# Female urinary incontinence: aetiology and pathophysiology

*Robert M Freeman*

***Genuine stress incontinence and detrusor instability are the main causes of female urinary incontinence. Treatments are not always satisfactory and a better understanding of the aetiology and pathophysiology should result in more successful therapies and aid prevention.***

Urinary incontinence is a common problem. It is estimated to affect 2–3 million individuals in the UK. The aetiology and pathophysiology have not been well understood, which might explain why so many treatments fail or have unacceptable side-effects or complications. A better understanding might help in treatment and prevention.

Continence is maintained by a series of complex neurological mechanisms, which allows the pressure in the urethra to exceed the pressure in the bladder. If the converse occurs, incontinence results: genuine stress incontinence (GSI) caused by a relative reduction in urethral pressure, and overactive bladder (detrusor instability) from increased bladder pressure. Bladder pressure can rise indirectly as a result of increasing abdominal pressure with obesity or chronic straining, e.g. constipation or coughing.

## GENUINE STRESS INCONTINENCE

GSI is a urodynamically diagnosed condition in which the pressure in the bladder exceeds the pressure in the urethra because of the intra-abdominal pressure rise rather than a detrusor contraction. This may arise because the bladder neck is excessively mobile (urethral hypermobility) and/or the sphincteric mechanism is poorly functioning (intrinsic sphincter deficiency).

### Urethral hypermobility

When the bladder neck and urethra are in an intrapelvic position, with a cough, the abdominal pressure is transmitted equally to the bladder neck, distal sphincter, urethra and to the bladder itself (*Figure 1*). Here, the pressure rise in the urethra occurs 250 ms before the rise in intravesical pressure (Constantinou and Govan, 1982). Continence is therefore maintained.

If the anatomical supports are inadequate, the bladder neck and urethra descend below the level of the pelvic floor, the pressure transmission to the urethra does not occur, nor does the resultant pressure rise. The intravesical pressure therefore exceeds the intraurethral pressure, resulting in GSI. This is the 'pressure transmission theory' (Enhorning, 1961).

This theory has had a major influence on the management of women with GSI, for example by prevention of bladder neck descent, by pelvic floor exercises or repositioning of the bladder neck and urethra with surgery.

The pressure transmission theory has been questioned as, anatomically, the bladder neck and urethra do not always lie in an intrapelvic position. For example, many patients with large cystourethrocoeles have the bladder neck and urethra below the level of the pelvic floor, yet are continent (De Lancey, 1994).

Nonetheless, hypermobility does appear to be a cause of GSI. To understand how this occurs, a knowledge of the anatomical supports is useful.

### Anatomical considerations

A 'hammock hypothesis' has been proposed for bladder neck and urethral support (De Lancey, 1994) (*Figure 2*). The bladder neck and urethra are thought to lie on a hammock-like structure

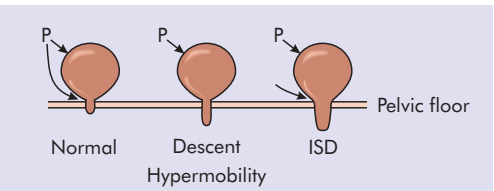


*Figure 1. Pressure transmission theory, hypermobility and intrinsic sphincter deficiency (ISD).*

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composed of endopelvic fascia and anterior vaginal wall. The endopelvic fascia is draped over the arcus tendinius fascia pelvis (ATFP). Also attached to this are the levator ani (*Figure 3*).

When an individual coughs, the hammock is tightened to elevate the bladder neck and urethra and to resist the effects of increasing intra-abdominal pressure. With contraction of the levators the hammock is straightened, the bladder neck and urethra elevated, supported and compressed.

Hypermobility may occur therefore because of defects in the endopelvic fascia, the levator ani or its nerve supply.

**Endopelvic fascia**

It has been shown that the collagen content of the endopelvic fascia in patients with GSI and prolapse is reduced and weakened (Jackson et al, 1996). This reduction in collagen might be congenital. It has been shown that, in premenopausal nulliparous women with GSI, collagen in the anterior vaginal wall is reduced and weakened compared with controls (Keane et al, 1997). This would suggest that there is a group of women with inherently weak endopelvic fascia, who might be at risk of developing GSI with ageing or earlier as a result of the trauma of childbirth.

Using perineal ultrasound, 30% of primigravidae have been shown to have excessive bladder neck mobility antenatally. These women appear to be at higher risk of developing postnatal stress incontinence (King and Freeman, 1998). It is possible that the endopelvic fascia is inherently weak in these individuals, producing bladder neck mobility.

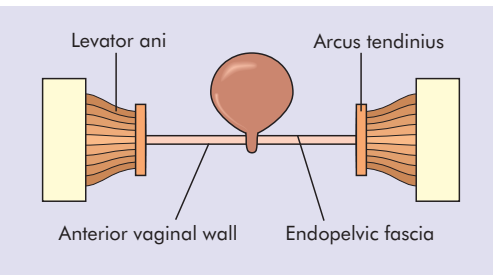


*Figure 2. Hammock theory.*

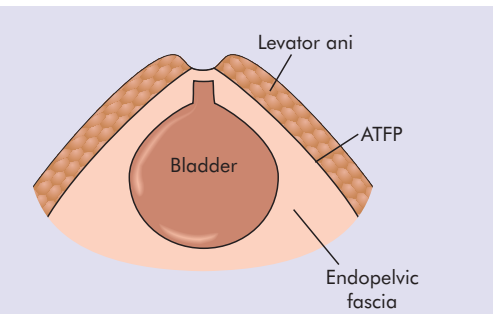


*Figure 3. Anatomical bladder neck and urethral support. ATFP = arcus tendinius fascia pelvis.*

**Levator ani**

The 'hammock' can also be affected by damage to the levator ani muscles. Objective evidence, using ultrasound and perineometry, has confirmed the long-held view that vaginal delivery can damage the levators (Peschers et al, 1997).

The main factor associated with muscle weakness as a result of delivery appears to be neuromuscular damage, i.e. pudendal nerve injury (Snooks et al, 1984). Prolonged pudendal nerve terminal motor latencies have been seen in women with postnatal urinary and faecal incontinence when compared with antenatal values (Sultan et al, 1994). The factors associated with prolonged pudendal nerve latencies include increased birth weight and longer second stages of labour.

The role of instrumental delivery in neuromuscular damage is unclear. Some studies suggest that forceps are implicated (Snooks et al, 1984) while others have failed to show any association (Sultan et al, 1994).

**Pregnancy and vaginal delivery**

It would appear that vaginal delivery plays an important part in the pathogenesis of GSI. Are the defects in the endopelvic fascia or the levator ani, or are they neuromuscular? Magnetic resonance imaging has suggested that all are implicated, as judged by thinning of the levators and tears in the endopelvic fascia after delivery (De Lancey et al, 1996).

It is possible that patients with inherently defective collagen in the endopelvic fascia are at higher risk of developing postnatal stress incontinence. It might be that obstetric factors, e.g. large babies or long second stages, only become risk factors in these patients.

Pregnancy itself might have an effect on the development of postnatal incontinence. It is rare for patients to complain of stress incontinence for the first time after delivery (Dimpfl et al, 1992). Antenatal factors are probably important. Hormonal factors might affect supporting structures as well as the distal urethral sphincter. For example, reduced sphincter volume (as seen on three-dimensional ultrasound) seems to be a risk factor for postnatal stress incontinence (Toozs-Hobson et al, 1998).

Prevention by identifying at-risk groups might be possible. Whether caesarean section is appropriate is unclear and, at present, there are no prospective long-term follow-up data to answer this question.

## INTRINSIC SPHINCTER DEFICIENCY

When failure of the urethral closure mechanism rather than bladder neck mobility is the cause of GSI, the condition is known as intrinsic sphincter deficiency (ISD). This has been defined as a maximum urethral closure pressure less than 20 $cmH_2O$ (Richardson et al, 1995) or a Valsalva leak point pressure of less than 60 $cmH_2O$ (McGuire et al, 1993).

Functionally, the urethra stays closed at four levels: the bladder neck, the distal sphincter (intramural striated muscle), the hermetic seal and extrinsic compressive forces.

### Bladder neck

This is composed of elastic fibres, a small quantity of smooth muscle and collagen. Closure is passive. Opening is by traction following detrusor contraction. In 20% of nulliparous, continent women, the bladder neck can be open at rest (Chapple et al, 1989). These individuals probably maintain continence at the distal sphincter.

### The distal sphincter

Anatomically and functionally, the distal sphincter (rhabdosphincter) appears to be the main area for urethral closure (Gosling et al, 1981). The nerve supply is via the sympathetic (T11/L2) and the somatic system via the pudendal nerve. Recently, other nerves have been found running underneath the trigone which also supply the distal sphincter (Borirakchanyavat et al, 1997). These could potentially be damaged during surgery (Zivkovic et al, 1996).

### Hermetic seal

Anatomically, the urethral mucosa is thrown into folds. The sub-mucosa has a rich network of veins, elastic fibres and collagen, producing a spongy intraurethral tissue, which fills the mucosal folds and allows apposition, resulting in sealing. This and extrinsic compressive forces (the periurethral striated muscle, anterior vaginal wall and possibly cervix and rectum) aid closure.

It has been shown that both the hermetic seal and extrinsic forces can be affected by ageing (Rud et al, 1980). The menopause, therefore, either as a result of reduced vascularity or reduced collagen, might affect urethral closure.

## THERAPEUTIC IMPLICATIONS

### Intrinsic sphincter deficiency

It has been suggested that the failure rate from colposuspension in patients with ISD is high (Sand et al, 1987), although this has not been confirmed in other studies (Richardson et al, 1991). In the USA slings have become popular as a treatment for ISD, but complication rates can be high (Leach et al, 1997).

An alternative to slings is urethral injectables, e.g. collagen, silastic. One report using collagen in patients with ISD has quoted a success rate of 83% at 4 years (Richardson et al, 1995).

Hormone replacement therapy (HRT) might be expected to have a therapeutic effect in ISD. However, the evidence suggests that neither subjective nor objective changes occur (Fantl et al, 1996; Jackson et al, 1999).

### Hypermobility

If the levators are weak, but neurologically intact, then pelvic floor exercises are indicated. If the pelvic floor is denervated it is questionable whether these will be effective (De Lancey, 1996).

The traditional surgical aims have been to elevate the bladder neck with colposuspension or needle suspension, or to support it using a sling. Excessive elevation and/or urethral compression has been shown to be responsible for the complications of voiding difficulty and detrusor instability (Bombieri and Freeman, 1997, 1998a).

Correction of the anatomical defect (i.e. paravaginal defect) with a paravaginal repair might result in fewer complications. One study reported a success rate of 90% with minimal complications (Schull and Baden, 1989). However, this has not been confirmed in other studies (Colombo et al, 1996). A large randomized trial comparing these procedures is required.

## GSI AFTER GYNAECOLOGICAL SURGERY

Compressive forces from the anterior vaginal wall, cervix and rectum are thought to transmit abdominal pressure to the bladder neck and urethra. Surgery to the posterior structures can result in GSI, for example, 6 years after hysterectomy, the risk of developing stress incontinence has been reported as 15%. If stress incontinence had been present before the hysterectomy, the chances of this worsening were 36% (Brown et al, 1994).

Following vaginal prolapse surgery, de novo incontinence has been reported in up to 22% of patients (Bump et al, 1996) and after abdominal sacrocolpopexy in 29% (Snyder and Krantz, 1991). Whether this is caused by alteration of pressure transmission through the posterior structures is unclear. Denervation related to dissection could also be responsible (Zivkovic et al, 1996).

The pressure transmission from posterior structures is thought to be important in the mechanism of continence following colposuspension (Hertogs and Stanton, 1985). Here, pos-

terior repairs and vault suspensions might result in recurrence of incontinence despite a previously successful colposuspension. One report has highlighted the problem and suggests measures that can be taken to prevent this worrying complication (Bombieri and Freeman, 1998b).

It is recommended that patients are advised of potential incontinence after gynaecological surgery. It is important to identify such patients with appropriate preoperative investigation and, if possible, to prevent postoperative incontinence by appropriate prophylactic measures (e.g. additional anti-incontinence surgery in those at risk).

## OVERACTIVE BLADDER/ DETRUSOR INSTABILITY

The International Continence Society has defined this as a condition in which the detrusor is shown objectively to contract spontaneously on provocation during bladder filling while the patient is attempting to inhibit micturition (Abrams et al, 1988).

When the cause is a neuropathy the term used is detrusor hyperreflexia. Recently, a new classification has been proposed to enable patients to understand overactive bladder, as outlined in *Figure 4* (Hample et al, 1997).

### Detrusor hyperreflexia

This may arise from damage to the central inhibitory pathways in the brain or by increased sensitivity of the bladder afferents as seen in spinal cord disease (De Groat, 1997). In spinal cord disease primitive reflexes appear which are triggered by C-fibre afferents. These are supersensitive and can lead to reflex incontinence in the absence of sensation.

It has been shown that capsaicin, a potent C-fibre afferent endotoxin, is effective both subjectively and objectively in detrusor hyperreflexia resulting from spinal cord disease (Fowler et al, 1994). This effect can persist, without complications, for between 3 and 5 years in 80% of patients (De Ridder et al, 1997).

### Psychological factors

Failure of higher centre inhibition can arise as a result of psychological factors.

Support for a psychosomatic hypothesis for primary unstable bladder comes from psychological and treatment evidence. It has long been known that the psyche can affect the bladder (Straub et al, 1949). Psychological questionnaire studies have been inconclusive, with some suggesting high levels of psychological morbidity while others have failed to show any significant differences from controls.

Support for psychological factors comes from clinical features, reports of sexual abuse and the results of psychologically orientated treatments, such as bladder retraining, biofeedback, hypnotherapy and psychotherapy (Freeman, 1989). In addition, a very high placebo response from drug trials suggests that the increased attention paid to the patient (itself a form of behavioural therapy) is relevant (Meyhoff et al, 1983). Attention to these factors is important in treatment.

### Neurotransmitters

The main neurotransmitter released following stimulation of the parasympathetic nerves is acetylcholine. This binds onto muscarinic receptors, leading to the release of calcium ions and a detrusor contraction. This should be blocked by atropine. However, it has been suggested that in idiopathic unstable bladder such blocking does not occur ('atropine resistance'). This might explain the often poor results from anticholinergic therapy. Alternative non-adrenergic/non-cholinergic neurotransmitters, e.g. vasoactive intestinal polypeptide (VIP) (Gu et al, 1983), substance P or nitric oxide, might be responsible. If the role of these neurotransmitters could be confirmed then drugs to counteract their effect might result in more successful drug treatment.

### Pelvic floor inhibition

There are inhibitory reflexes in the pelvic floor, e.g. the perineodetrusor inhibitory reflex (*Figure 5*). If stimulation of the pelvic floor fails to inhibit the detrusor, then increased efferent activity could result leading to detrusor contractions (Artibani, 1997). This hypothesis is based on experimental work where pudendal nerve stimulation has resulted in detrusor inhibition (Vodusek et al, 1988). Treatment with intravaginal electrical stimulation has shown short-term success rates of approximately 50% (Plevnik et al, 1989).

### Myogenic theory

It has been proposed that detrusor overactivity might not be the result of neurological reflex activ-

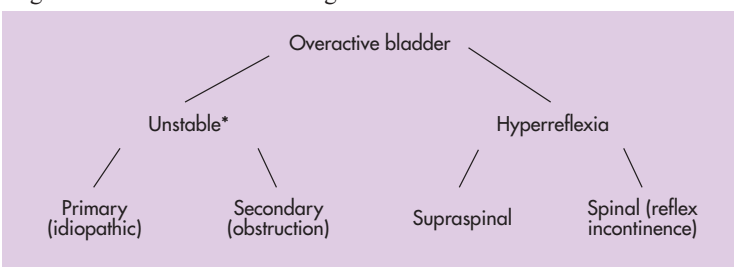


***Figure 4. Classification of overactive bladder. *'Unstable' can occur as a result of failure of higher centre inhibition, abnormality of the detrusor smooth muscle, obstruction, abnormality in neurotransmission or failure of pelvic floor inhibition.***

ity, but of a disorder of the detrusor smooth muscle itself. There might be a common mechanism for detrusor overactivity irrespective of aetiology, i.e. partial denervation, which alters the properties of the smooth muscle cell, leading to increased excitability, coordinated contraction of the whole detrusor and detrusor overactivity (Brading and Turner, 1994). Potassium channel-opening drugs (e.g. pinacidil) have more effect on unstable detrusor muscle than normal muscle in vitro. However, clinical effects have not been seen.

**Obstruction**

It has been thought that partial denervation might result from ischaemia secondary to obstruction (Sibley, 1997). While there is debate as to whether obstruction causes detrusor overactivity, obstructive muscle has been shown to have increased sensitivity to acetylcholine.

In the female obstruction is not thought to be a common cause of overactivity. However, functional obstruction might result from infrequent voiding and subsequent sphincter hypertrophy. As yet, there is no evidence that this produces bladder overactivity.

**Post-colposuspension**

Detrusor overactivity can occur in 12% of patients following colposuspension for GSI (Jarvis, 1994). This might be caused by obstruction secondary to over-elevation of the bladder neck (De Lancey, 1996; Bombieri and Freeman, 1998a).

Further support for the role of obstruction comes from the results of urethrolysis or 'take-down' procedures for patients with severe urge incontinence following colposuspension. Here, 50–85% success rates have been quoted (Nitti and Raz, 1994).

Denervation as a result of dissection might also be responsible. Following sling insertion, the incidence of overactive bladder is 16.6%, compared with 5.8% for Stamey operation where arguably there is much less dissection than with slings (Jarvis, 1994).

Alternatively, the higher rate of postoperative detrusor instability might be a result of failure to diagnose this preoperatively. Ambulatory studies (Eckford et al, 1995) or bladder wall thickness on ultrasound (Khullar et al, 1995) might detect more cases preoperatively and prevent unnecessary surgery.

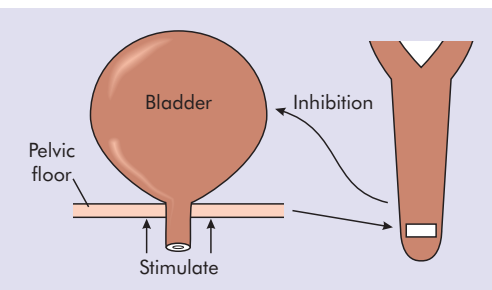


*Figure 5. Perineodetrusor inhibitory reflex.*

**Therapeutic implications for detrusor overactivity**

While the myogenic theory might lead to drugs which will relax the detrusor, e.g. potassium channel-opening agents, in the mean time, the initial results of selective anticholinergics, e.g. tolterodine, are promising.

The pelvic floor inhibition theory suggests that electrical stimulation could be an appropriate treatment. However, bladder retraining still has a high rate of success in the short term and is still first-line treatment. In intractable cases, surgery, e.g. clam cystoplasty, has a high success rate.

## CONCLUSIONS AND RECOMMENDATIONS

An understanding of the aetiology and pathophysiology of GSI and overactive bladder (detrusor instability) should hopefully result in appropriate treatments with few complications or side-effects. For example, in urethral hypermobility, surgical correction of the fascial defect might be more appropriate than elevation. Alternatively, elevation carried out objectively could lead to a reduction in complication rates. Further research is needed in this area.

An understanding of the factors involved in childbirth and identification of patients at risk might result in preventative measures being introduced, e.g. antenatal pelvic floor exercises or possibly elective caesarean section.

For ISD the encouraging results of injectables would suggest that this would be an appropriate treatment unless significant hypermobility co-exists.

Finally, in overactive bladder, an understanding of the possible aetiological factors will hopefully lead to improved treatments for this disabling condition. HM

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## KEY POINTS

- Genuine stress incontinence can be caused by bladder neck hypermobility or intrinsic sphincter deficiency (ISD).
- Hypermobility results from failure of urethral support.
- Pregnancy is a risk factor.
- ISD can be associated with higher surgical failure rates.
- Potential incontinence (after gynaecological surgery) needs to be identified preoperatively.
- The aetiology of overactive bladder/detrusor instability is less uncertain.
- Treatment results can be improved.