Voiding dysfunction in women is common, but is frequently undiagnosed until the patient presents with symptoms. The aetiology of voiding dysfunction includes the following, any of which may lead to acute or chronic disorders: obstructive causes; postsurgical conditions; neurological disorders; overdistension; inflammatory, pharmacological, psychogenic causes and learned voiding dysfunction; detrusor myopathy and urethral sphincter hypertrophy. Clinical assessment should include history, and general, neurological and pelvic examinations. Investigations may include uroflowmetry, ultrasound for residual urine and upper urinary tract dilatation, urodynamic assessment and electromyography. New surgical techniques to identify vesical branches of the pelvic nerves intraoperatively during radical hysterectomy have been shown to help prevent voiding dysfunction postoperatively. If acute retention occurs, then bladder drainage is the most important measure. Suprapubic catheters are superior to transurethral catheters if long-term voiding difficulties are expected. Whenever possible, patients with chronic retention should be taught clean intermittent self-catheterization. Depending on the cause, other possible treatment options include urethral dilatation, insertion of an intraurethral device, and neuromodulation. Voiding dysfunction in women is still poorly understood. Prompt management of acute retention is essential, and clean intermittent self-catheterization remains the most effective therapy for chronic retention. Curr Opin Obstet Gynecol 2001, 13:507-512. © 2001 Lippincott Williams & Wilkins.
Chronic retention is an insidious and painless failure of bladder emptying, in which catheterization yields a volume equal to at least 50% of normal bladder capacity. Shah et al. [2] suggested that normally functioning bladders do not retain residual urine, but it seems reasonable to suggest a catheterization volume of 50% of normal bladder capacity as a working definition. Chronic retention may cause urinary incontinence and recurrent urinary tract infections. It can occur without obvious cause.

There are usually two phases through which women pass before developing acute or chronic retention. The first is asymptomatic voiding difficulty, in which the woman is unaware of impaired bladder emptying. Urinary stream is reduced and the peak flow rate is below 15 ml/s. The maximum voiding pressure is usually normal, although it may be raised in the presence of infravesical obstruction, and there is no residual urine. The second stage is that of bladder decompensation, during which symptoms of voiding difficulty appear (e.g. hesitancy, poor stream, straining to void and incomplete emptying, with or without urinary tract infection). Peak flow is below 15 ml/s, voiding pressure is reduced and there is residual urine.

There is a paucity of data regarding the incidence of voiding disorders. Among 600 women attending a urodynamic clinic without neuropathy, Stanton et al. [3] described a 2% incidence of asymptomatic and a 14% incidence of symptomatic voiding difficulties. Dwyer and Desmedt [4] examined clinical and urodynamic findings among 1193 consecutive women referred for investigations of symptoms of lower urinary tract dysfunction; those investigators identified 165 with voiding difficulties, of whom one-third were asymptomatic.

**Table 1. International Continence Society classification of voiding phase dysfunction**

| Voiding function during voiding | Classifications |
|---------------------------------|----------------|
| Detrusor function                | Normal, Underactive, Acontractile |
| Urethral function                | Normal, Obstructive: Overactive, Mechanical |

Aetiology and pathophysiology

Voiding in the female can occur via one of three mechanisms, or a combination of all three: contraction of the detrusor muscle; a rise in abdominal pressure; and relaxation of the urethra and pelvic floor muscle. Voiding disorders may occur when these mechanisms fail, namely when the detrusor muscle is unable to maintain an effective contraction, the urethra fails to relax or additional urethral resistance is present, or if there is a failure in the synchronization of these two mechanisms, resulting in detrusor sphincter dyssynergia. The latter occurs in suprasacral neurological lesions.

**Obstructive causes**

Urethral stenosis is uncommon in the female. Distal urethral stenosis usually results from urogenital atrophy in postmenopausal women, but can also result from chronic fibrosis after chronic inflammation, urethral instrumentation (e.g. urethrotomy or traumatic catheter insertion) and scarring after surgery (e.g. anterior repair). This condition is detected during cystourethroscopy.

Acute urethral oedema may occur after bladder neck surgery. Foreign bodies and calculi are rare causes of obstruction in women. Extrinsic causes of obstruction include impaction of a retroverted gravid uterus in association with a posterior wall fibroid [5]. Pelvic masses (e.g. ovarian cysts, fibroids or faecal impaction) can also cause urethral obstruction. Urethral distortion due to genital prolapse occurs in 2% of women with grade 1 and 2 prolapse, and in 33% of women with grade 3 and 4 prolapse [6].

**Postsurgical causes**

Voiding dysfunction is frequently observed after continence surgery, and incidence ranges from 2.5 to 24%. There is sufficient evidence to suggest that voiding difficulty is a consequence of both vaginal and abdominal procedures. Akpinar et al. [7] assessed 50 patients 50 months after Burch colposuspension, and found a 4% incidence of voiding dysfunction with obstructive flows and significant residual urine.

Tension-free vaginal tape is also associated with voiding dysfunction (Table 2) [8•,9,10,11•,12]. Interestingly, in a randomized trial of colposuspension versus tension-free vaginal tape [13] there was no difference in voiding difficulty at 6-month follow up. Objective evidence for voiding difficulty was found in 9% of the tension-free vaginal tape group and in 7% of the colposuspension group.

**Table 2. Incidence of voiding difficulties following use of tension-free vaginal tape**

| Reference | Number of patients | Incidence of voiding difficulties |
|-----------|--------------------|----------------------------------|
| [8•]  | 161               | 4.3% short term, 0% long term    |
| [9] | 20                | 5%                               |
| [10] | 40                | 5%                               |
| [11•] | 156               | 5.7%                             |
| [12] | 50                | 10% short term, 0% long term     |
Bladder neck bulking agents are seldom associated with acute retention. Delayed allergy reactions to collagen injections have been reported [14], however, with women presenting with acute retention because of swelling at the bladder neck injection site.

**Neurological disorders**
Lesions in the nervous system tend to present with specific types of voiding abnormalities, depending on the site of neurological dysfunction or injury. Neurological components that control the storage and voiding phases are found in cerebral, pontine, segmental and peripheral regions. The neurological control for initiating voiding is located in the pontine micturition centre of Barrington. During the filling phase this centre is inactive, and the detrusor remains inhibited, the bladder neck closed and striated sphincter tone is maintained. When bladder capacity is reached and voiding is socially convenient, the micturition cycle begins with relaxation of the striated sphincter, bladder neck opening and detrusor muscle contraction. Voiding difficulties may occur as a result of lesions in the brain located in the frontal lobe, internal capsule, reticular formation and cerebellum, or following lesions to the spinal cord (e.g. trauma, spina bifida, tumour or prolapse of an intervertebral disc).

Many neurological illnesses affect the nervous system at various levels and to different extents. The mechanism of dysfunction may involve lack of proprioceptive impulses from the bladder wall, signalling fullness and leading to lack of bladder sensation and overflow incontinence; disturbance of pontine or distally located signals, resulting in voiding difficulties; or denervation of bladder after radical surgery, with complete failure of detrusor contraction.

Cerebral and spinal cord diseases can be congenital, neoplastic, degenerative, inflammatory, vascular, traumatic or functional. Many disorders can result in loss of inhibition and inappropriate voiding. Unilateral central lesions usually do not have a permanent effect on micturition. Unilateral peripheral lesions (e.g. unilateral pudendal nerve damage) can have a negative impact, but are more likely to result in deteriorating continence than in voiding disorders.

Reflex voiding in patients with spinal trauma is segmentally organized and can be initiated by a full bladder, external stimuli, or a combination of these. Application of external stimuli to achieve voiding is not recommended because it usually results in incomplete emptying. In addition to this, there may be associated sphincter activity at the same time as detrusor contraction, provoking high intravesical pressures, and this may lead to vesicoureteral reflux later.

In such patients clean intermittent self-catheterization is recommended (see below). Lesions that occur below the reflex arc may result in bladder acontractility, with the additional problem of a nonrelaxing urethral sphincter.

Chronic illnesses such as Parkinson’s disease or Multiple Sclerosis may be associated with detrusor hyperreflexia, resulting in frequency and urgency or chronic retention, or even a combination of these. In such cases, treatment may include anticholinergics in combination with clean intermittent self-catheterization. Cauda equina syndrome caused by compression is usually associated with pain, acute urinary retention, sensory loss of the perineum and leg weakness.

**Overdistension**
Bladder overdistension as a result of mismanagement of acute or chronic retention develops insidiously. It often results after failure to detect retention following pelvic surgery (e.g. hysterectomy) or epidural/peridural anaesthesia. Overdistension may occur without obvious cause, is more frequently observed in elderly women, and in such patients it results in large acontractile bladders. It leads to ischaemic damage to the detrusor muscle, and the prime affected site is the basal urothelium [15]. Continued ischaemia provokes a proliferative vascular response, laying down of new collagen and irreversible damage.

**Inflammatory causes**
Any painful vulvo-vaginal lesion may result in disturbance of normal micturition. The commonest causes are infective, chemical or allergic. Voiding difficulties may result from painful stimuli produced by urine coming into contact with the inflamed mucosa of either urethra or vagina. Primary anogenital herpetic infection may produce urinary retention both by the effect of local inflammatory lesions and by lumbosacral meningomyelitis affecting the central nervous system [16].

**Pharmacological causes**
Obstetric epidural anaesthesia is the most common pharmacologic cause of voiding dysfunction. If retention is overlooked, then overdistension injury may result in long-term voiding difficulty [17]. Drugs that interfere with the release and action of acetylcholine at cholinergic synapses or neuromuscular junctions can result in voiding dysfunction, especially when mild impairment is already present. Anticholinergic agents that are used to treat urgency and frequency (e.g. tolterodine, oxybutinin, probanthine and imipramine) are common culprits. In patients with symptoms of impaired voiding and frequency/urgency, it is therefore vital to exclude residual urine before prescribing these drugs.
Learned voiding dysfunction (non-neurogenic, neurogenic bladder)
Non-neurogenic, neurogenic bladder is a functional bladder outlet syndrome that is caused by the voluntary contractions of the external urethral sphincter during voiding. This condition is also known as Hinman syndrome, and is considered to be a learned behavioral disturbance; it may be treated by re-education therapy. Groutz et al. [18••] investigated 1015 consecutive men and women referred for voiding dysfunction by videourodynamics, and found an overall incidence of 2% of learned voiding dysfunction that was equally distributed between the men and women.

Psychogenic causes
Psychogenic causes of voiding dysfunction are well documented [19,20]. Criteria for this diagnosis are an absence of neurological or other significant organic disease, correlation with psychological disturbance, and response to psychotherapy or psychopharmacological agents. This diagnosis should be made after careful evaluation and exclusion of other causes.

Detrusor myopathy
Primary changes in the detrusor muscle have been reported as a cause of retention. Lipid inclusion bodies are observed within the muscle cells [21].

Urethral sphincter hypertrophy
Fowler and Kirby [22] described a group of women who presented with voiding difficulties that were due to a primary defect within the striated urethral sphincter. The muscle was hypertrophied and failed to relax. Characteristic electromyographic patterns were observed, with complex repetitive discharges and decelerating bursts. There appears to be an association with polycystic ovarian syndrome. This is a particularly difficult group of women to treat.

Clinical assessment and investigations
In a few patients impaired voiding may be asymptomatic, but the majority present with infrequent voiding, poor flow, intermittent stream, incomplete emptying, straining to void and hesitancy. Others may present with overflow incontinence and frequency, or urinary tract infections due to stasis. History should be tailored to determine a primary cause. Neuropathy should be enquired about, and a detailed drug, medical and surgical history, including genital and urinary tract infection, should be obtained. Continence surgery is a well recognized causative factor.

A careful general, abdominal, neurological and pelvic examination should be performed. Abdominal and pelvic examination should be performed after bladder emptying and masses noted (e.g. ovarian cysts and fibroids). A bladder may be palpable and this finding usually means a residual of 200 ml or more. Any urethral or vulvovaginal inflammation or atrophy is noted and the urethra is palpated for tenderness or scarring. Finally, the patient’s general demeanour should be carefully monitored in order to detect any signs of psychiatric disorder.

Urinary tract infection should be excluded because it may predispose to voiding difficulty. The simplest investigations to exclude voiding dysfunction are uroflowmetry and ultrasound for residual urine and upper urinary tract dilatation, but urodynamic assessment and electromyography may also be useful in addition to cystourethroscopy. Investigations are tailored to the suspected cause.

Uroflowmetry is the most important initial screening procedure, and is simple and noninvasive. The measurement should be made in privacy and the patient should attend with a comfortably full bladder. Repetitive flow rates below 15 ml/s for a volume in excess of 150 ml indicate impaired voiding, and this may be a precursor for retention.

Additional radiography studies are not helpful in patients with voiding dysfunction, as demonstrated by Probert et al. [23] in 320 patients with obstructive symptoms; those investigators recommended lower urinary tract ultrasound as the only routine imaging performed in the assessment. Automated bladder ultrasound correlates well with catheterized urine volume, but can occasionally be falsely elevated because of cystic pelvic pathology [24•]. Although absence of residual urine does not exclude voiding dysfunction, the presence of residual urine is abnormal. Residual volumes greater than 100 ml are seen as significant. An isolated instance of residual urine is not usually clinically significant.

Prevention and treatment
Prevention or early recognition of retention may avoid long-term voiding difficulty. Difficulty in resumption of spontaneous voiding occurs in over 45% of patients after radical pelvic surgery for gynaecological malignancy [25,26]. Attempts at intraoperative identification of vesical branches of the pelvic nerves by electrical stimulation can reduce bladder dysfunction significantly after radical hysterectomy [27••]. Twelve patients with stage Ib cervical cancer showed significantly reduced residual urine volumes postoperatively.

If significant residual urine is detected, then cholinergic drugs, prostaglandins and progesterone are usually of little value, and a catheter must be inserted. If a catheter is intended to be in place for a short-term period (e.g. after epidural or immediately after surgery), then a urethral catheter will suffice. Where longer term problems may ensue (e.g. after continence surgery), a
suprapubic catheter allows better assessment of voiding and residual volumes with a lower associated urinary tract infection rate. In women with evidence of voiding difficulty before continence surgery, it is reasonable to counsel and teach intermittent self-catheterization.

Indwelling catheters are used less frequent in patients with neurologic disorders because they are associated with urinary tract infections, progressive urethral dilatation leading to leakage past the catheter, and discomfort. In cases in which clean intermittent self-catheterization is not feasible, insertion of a suprapubic catheter may be required or, as a final option, urinary diversion.

**Intermittent self-catheterization**

Intermittent self-catheterization as a nonsterile procedure was first described by Lapides in 1972 [28]. Originally used in neurogenic conditions, it is now the primary treatment for chronic urinary retention. It allows women to lead independent normal lives, with efficient bladder emptying and low infection rates. Although patients are initially apprehensive at the idea of such a technique, if properly counselled by professional staff most will easily master the technique and enjoy the consequent improvement in quality of life.

There are two forms of intermittent self-catheterization: sterile and clean. The former is reserved for patients with neuropathic bladders in a hospital environment in order to prevent cross-infection.

Clean intermittent self-catheterization is usually performed by the patient, but can also be performed by a carer or a relation. The technique is designed for everyday use. The patient has to have reasonable manual dexterity, and is taught to use a clean technique and mirror; the patient lies down initially and inserts a fine-bore catheter. When proficient with the mirror, she is taught to insert the catheter by feel in the sitting or standing position. Disposable catheters are available, but reusable ones do not pose a significant risk of infection. A salt coating allows easier insertion with less urethral friction. The frequency of catheterization varies, with the aim of avoidance of incontinence and of filling beyond normal bladder capacity. Good long-term results have been reported with the procedure [29].

In women with combined urge incontinence and retention, anticholinergic agents such as tolterodine may be used effectively in conjunction with clean intermittent self-catheterization.

**Surgery**

If voiding difficulty is due to urethral stenosis, then urethral dilatation using Hegar dilators or the Otis urethrotome are appropriate options.

**Turbine valve**

The turbine valve is a replaceable intraurethral sphincter prosthesis (In Flow system, AMS, Minneapolis, USA), with a self-contained urinary pump for treatment of chronic female urinary retention. Activation is achieved by turning on a small battery-operated remote control unit that is placed over the lower abdomen. Early results have been encouraging, but recent long-term follow-up periods longer than 1 year have revealed complications such as migration of the device into the bladder, infections and dyspareunia, despite good patient satisfaction [30]. A rate of removal of 50% due to side effects has been reported [31].

**Neuromodulation**

This two-stage procedure involves stimulation of the S3 nerve root through the S3 foramen. The first stage is that of percutaneous nerve evaluation using a temporary stimulation wire. If this has a beneficial effect, then a permanent stimulator is implanted. Encouraging early results have been reported, especially in patients with Fowler’s syndrome (personal communication by CJ Fowler), but the mechanism of action is not understood.

**Conclusion**

Voiding difficulties in women are poorly investigated. Prompt treatment of acute retention and pre-emptive management in order to avoid overdistension are essential. Clean intermittent self-catheterization remains the most effective therapy for chronic retention, although newer modalities are being investigated. The roles of pharmacological agents and surgery are limited in women.

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This study evaluated 161 women with urodynamically proven stress incontinence who underwent treatment with tension-free vaginal tape. Average follow-up time was 16 months. Intraoperative complications (e.g. bladder perforation and haemorrhage) and postoperative complications (e.g. de-novo urgency, retention and urinary tract infections) were recorded. The success rate was 94% and the voiding difficulty rate was 0%.

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In this study 171 patients undergoing primary treatment with tension-free vaginal tape were followed up. Differences in success rates, voiding problems and de-novo urgency were compared between patients who underwent tension-free vaginal tape treatment alone and those who underwent tension-free vaginal tape treatment in combination with prolapse operations. De-novo urgency was more frequently observed in women undergoing concurrent surgery for prolapse. De-novo voiding difficulties were similar in both groups, with 5.2% in the tension-free vaginal tape only group versus 6.5% in the group with combined tension-free vaginal tape and prolapse operation. Success rates for cure of incontinence were similar in both groups.

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Because radical hysterectomy is associated with an increased risk for voiding dysfunction, the authors sought a new method to prevent bladder dysfunction by employing intraoperative electrical stimulation to identify the vesical branches of the pelvic nerves and avoid injury to them during surgery. Grades of bladder dysfunction were compared between the study group (n=19) and the conventional method. The study group achieved normal voiding with low urinary residual volume (less than 50 ml) in significantly less time than did the conventionally operated group. Urodynamic assessment of bladder sensation showed full preservation in the study group.

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