

Epidemiology

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Urinary incontinence is highly prevalent, affecting 25–45% of men and women, and this increases with age. In men, the most common cause of SUI is radical prostatectomy for prostate cancer, with prevalence estimates of 5% at 24–36 months after surgery. Continence is primarily dependent on normal bladder compliance, an intact urethral sphincter, strong urethral support by the pelvic floor and a leakproof mucosal seal. Compliance is the ability of the bladder to expand in volume without any significant rise in pressure, and during the normal storage phase the bladder pressure remains low until maximum capacity is reached. This enables normal renal drainage and is dependent on the viscoelastic properties of the bladder wall (low collagen levels). However, detrusor overactivity during the storage phase, bladder muscle hypertrophy or increased levels of bladder wall collagen (e.g. due to fibrosis) can all reduce compliance and lead to incontinence and deterioration in renal drainage. This may occur as a result of pelvic surgery, irradiation, neurological conditions, chronic inflammatory bladder conditions leading to bladder fibrosis or longstanding bladder outlet obstruction. Deficiencies in the active urethral sphincter mechanism, the urethral mucosal seal and the pelvic floor support contribute to varying degrees of SUI. Hypermobility of the bladder base and proximal urethra due to laxity of the usual supporting ‘hammock’, consisting of endopelvic and pubocervical fascia attached to the ATFP and levator ani, is thought to lead to displacement of the urethra out of the pelvis. As a result, during stress manoeuvres the raised intra-abdominal pressure is not transmitted to the urethra and so incontinence occurs. Laxity of the vaginal wall and pubourethral ligaments is thought to contribute to this deficiency in urethral support. These theories are the basis for retropubic suspension and mid-urethral sling procedures to treat SUI. Intrinsic sphincter deficiency occurs when the normal submucosal vascularity of the urethra and sphincter muscle tone are deficient. This may occur as a result of previous surgery, causing fibrosis, irradiation and nerve injury, or loss of oestrogenisation. Hypermobility and intrinsic sphincter deficiency exist on a spectrum and most women with SUI have elements of both.

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TABLE 83.5 Predisposing and exacerbating factors for urinary incontinence (UI)

Predisposing	Exacerbating
Familial (increased risk in those with family history of UI)	Increased intra-abdominal pressure (chronic cough, Congenital or acquired straining due to constipation, anatomical abnormalities (e.g. exercise) ectopic ureter, urinary tract infection, stulae, urethral

diverticulum) Cognitive impairment Neurological conditions (e.g. Restricted mobility spinal cord injury, Parkinson's disease, Drugs (e.g. diuretics) stroke, multiple sclerosis) Menopause causing atrophic vaginitis Pregnancy and childbirth Pelvic surgery Fluid intake (e.g. excess caffeine) Pelvic radiotherapy Chronic inflammatory conditions resulting in bladder fibrosis (tuberculous cystitis, ketamine cystitis, interstitial cystitis)

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