can preoperative urodynamic investigation be omitted in women with stress urinary incontinence? A non-inferiority randomized controlled trial. Neurourol Urodyn. 2012; 31:1118–1123. [PubMed: 22488817]
97. Stöhrer M, et al. EAU guidelines on neurogenic lower urinary tract dysfunction. Eur Urol. 2009; 56:81–88. [PubMed: 19403235]
98. Bai SW, et al. Relationship between stress urinary incontinence and pelvic organ prolapse. Int Urogynecol J. 2002; 13:256–260.
99. Burgio KL. Update on behavioral and physical therapies for incontinence and overactive bladder: the role of pelvic floor muscle training. Curr Urol Rep. 2013; 14:457–464. [PubMed: 23913199]
100. Fonda D, Abrams P. Cure sometimes, help always — a 'continence paradigm' for all ages and conditions. Neurourol Urodyn. 2006; 25:290–292. [PubMed: 16496393]
101. Elkadry EA, Kenton KS, FitzGerald MP, Shott S, Brubaker L. Patient-selected goals: a new perspective on surgical outcome. Am J Obstet Gynecol. 2003; 189:1551–1557. [PubMed: 14710061]
102. Mahajan ST, Elkadry EA, Kenton KS, Shott S, Brubaker L. Patient-centered surgical outcomes: the impact of goal achievement and urge incontinence on patient satisfaction one year after surgery. Am J Obstet Gynecol. 2006; 194:722–728. [PubMed: 16522404]
103. Vrijens DMJ, Spakman JI, van Koeveringe GA, Berghmans B. Patient-reported outcome after treatment of urinary incontinence in a multidisciplinary pelvic care clinic. Int J Urol. 2015; 22:1051–1057. [PubMed: 26264653]
104. Myers DL. Female mixed urinary incontinence: a clinical review. JAMA. 2014; 311:2007–2014. [PubMed: 24846038]
105. Borrie MJ, Bawden M, Speechley M, Kloseck M. Interventions led by nurse continence advisers in the management of urinary incontinence: a randomized controlled trial. CMAJ. 2002; 166:1267–1273. [PubMed: 12041843]
106. Huang AJ. Nonsurgical treatments for urinary incontinence in women. JAMA Intern Med. 2013; 173:1463–1464. [PubMed: 23817490]
107. Hunskaar S. A systematic review of overweight and obesity as risk factors and targets for clinical intervention for urinary incontinence in women. Neurourol Urodyn. 2008; 27:749–757. [PubMed: 18951445]
108. Subak LL, Richter HE, Hunskaar S. Obesity and urinary incontinence: epidemiology and clinical research update. J Urol. 2009; 182:S2–S7. [PubMed: 19846133]
109. Subak LL, et al. Weight loss to treat urinary incontinence in overweight and obese women. N Engl J Med. 2009; 360:481–490. [PubMed: 19179316]
110. Burgio KL, Richter HE, Clements RH, Redden DT, Goode PS. Changes in urinary and fecal incontinence symptoms with weight loss surgery in morbidly obese women. Obstet Gynecol. 2007; 110:1034–1040. [PubMed: 17978117]
111. Dumoulin C, Hay-Smith J, Habée-Séguin Mac, G, Mercier J. Pelvic floor muscle training versus no treatment, or inactive control treatments, for urinary incontinence in women: a short version Cochrane systematic review with meta-analysis. Neurourol Urodyn. 2014; 34:300–308. [PubMed: 25408383]
112. Sjöström M, et al. Internet-based treatment of stress urinary incontinence: 1- and 2-year results of a randomized controlled trial with a focus on pelvic floor muscle training. BJU Int. 2015; 116:955–964. [PubMed: 25683075]

113. Sjöström M, Umefjord G, Lindholm L, Samuelsson E. Cost-effectiveness of an Internet-based treatment program for stress urinary incontinence. Neurourol Urodyn. 2013; 34:244–250. [PubMed: 24347521]
114. Bø K. Pelvic floor muscle training is effective in treatment of female stress urinary incontinence, but how does it work? Int Urogynecol J Pelvic Floor Dysfunct. 2004; 15:76–84. [PubMed: 15014933]
115. Wyman JF, Burgio KL, Newman DK. Practical aspects of lifestyle modifications and behavioural interventions in the treatment of overactive bladder and urgency urinary incontinence. Int J Clin Pract. 2009; 63:1177–1191. [PubMed: 19575724]
116. Miller JM, Sampselle C, Ashton-Miller J, Hong G-RS, DeLancey JOL. Clarification and confirmation of the Knack maneuver: the effect of volitional pelvic floor muscle contraction to preempt expected stress incontinence. Int Urogynecol J Pelvic Floor Dysfunct. 2008; 19:773–782. [PubMed: 18204797]
117. Dumoulin C, Hay-Smith EJC, Mac Habée-Séguin G. Pelvic floor muscle training versus no treatment, or inactive control treatments, for urinary incontinence in women. Cochrane Database Syst Rev. 2014; 5:CD005654.
118. Beyar N, Groutz A. Pelvic floor muscle training for female stress urinary incontinence: five years outcomes. Neurourol Urodyn. 2015; 36:132–135. [PubMed: 26397715]
119. Glazener CMA, et al. Twelve-year follow-up of conservative management of postnatal urinary and faecal incontinence and prolapse outcomes: randomised controlled trial. BJOG. 2013; 121:112–120. [PubMed: 24148807]
120. Richter HE, et al. Continence pessary compared with behavioral therapy or combined therapy for stress incontinence. Obstet Gynecol. 2010; 115:609–617. [PubMed: 20177294]
121. Morris AR, Moore KH. The Contiform incontinence device? Efficacy and patient acceptability. Int Urogynecol J Pelvic Floor Dysfunct. 2003; 14:412–417. [PubMed: 14677003]
122. Weber MA, et al. Local oestrogen for pelvic floor disorders: a systematic review. PLoS ONE. 2015; 10:e0136265. [PubMed: 26383760]
123. Cody JD, Richardson K, Moehrer B, Hextall A, Glazener CMA. Oestrogen therapy for urinary incontinence in post-menopausal women. Cochrane Database Syst Rev. 2009; 4:CD001405.
124. Novara G, et al. A systematic review and metaanalysis of randomized controlled trials with antimuscarinic drugs for overactive bladder. Eur Urol. 2008; 54:740–764. [PubMed: 18632201]
125. Chapple CR, et al. The effects of antimuscarinic treatments in overactive bladder: an update of a systematic review and meta-analysis. Eur Urol. 2008; 54:543–562. [PubMed: 18599186]
126. Kubota Y, et al. Correlation between improvements in overactive bladder symptom score and health-related quality of life questionnaires in overactive bladder patients treated with an antimuscarinic drug. Neurourol Urodyn. 2011; 30:1309–1314. [PubMed: 21560155]
127. Vouri SM, Kebodeaux CD, Stranges PM, Teshome BF. Adverse events and treatment discontinuations of antimuscarinics for the treatment of overactive bladder in older adults: a systematic review and meta-analysis. Arch Gerontol Geriatr. 2017; 69:77–96. [PubMed: 27889591]
128. Oefelein MG. Safety and tolerability profiles of anticholinergic agents used for the treatment of overactive bladder. Drug Saf. 2011; 34:733–754. [PubMed: 21830836]
129. Kessler TM, et al. Adverse event assessment of antimuscarinics for treating overactive bladder: a network meta-analytic approach. PLoS ONE. 2011; 6:e16718. A useful meta-analysis for choosing a drug for the treatment of overactive bladder based on tolerability, covering the majority of currently licensed preparations. [PubMed: 21373193]
130. Kim TH, Lee K-S. Persistence and compliance with medication management in the treatment of overactive bladder. Investig Clin Urol. 2016; 57:84–93.
131. Linnér L, Schiöler H, Samuelsson E, Milsom I, Nilsson F. Low persistence of anticholinergic drug use in Sweden. Eur J Clin Pharmacol. 2010; 67:535–536.
132. Yeaw J, Benner JS, Walt JG, Sian S, Smith DB. Comparing adherence and persistence across 6 chronic medication classes. J Manag Care Pharm. 2009; 15:728–740. [PubMed: 19954264]

133. Reynolds WS, et al. Comparative effectiveness of anticholinergic therapy for overactive bladder in women: a systematic review and meta-analysis. Obstet Gynecol. 2015; 125:1423–1432. [PubMed: 26000514]

134. Buser N, Ivic S, Kessler TM, Kessels AGH, Bachmann LM. Efficacy and adverse events of antimuscarinics for treating overactive bladder: network meta-analyses. Eur Urol. 2012; 62:1040–1060. [PubMed: 22999811]

135. Gray SL, et al. Cumulative use of strong anticholinergics and incident dementia: a prospective cohort study. JAMA Intern Med. 2015; 175:401–407. [PubMed: 25621434]

136. MacDiarmid S, et al. Mirabegron as add-on treatment to solifenacin in patients with incontinent overactive bladder and an inadequate response to solifenacin monotherapy. J Urol. 2016; 196:809–818. [PubMed: 27063854]

137. Drake MJ, et al. Efficacy and safety of mirabegron add-on therapy to solifenacin in incontinent overactive bladder patients with an inadequate response to initial 4-week solifenacin monotherapy: a randomised double-blind multicentre phase 3B study (BESIDE). Eur Urol. 2016; 70:136–145. [PubMed: 26965560]

138. Canadian Agency for Drugs and Technologies in Health. Common Drug Review. CADTH; 2015. https://www.ncbi.nlm.nih.gov/books/NBK349284/

139. Maman K, et al. Comparative efficacy and safety of medical treatments for the management of overactive bladder: a systematic literature review and mixed treatment comparison. Eur Urol. 2014; 65:755–765. [PubMed: 24275310]

140. Freemantle N, et al. Comparative assessment of onabotulinumtoxinA and mirabegron for overactive bladder: an indirect treatment comparison. BMJ Open. 2016; 6:e009122.

141. Pindoria N, et al. Persistence with mirabegron therapy for overactive bladder: a real life experience. Neurourol Urodyn. 2015; 36:404–408. [PubMed: 26669282]

142. Jost W, Marsalek P. Duloxetine: mechanism of action at the lower urinary tract and Onuf's nucleus. Clin Auton Res. 2004; 14:220–227. [PubMed: 15316838]

143. Li J, et al. The role of duloxetine in stress urinary incontinence: a systematic review and meta-analysis. Int Urol Nephrol. 2013; 45:679–686. [PubMed: 23504618]

144. Hunskaar S, Welle-Nilsen LK. The market lifecycle of duloxetine for urinary incontinence in Norway. Acta Obstet Gynecol Scand. 2010; 89:217–222. [PubMed: 20121337]

145. Ford AA, Rogerson L, Cody JD, Ogah J. Mid-urethral sling operations for stress urinary incontinence in women. Cochrane Database Syst Rev. 2015; 7:CD006375. The most recent Cochrane comparison of mid-urethral slings for stress urinary incontinence.

146. Latthe PM, Singh P, Foon R, Toozs-Hobson P. Two routes of transobturator tape procedures in stress urinary incontinence: a meta-analysis with direct and indirect comparison of randomized trials. BJU Int. 2009; 106:68–76. [PubMed: 19912182]

147. Novara G, et al. Updated systematic review and meta-analysis of the comparative data on colposuspensions, pubovaginal slings, and midurethral tapes in the surgical treatment of female stress urinary incontinence. Eur Urol. 2010; 58:218–238. [PubMed: 20434257]

148. Rehman H, Bezerra CC, Bruschini H, Cody JD. Traditional suburethral sling operations for urinary incontinence in women. Cochrane Database Syst Rev. 2011; 1:CD001754.

149. Lapitan MCM, Cody JD. Open retropubic colposuspension for urinary incontinence in women. Cochrane Database Syst Rev. 2012; 6:CD002912.

150. Parden AM, et al. Incontinence outcomes in women undergoing primary and repeat midurethral sling procedures. Obstet Gynecol. 2013; 121:273–278. [PubMed: 23344276]

151. Abdel-Fattah M, Cao G, Mostafa A. Long-term outcomes for transobturator tension-free vaginal tapes in women with urodynamic mixed urinary incontinence. Neurourol Urodyn. 2016; 36:902–908. [PubMed: 28028822]

152. Leong RK, de Wachter SGG, Joore MA, van Kerrebroeck PEV. Cost-effectiveness analysis of sacral neuromodulation and botulinum toxin A treatment for patients with idiopathic overactive bladder. BJU Int. 2010; 108:558–564. [PubMed: 21166750]

153. MacDiarmid SA, et al. Long-term durability of percutaneous tibial nerve stimulation for the treatment of overactive bladder. J Urol. 2010; 183:234–240. [PubMed: 19913821]

154. Peters KM, et al. Randomized trial of percutaneous tibial nerve stimulation versus extended-release tolterodine: results from the Overactive Bladder Innovative Therapy Trial. J Urol. 2009; 182:1055–1061. [PubMed: 19616802]

155. Amundsen CL, et al. OnabotulinumtoxinA versus sacral neuromodulation on refractory urgency urinary incontinence in women. JAMA. 2016; 316:1366–1374. [PubMed: 27701661]

156. Visco AG, et al. Anticholinergic therapy versus onabotulinumtoxinA for urgency urinary incontinence. N Engl J Med. 2012; 367:1803–1813. An important RCT that demonstrates the effectiveness of onabotulinumtoxinA for urgency urinary incontinence. [PubMed: 23036134]

157. Bartoli S, Aguzzi G, Tarricone R. Impact on quality of life of urinary incontinence and overactive bladder: a systematic literature review. Urology. 2010; 75:491–500. [PubMed: 19962738]

158. Monz B, et al. Patient characteristics associated with quality of life in European women seeking treatment for urinary incontinence: results from PURE. Eur Urol. 2007; 51:1073–1082. [PubMed: 17081676]

159. Haslam J. Urinary incontinence: why women do not ask for help. Nurs Times. 2005; 101:47–48.

160. Temml C, Haidinger G, Schmidbauer J, Schatzl G, Madersbacher S. Urinary incontinence in both sexes: prevalence rates and impact on quality of life and sexual life. Neurourol Urodyn. 2000; 19:259–271. [PubMed: 10797583]

161. de Vries HF, Northington GM, Bogner HR. Urinary incontinence (UI) and new psychological distress among community dwelling older adults. Arch Gerontol Geriatr. 2012; 55:49–54. [PubMed: 21601929]

162. Salonia A, et al. Sexual dysfunction is common in women with lower urinary tract symptoms and urinary incontinence: results of a cross-sectional study. Eur Urol. 2004; 45:642–648. [PubMed: 15082208]

163. Yip S-K, et al. The impact of urodynamic stress incontinence and detrusor overactivity on marital relationship and sexual function. Am J Obstet Gynecol. 2003; 188:1244–1248. [PubMed: 12748492]

164. Nygaard I. Idiopathic urgency urinary incontinence. N Engl J Med. 2010; 363:1156–1162. [PubMed: 20843250]

165. Gotoh M, et al. Impact of urinary incontinence on the psychological burden of family caregivers. Neurourol Urodyn. 2009; 28:492–496. [PubMed: 19090589]

166. Bekker MD, et al. Sexual experiences of men with incontinent partners. J Sex Med. 2010; 7:1877–1882. [PubMed: 20233280]

167. Nilsson M, Lalos O, Lindkvist H, Lalos A. Impact of female urinary incontinence and urgency on women's and their partners' sexual life. Neurourol Urodyn. 2011; 30:1276–1280. [PubMed: 21351129]

168. Kang Y, Phillips LR, Kim S-S. Incontinence quality of life among Korean-American women. Urol Nurs. 2010; 30:130–136. [PubMed: 20469572]

169. Perez LM, Webster GD. The history of urodynamics. Neurourol Urodyn. 1992; 11:1–21.

170. Sheng, W., Zhang, H., Ruth, K-H. Could urinary nerve growth factor be a biomarker for overactive bladder? A meta-analysis. Neurourol Urodyn. 2017. http://dx.doi.org/10.1002/nau.23210

171. Rachaneni S, et al. Bladder ultrasonography for diagnosing detrusor overactivity: test accuracy study and economic evaluation. Health Technol Assess. 2016; 20:1–150.

172. Richter HE, et al. Genetic contributions to urgency urinary incontinence in women. J Urol. 2015; 193:2020–2027. [PubMed: 25524241]

173. Cartwright R, et al. Identification of two novel genomic loci associated with stress and urgency urinary incontinence in a genome wide association study. Int Urogynecol J. 2015; 26:S53–S54.

174. Raz R, Stamm WE. A controlled trial of intravaginal estriol in postmenopausal women with recurrent urinary tract infections. N Engl J Med. 1993; 329:753–756. [PubMed: 8350884]

175. Brumbaugh AR, Mobley HLT. Preventing urinary tract infection: progress toward an effective *Escherichia coli* vaccine. Expert Rev Vaccines. 2012; 11:663–676. [PubMed: 22873125]

176. von Gontard A, Heron J, Joinson C. Family history of nocturnal enuresis and urinary incontinence: results from a large epidemiological study. J Urol. 2011; 185:2303–2307. [PubMed: 21511300]

177. Kuh D, Cardozo L, Hardy R. Urinary incontinence in middle aged women: childhood enuresis and other lifetime risk factors in a British prospective cohort. J Epidemiol Community Health. 1999; 53:453–458. [PubMed: 10562862]

178. Wilson D, Dornan J, Milsom I, Freeman R. UR-CHOICE: can we provide mothers-to-be with information about the risk of future pelvic floor dysfunction? Int Urogynecol J. 2014; 25:1449–1452. [PubMed: 24740445]

179. Hallock JL, Handa VL. The epidemiology of pelvic floor disorders and childbirth. Obstet Gynecol Clin North Am. 2016; 43:1–13. [PubMed: 26880504]

180. Hunskaar S, Lose G, Sykes D, Voss S. The prevalence of urinary incontinence in women in four European countries. BJU Int. 2004; 93:324–330. [PubMed: 14764130]

181. Ferroni MC, et al. Role of the brain stem in tibial inhibition of the micturition reflex in cats. Am J Physiol Renal Physiol. 2015; 309:F242–F250. [PubMed: 26017973]

182. Fowler CJ, Griffiths DJ. A decade of functional brain imaging applied to bladder control. Neurourol Urodyn. 2009; 29:49–55.

183. Birder L, Andersson K-E. Urothelial signaling. Physiol Rev. 2013; 93:653–680. [PubMed: 23589830]

184. Fatton B, de Tayrac R, Costa P. Stress urinary incontinence and LUTS in women — effects on sexual function. Nat Rev Urol. 2014; 11:565–578. [PubMed: 25201620]

185. Lukacz ES, et al. Epidemiology of prolapse and incontinence questionnaire: validation of a new epidemiologic survey. Int Urogynecol J. 2005; 16:272–284.

186. Twiss C, et al. Validating the Incontinence Symptom Severity Index: a self-assessment instrument for voiding symptom severity in women. J Urol. 2009; 182:2384–2391. [PubMed: 19758631]

187. Shaw C, et al. Validity and reliability of a questionnaire to measure the impact of lower urinary tract symptoms on quality of life: the Leicester Impact Scale. Neurourol Urodyn. 2004; 23:229–236. [PubMed: 15098218]

188. Abraham L, et al. Development and validation of a quality-of-life measure for men with nocturia. Urology. 2004; 63:481–486. [PubMed: 15028442]

189. Coyne KS, Thompson CL, Lai J-S, Sexton CC. An overactive bladder symptom and health-related quality of life short-form: validation of the OAB-q SF. Neurourol Urodyn. 2015; 34:255–263. [PubMed: 25783168]

190. Barber MD, Kuchibhatla MN, Pieper CF, Bump RC. Psychometric evaluation of 2 comprehensive condition-specific quality of life instruments for women with pelvic floor disorders. Am J Obstet Gynecol. 2001; 185:1388–1395. [PubMed: 11744914]

191. Barber MD, Walters MD, Bump RC. Short forms of two condition-specific quality-of-life questionnaires for women with pelvic floor disorders (PFDI-20 and PFIQ-7). Am J Obstet Gynecol. 2005; 193:103–113. [PubMed: 16021067]

192. Brown JS, et al. The sensitivity and specificity of a simple test to distinguish between urge and stress urinary incontinence. Ann Intern Med. 2006; 144:715–723. [PubMed: 16702587]

Box 1

Overactive bladder syndrome

Overactive bladder syndrome is formally defined as urinary urgency, with or without urgency incontinence, usually with urinary frequency and nocturia (the need to wake and pass urine at night), in the absence of a urinary tract infection or other obvious pathology. Given that urgency incontinence by definition occurs with urgency, having excluded other pathology or infection, the presence of urgency incontinence is sufficient, but not necessary, for a diagnosis of overactive bladder syndrome. Currently available therapies used for urgency incontinence typically receive licences for the broader indication of overactive bladder. However, the two terms are not synonymous.

Box 2

Factors assessed during medical history

Fluid intake

- The amount and types of fluid (particularly caffeinated, alcoholic and carbonated)

Urinary frequency

- Increased frequency is typically >7 micturition episodes during waking hours

Nocturia

- Interruption of sleep at least once because of the need to micturate; each void is preceded and followed by sleep

Urinary urgency

- Sudden compelling desire to pass urine that is difficult to defer (as opposed to urge, which is considered normal)

Bladder sensation during filling

- Increased, reduced or absent

Urinary stream

- Delay in initiating micturition (hesitancy), a slower stream than expected, intermittent stream that stops and starts on more than one occasion, urinary stream that splits or sprays instead of a single discrete stream

Straining

- The need to abdominally strain, or provide suprapubic pressure to initiate, maintain or improve stream

Incomplete emptying

- Bladder does not feel empty after micturition

Need to immediately re-void

- Further micturition is necessary soon after passing urine

Position-dependent voiding

- The need to take specific positions to improve emptying

Urinary retention

- Inability to pass urine despite persistent effort

Dysuria

- Lower urinary tract or vulval burning or discomfort with micturition

Lower urinary tract pain

- Suprapubic or retropubic pain, pressure or discomfort in the bladder that increases with filling that persists or is relieved after voiding
- Pain in the urethra, vulva, vagina, perineum or pelvis

Lower urinary tract infection

- Microbiological evidence of pathological bacteriuria with bladder storage symptoms or pain

Recurrent urinary tract infection

- At least three symptomatic, medically diagnosed urinary tract infections in the previous 12 months with evidence of resolution between episodes

Box 3

Common medications that can cause urinary incontinence

α-Adrenergic agonists

- Increase smooth muscle tone in the urethra
- Can precipitate urinary retention and related symptoms

α-Adrenergic antagonists

- Decrease smooth muscle tone in the urethra
- Can precipitate stress urinary incontinence

Angiotensin-converting enzyme inhibitors

- Cause a cough that can exacerbate stress urinary incontinence

Anticholinergics

- Can cause impaired emptying, urinary retention and constipation that can contribute to urinary incontinence
- Can cause cognitive impairment and reduce effective toileting ability

Calcium channel blockers

- Can cause impaired emptying, urinary retention and constipation that can contribute to urinary incontinence
- Can cause dependent oedema that can contribute to nocturnal polyuria

Cholinesterase inhibitors

- Increase bladder contractility
- Can precipitate urgency urinary incontinence

Diuretics

- Cause diuresis and precipitate urinary incontinence

Lithium

- Causes polyuria via induced diabetes insipidus

Opioid analgesics

- Can cause urinary retention, constipation, confusion and immobility, all of which can contribute to urinary incontinence

Psychotropic drugs, sedatives, hypnotics, antipsychotics and histamine 1 receptor antagonists

- Can cause confusion and impaired mobility, and precipitate urinary incontinence

Selective serotonin reuptake inhibitors

- Increased cholinergic transmission can lead to urgency urinary incontinence

Gabapentin, glitazones and NSAIDs

- Can cause oedema, which can lead to nocturnal polyuria causing nocturia and nocturnal enuresis

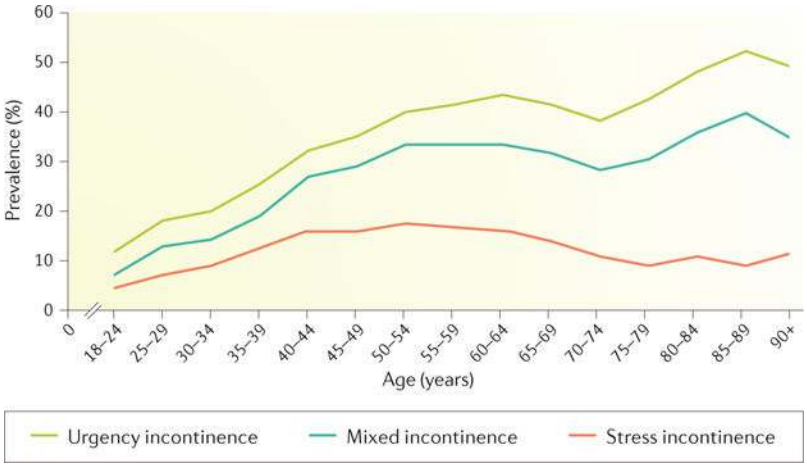

Figure 1. Prevalence of stress, urgency and mixed incontinence stratified by age

The prevalence of stress incontinence peaks in the fifth decade and then declines, whereas the prevalence of both mixed and urgency incontinence continues to increase with age. Observational data are from France, Germany, Spain and the United Kingdom[180]. Median prevalence data from a review of epidemiological studies from around the world have shown similar trends[22]. Reproduced with permission from REF.[180], Wiley.

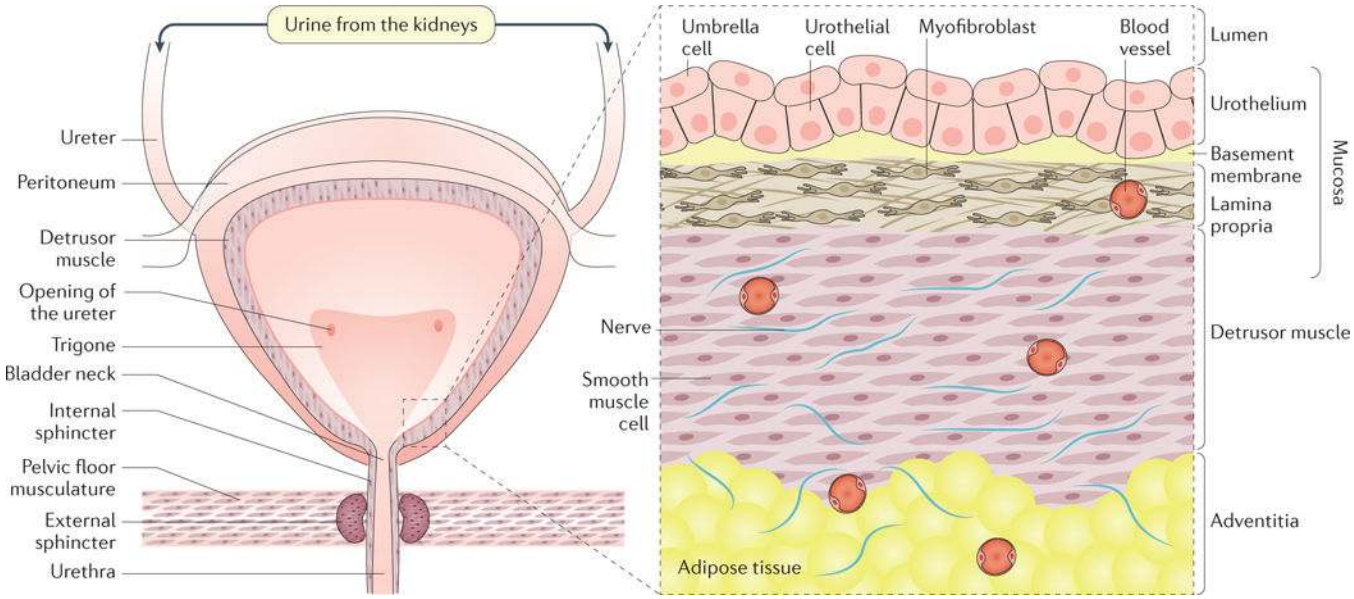

Figure 2. Anatomy and histology of the female bladder

The bladder lies immediately behind the pubic bones. When empty, the bladder has a pyramidal shape. As it fills and distends, the bladder balloons up above the pubic bones in an ovoid shape. The muscle of the bladder wall (the detrusor) consists of interdigitating fibres of smooth muscle, arranged in circular and longitudinal layers. These can stretch up to four times their resting length, so there is no increase in linear tension (or pressure) during normal bladder filling. The bladder and the ureters are both lined by a transitional epithelium, the urothelium. It contains flattened (‘umbrella’) cells and cuboidal cells, which also enable stretch as the bladder fills. The base of the bladder is a triagonal area, called the trigone. The ureters enter at the two superior corners of this triangle and the bladder neck lies at the inferior corner. The bladder neck is in continuity with the urethra, which, in women, is 2.5–4 cm long. The internal sphincter is formed of rings of smooth muscle at the bladder neck, whereas the external sphincter is formed by the muscles of the pelvic floor. Both sphincters help to close off the urethra to maintain continence.

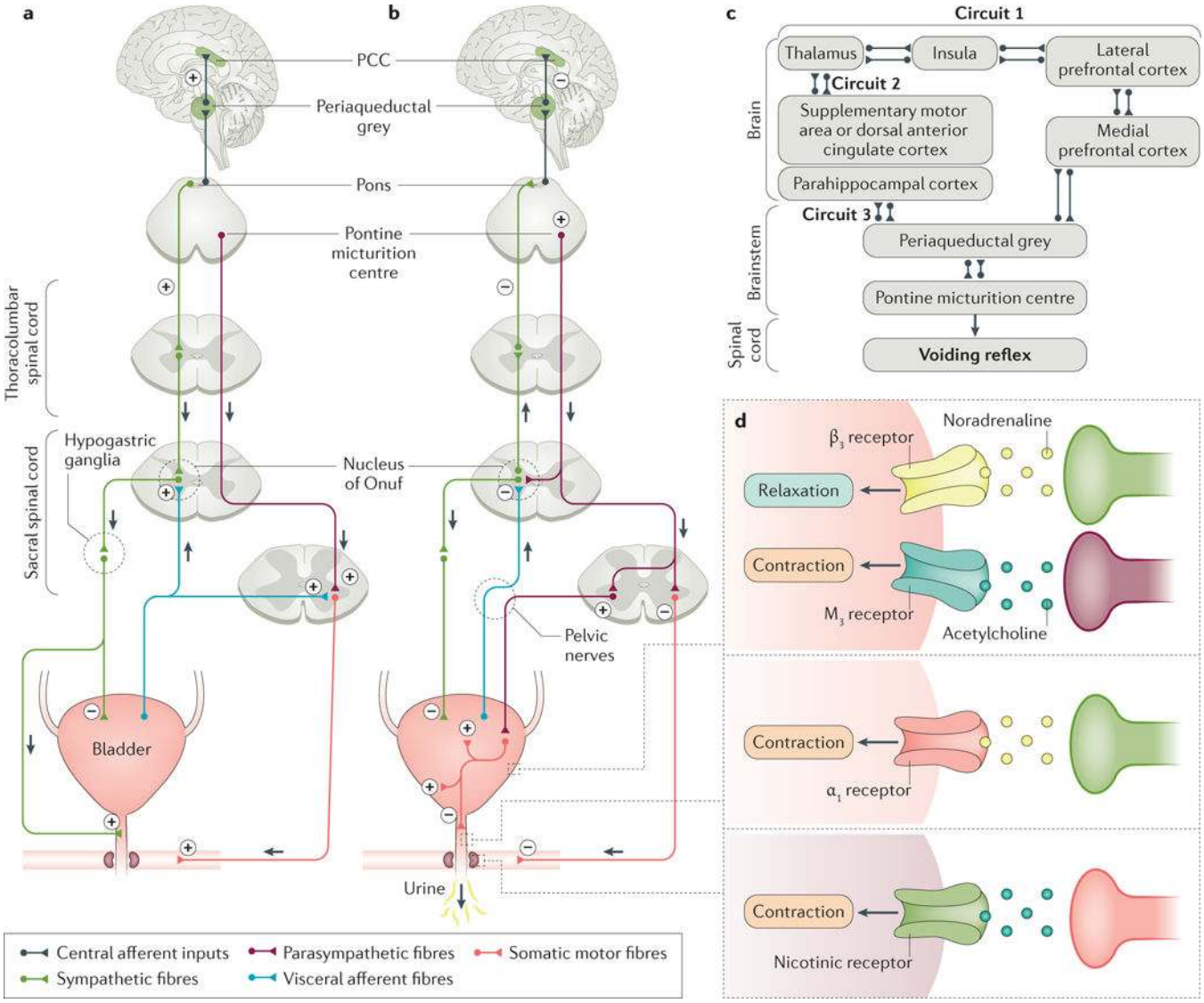

Figure 3. Neurological control of the urinary bladder

a | The sympathetic system predominates during the storage phase and maintains continence through the paravertebral ganglia and the hypogastric nerves and plexus. **b** | The parasympathetic system coordinates the voiding phase, through the sacral plexus and pelvic nerves (S2–S4)[39]. Afferent signals come from the urothelium and the bladder wall, through the pelvic nerves, and then go to the dorsal root ganglia and are projected to the periaqueductal grey, then to the posterior cingulate cortex (PCC). **c** | The main circuits for regulating desire to void include the insula and the lateral and medial prefrontal cortices, which feed back to the periaqueductal grey[18,182]. The periaqueductal grey serves as a relay station for bladder information and activates the pontine micturition centre, which contracts the bladder and relaxes the urethral sphincter mechanism during voiding. **d** | The neurotransmitters responsible for the execution of these commands are acetylcholine and noradrenaline.

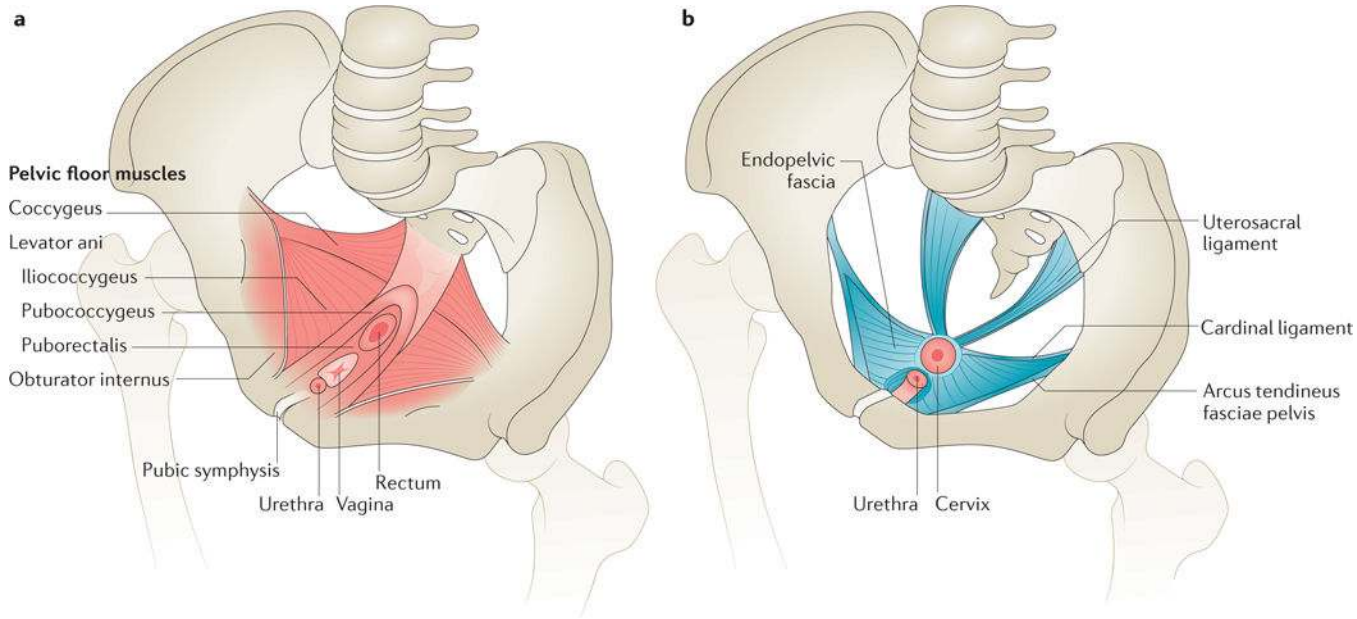

Figure 4. Urethral support

The posterior urethra lies on a supportive tissue layer composed of the anterior vaginal wall (part **a**) and the endopelvic fascia (part **b**). These structures are suspended from the arcus tendineus and in combination with a functional levator ani, create a 'hammock' that results in compression of the urethra with increased intra-abdominal pressure, preventing urinary leakage[41].

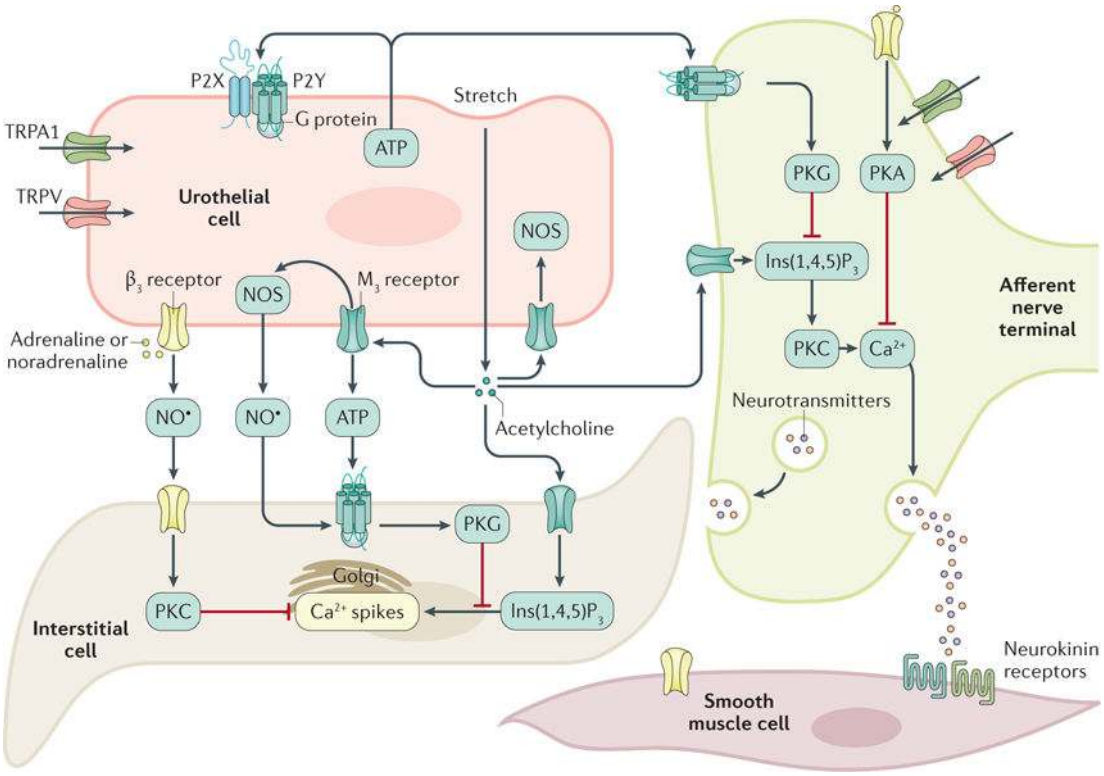

Figure 5. Uroepithelial sensory web

In the urotheliogenic hypothesis of overactive bladder syndrome (BOX 1), urothelial cells are targets for neurotransmitters released from nerves, are targets for signals from other cell types and can be activated by autocrine or paracrine mechanisms. The signalling cascades between bladder nerves, urothelial cells, smooth muscle cells, interstitial cells and blood vessels are mediated by noradrenaline and adrenaline (via the β_3-adrenergic receptors), acetylcholine (via the muscarinic M_3 receptors), Ca^{2+} (via the activity of transient receptor potential cation channel subfamily A member 1 (TRPA1) and transient receptor potential cation channel subfamily V (TRPV) channels) and ATP (via the purinoceptors P2X and the purinergic G protein-coupled receptors P2Y). $Ins(1,4,5)P_3$, inositol 1,4,5-trisphosphate; PKA, protein kinase A; PKC, protein kinase C; PKG, protein kinase G; NO•, nitric oxide; NOS, NO synthase. Adapted with permission from REF 183, American Physiological Society.

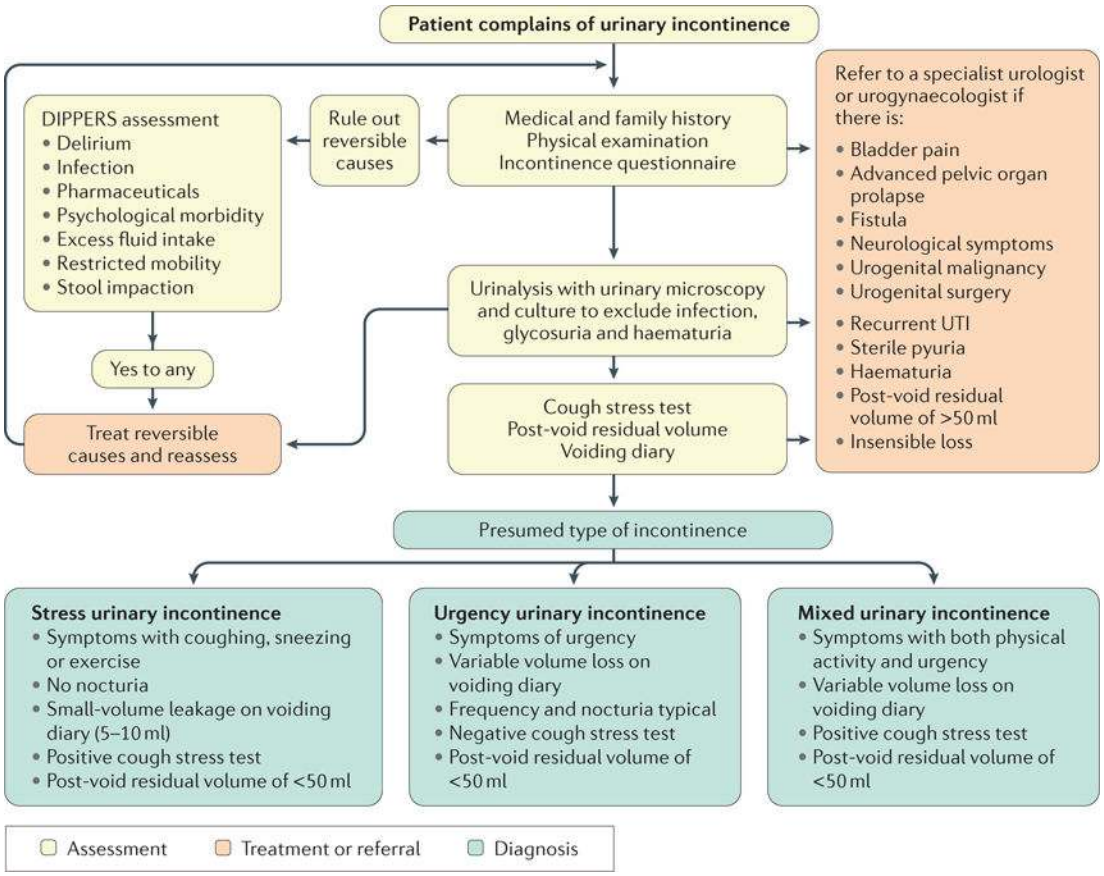

Figure 6. Diagnostic work-up of women with urinary incontinence

The initial management of urinary incontinence includes a detailed history and physical examination to identify potential reversible causes of symptoms, followed by urinalysis with microscopy, voiding diary, assessment of post-void residual volume and cough stress test to assist with diagnosis and initial management. In cases of advanced pelvic organ prolapse, prior pelvic surgery, haematuria or urinary retention, patients may be referred to a urologist or urogynaecologist. UTI, urinary tract infection.

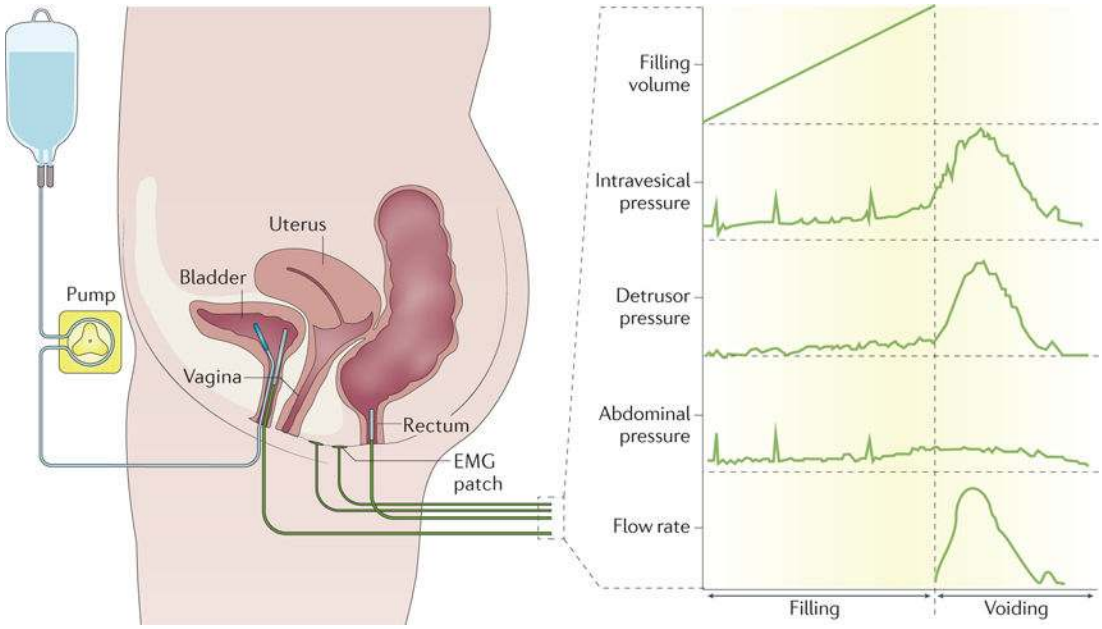

Figure 7. Multichannel urodynamic testing

Invasive (catheterized) pressure measurements during urodynamic studies include intravesical pressure (using a probe in the bladder) and abdominal pressure (using a probe in the rectum as shown or in the vagina (not shown)). In addition, electromyography (EMG) can be used to evaluate the activity of the muscles of the pelvic floor. During the test, the bladder is filled and then the patient is asked to void, with continuous pressure monitoring during filling and emptying. In women with urgency incontinence, findings can include uninhibited contraction of the detrusor muscle during filling (detrusor overactivity) or a gradual uncomfortable rise in pressure during filling (low compliance). A formal diagnosis of stress incontinence is made by observation of leakage with coughing or exertion in the absence of detrusor contraction.

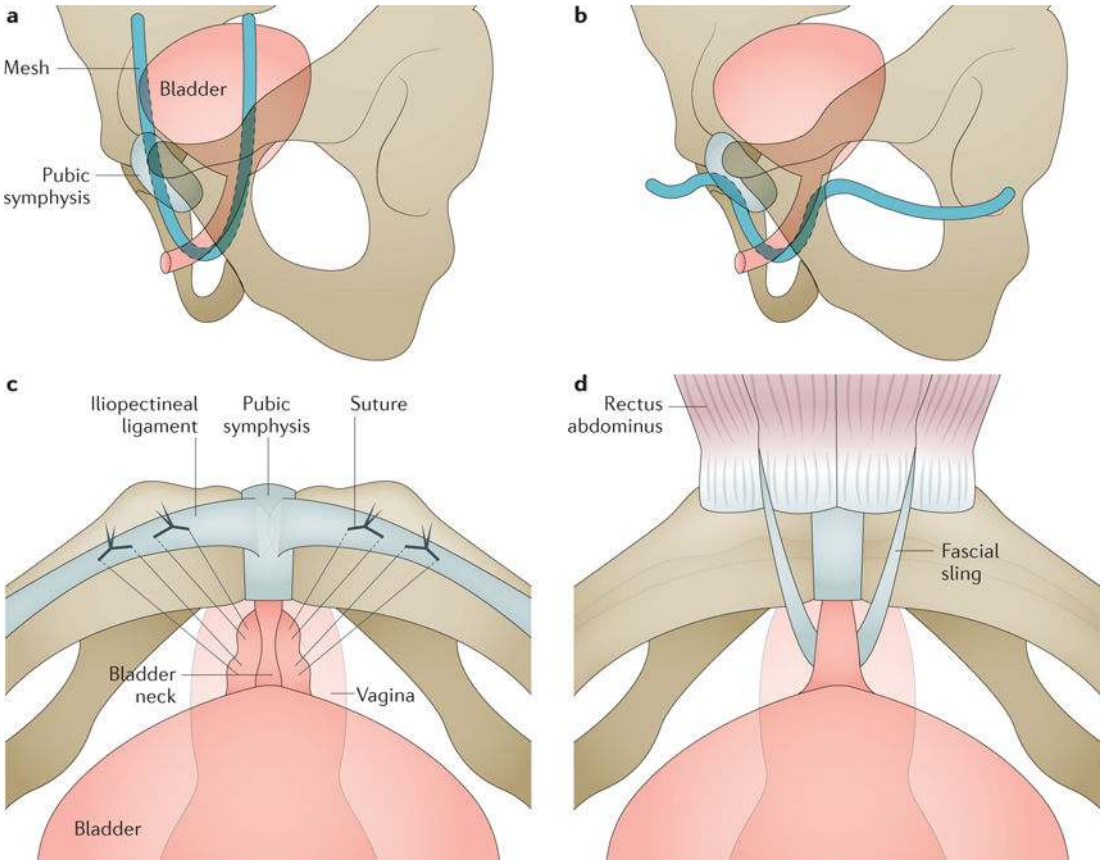

Figure 8. Surgical treatment for urinary incontinence

Surgical correction of urethral hypermobility, which results from loss of support of the bladder neck and proximal urethra such that they move during peaks of abdominal pressure, can involve the use of a synthetic mesh, suture or autologous tissue. A synthetic mesh is placed inside the vagina at the level of the mid-urethra and is passed either retropubically (part **a**) or via the transobturator approach (part **b**). Sutures are not used in either of these 'tension-free' procedures; the body tissues and fibrosis hold the mesh in place. Conversely, retropubic urethropexy (part **c**) involves the placement of permanent sutures in the anterior vaginal wall at the level of the bladder neck and proximal urethra. Finally, autologous fascial sling placement (part **d**) involves harvesting a strip of rectus fascia that is placed beneath the proximal urethra through a vaginal incision; the two ends of the sling are passed behind the pubic bone and are secured with permanent sutures either to each other or to the rectus fascia. Part **a** and part **b** are reproduced with permission from REF 184, Macmillan Publishers Limited.

Table 1

Drugs licensed for urgency urinary incontinence

Class	Medications	Common adverse effects
Non-selective antimuscarinic	• Fesoterodine fumarate • Oxybutynin chloride * • Oxybutynin transdermal patch • Oxybutynin gel • Tolterodine tartrate * • Trospium chloride *	• Dry mouth • Blurred vision • Constipation • Impaired cognition • Impaired memory
Selective M_3 antimuscarinic	• Darifenacin • Imidafenacin‡ • Solifenacin succinate	• Dry mouth • Dry eyes • Constipation • Blurred vision
β_3-adrenergic agonist	Mirabegron	• Headache • Dizziness • Dry mouth • Hypertension

* Immediate and extended release.

‡ Currently licensed only in Japan, Thailand, the Philippines and South Korea.

Table 2

LUTS and urinary incontinence questionnaires for use in women

Questionnaire	Items	Symptom severity	Quality of life	Source or refs
EPIQ	49	Yes	Yes	See appendix of REF.[185]
ICIQ-FLUTS Module	12	Yes	Yes	www.iciq.net/ICIO.FIUTS.html
ICIQ-UI Structure Short Form	4	Yes	Yes	www.iciq.net/ICIO-UIshortform.html
Incontinence Impact Questionnaire Long form *	30	Yes	Yes	www.wakehealth.edu/School/OWIMS/NO-and-UDI-Instrument.htm
Urinary Incontinence Quality of Life Scale (I-QOL)	22	No	Yes	http://depts.washinnton.edu/seaqol/IOOI
Incontinence Symptom Severity Index	8	Yes	Yes	See appendix of REF.[186]
King's Health Questionnaire	21	Yes	Yes	https://www.nice.ora.uk/auidance/ca171/resources/the-kinns-health-questionnaire-pdf-191574685
The Leicester Impact Scale	21	No	Yes	See appendix of REF.[187]
Nocturia Quality of Life Questionnaire	13	No	Yes	See appendix of REF.[188]
OAB-q Short Form	19	Yes	Yes	See appendix of REF.[189]
ICIQ-OABqol	33	Yes	Yes	www.iciq.net/ICIO-OABqolmodulepane.html
Pelvic Floor Distress Inventory	46‡	No	Yes	Via authors of REF.[190]
Pelvic Floor Distress Inventory Short Form	20§	No	Yes	[191]
Pelvic Floor Impact Questionnaire	93//	No	Yes	Via authors of REF.[190]
Pelvic Floor Impact Questionnaire Short Form	21//	No	Yes	[191]
The 3 incontinence questions#	3	No	No	REF[192] and http://www.racap.ora.au/your-practice/auidelines/redbook/appendices/appendix-13a-the-3-incontinence-questions-(3iq)/

EPIQ, Epidemiology of Prolapse and Incontinence Questionnaire; FLUTS, female lower urinary tract symptoms; ICIQ, International Consultation on Incontinence Modular Questionnaire; LUTS, lower urinary tract symptoms; OAB, overactive bladder syndrome; qol, quality of life; UI, urinary incontinence.

*Also available in a short form (7 items) that looks at quality of life only.

‡Of which 28 are in the urinary domain.

§Of which 6 are in the urinary domain.

[//] Of which 31 are in the urinary domain.

[¶] Of which 7 are in the urinary domain.

[#] Differentiates stress, urgency and mixed incontinence for clinical care.