

Lower Urinary Tract

Authors from the USA present a review of the treatment of BOO in the female. This topic is important, which should be of considerable help to the reader. It is covered systematically, dealing with anatomy and then therapy. There is also a meta-analysis comparing industry- and non-industry funded trials of antimuscarinic medication. This careful study shows no difference in outcomes between them, but suggests there are some shortcomings that need to be overcome.

The treatment of female bladder outlet obstruction

HOWARD B. GOLDMAN and PHILIPPE E. ZIMMERN*

Section of Voiding Dysfunction and Female Urology, Glickman Urological Institute, The Cleveland Clinic, Lerner College of Medicine of CASE University, Cleveland, Ohio, and *Department of Urology, University of Texas at Southwestern Medical Center at Dallas, Dallas, Texas, USA

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INTRODUCTION

The treatment of BOO in women depends to a large degree on its cause; 'one size does not fit all'. Once the appropriate cause of BOO has been identified the treatment is rather straightforward. BOO in women can present in a myriad of ways. Typical complaints of slow urinary flow and difficulty emptying make the clinician immediately consider its diagnosis. However, many other presenting symptoms, e.g. irritative voiding complaints, can be secondary to BOO but be more difficult to diagnose. This reinforces the crucial importance of a high level of suspicion for BOO in women, and the need for a thorough investigation, including a careful history and physical examination, and appropriate ancillary studies to help make the appropriate diagnosis. In this review we discuss the treatments based on each specific cause.

CAUSES OF BOO

The simplest way to classify the causes of BOO in women is by separating anatomical causes from those that are functional. Furthermore, within the anatomical causes extrinsic causes of BOO can be further separated from those that are intrinsic to the urethra and those that

are luminal (Table 1). We divide the treatment for the various causes of BOO in women according to this classification scheme.

TREATMENT

ANATOMICAL

Extrinsic causes

Pelvic prolapse: Up to 70% of women with significant uterovaginal prolapse can have BOO [1]. Indeed, a small group (3%) with mild prolapse might also have BOO [1]. The diagnosis is usually made either by a trial with a pessary for a few weeks, with the patient reporting symptomatic improvement, or by urodynamics, comparing the pressure-flow findings with and with no prolapse reduction. There is usually an increase in flow rate, with or with no concomitant decrease in voiding pressure when the prolapse is reduced (Fig. 1). Prolapse reduction is challenging and the method itself can cause obstruction [2]. In patients with a well-supported urethra from a previous urethropexy, a secondary cystocele can induce a marked angulation at the urethro-vesical junction, and result in irritative and/or obstructive symptoms (Fig. 2). BOO can result from the urethropexy or the secondary prolapse, or both. In these cases prolapse reduction can help to determine the mechanism of BOO and lead to a proper treatment decision between prolapse repair alone or associated with a urethrolisis.

Ultimately, treatment consists of use of an appropriate pessary or surgical correction of the prolapse. Details of surgical correction of prolapse and the current controversies within that area can be found in many current textbooks and journals.

Iatrogenic BOO After anti-incontinence surgery: BOO after anti-incontinence surgery is reported to occur in 5–20% of patients undergoing such surgery [3]. It appears that the number of patients undergoing anti-incontinence surgery has dramatically increased in recent years, and thus the number of potential patients with iatrogenic BOO might also increase [4].

The mechanism of obstruction can vary depending on the type of surgery, and this has important implications for appropriate treatment. While with a retropubic bladder neck suspension (Burch, etc.) the mechanism of obstruction is probably sutures that are too tight, or excessive scarification anterior to the urethra, whereas with a sling it is probably excessive ventral compression of the urethra by the sling (Fig. 3). Thus, in most cases of BOO secondary to a sling, whether a bladder neck fascial sling or a mid-urethral synthetic sling, simple sling incision usually gives significant symptomatic relief. On the other hand, effective treatment for an obstructing retropubic bladder neck suspension requires suture release and disintegration of the scar between the urethra and pubis. This can be done transvaginally, possibly with an associated Martius labial fat pad wrap to decrease re-scarring [5] or transabdominally, as recently advocated by some [6].

The most important component of treatment is accurate identification of the problem. There must be a high index of suspicion for BOO in all clinicians who perform anti-incontinence surgery. Patients who present with urinary retention or a markedly diminished force of stream after such surgery are easy to identify. However, many patients can have more subtle obstructive or irritative symptoms. Bending forward to void or having to change positions to empty effectively usually indicates an element of obstruction (Fig. 4). Recurrent UTIs associated with a high postvoid residual urine volume or new onset or worsened irritative symptoms, e.g. urgency, frequency, or urge incontinence, can be related to iatrogenic BOO. Importantly, the classical clinical findings (a negative Q-tip angle, a 'bump' in the urethra, etc.) can occur less often with the mid-urethral slings than

Type	Cause	TABLE 1 Causes of female BOO
Anatomical		
Extrinsic	Pelvic prolapse – cystocele, rectocele, etc. After anti-incontinence procedure Gynaecological – fibroids, etc. Poorly fitting pessary	
Urethral	Stricture Meatal stenosis Thrombosed caruncle Fibrosis Diverticulum Skene's gland cyst or abscess Excessive amount of bulking agent	
Luminal	Stone Bladder/urethral tumour Ureterocele Foreign body	
Functional		
	Primary bladder neck obstruction Pseudo-dyssynergia Detrusor-external sphincter dyssynergia; neurological	

with those of the bladder neck variety. The temporal association between surgery and a change in voiding pattern might be the clearest clue to the presence of iatrogenic BOO.

Urodynamic testing is useful when this temporal relationship is lacking, in patients in partial retention or with predominantly irritative symptoms but in whom BOO is clinically suspected, or when the interval is such that a reversible process is questionable even if it can be technically accomplished. Reviewing preoperative test findings (when available) is invaluable when plausible, and valid tracings show a baseline Valsalva voiding pattern or a low-pressure detrusor contraction (hypocontractility). Voiding urodynamics can be challenging because of issues of technique (clear annotations, valid pressure ranges) and interpretation. An elevation in detrusor pressure not commensurate with the flow indicates an obstructive element in a non-neurogenic patient. However, the threshold criteria for obstruction remain debated [7,8] and do not apply to patients in complete retention or unable to void during the study, unless they are able to mount a detrusor contraction even with no detectable flow (Fig. 5).

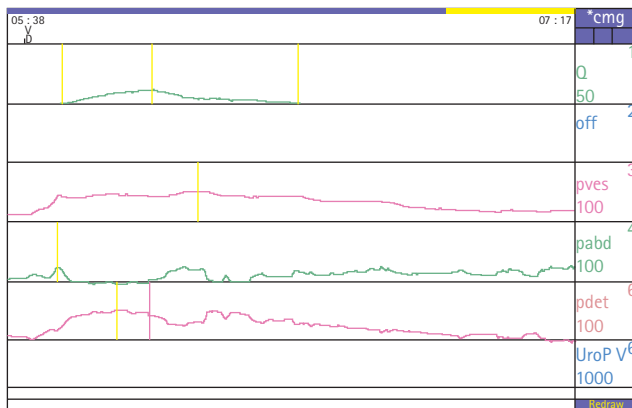
For an obstructing sling the simplest technique to resolve BOO is simple sling incision. Several studies showed an efficacy rivaling that of the traditional urethrolisis [9,10]. For those with a bladder neck sling, a

cystoscope sheath is placed in the urethra and slowly withdrawn while applying downward pressure. A 'step' should be felt; this is the site of the sling, and an anterior vaginal wall incision is made over this site. When approaching a mid-urethral sling a 'step' is usually not felt, and an incision is made ≈2.0 cm from the urethral meatus, and with careful dissection a rough edge can usually be palpated. In either case, once the sling is identified a right-angle clamp is carefully insinuated between the sling and urethra and gently spread, after which the sling is incised (Fig. 6). The surgeon must be cautious to be certain that the urethra is not injured during this manoeuvre. Occasionally the tissue between the sling and urethra can be attenuated and quite thin, and in some cases can be adherent to the sling material. Under these circumstances inadvertent urethral injury can occur. If there does not appear to be sufficient space to allow for clean separation of the sling from the urethra, it might be safer to start the dissection more laterally, and once in the appropriate plane to continue the dissection medially under the urethra. In most cases the sling edges retract because the tension has been released. It is crucial to be sure that the entire width of the sling has been cut. If there was a 'step' present, it should now be gone. Before finishing the procedure careful cysto-urethroscopy and visual inspection of the urethra, with the cystoscope fluid running and the tip of the cystoscope in the distal urethra to evaluate for fluid leakage from the

FIG. 1. Urodynamic tracing from a woman with prolapse and poor flow. (A) without pessary; note the maximum flow of 12 mL/s and detrusor pressure at maximum flow ($P_{detQ_{max}}$) of 43 cmH₂O. (B) with pessary; note the increase in flow to 42 mL/s and decrease in $P_{detQ_{max}}$ to 19 cmH₂O.

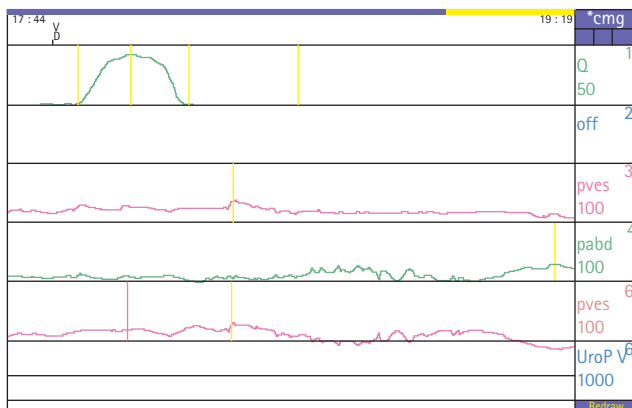
A Pressure/Flow (Voiding Phase) Report (cmH₂O)

Q max: 12 ml/s	T to Max: 16 s	T Flow: 41 s	Volume: 235 ml			
Q ave: 6 ml/s		T Void: 41 s	OVR : 30 ml			
Position: sitting	Pattern: continuous	Conclusion :				
	pves	pabd	pdet	pura	pcl	Rura
Maximum Pressure	52	25	52	NA	NA	
Pressure at Q max	46	3	43	NA	NA	0.4
Opening Pressure	46	23	23	NA	NA	
pmuo			23			
Descending Slope : 1 cmH ₂ O/ml/s						
Voiding Pressure :			Flow Rate :			
Urethral Resistance :			Opening Time : NA s			
EMG coordination :			EMG activity :			
EMG comments : EMG NOT DONE.						
Comments: STUDY DONE W/O VAGINAL PESSARY.						



B Pressure/Flow (Voiding Phase) Report (cmH₂O)

Q max: 42 ml/s	T to Max: 9 s	T Flow: 19 s	Volume: 495 ml			
Q ave: 26 ml/s		T Void : 19 s	PVR : 95 ml			
Position: sitting	Pattern: continuous	Conclusion :				
	pves	pabd	pdet	pura	pcl	Rura
Maximum pressure	36	27	28	NA	NA	
Pressure at Q max	28	9	19	NA	NA	0.0
Opening Pressure	25	9	16	NA	NA	
pmuo			16			
Descending Slope : 1 cmH ₂ O/ml/s						
Voiding Pressure :			Flow Rate :			
Urethral Resistance :			Opening Time : NA s			
EMG coordination :			EMG activity :			
EMG comments : EMG NOT DONE.						
Comments : STUDY DONE WITH VAGINAL PESSARY.						



surgical area, should be done to ensure that there is no urethral injury. Of patients undergoing simple sling incision, 70–90% will have a dramatic improvement in their symptoms [9,10]. In a minority (20%) recurrent stress urinary incontinence (SUI) can occur. In those in whom sling incision does not alleviate the symptoms, a full transvaginal urethrolysis with bilateral perforation of the endopelvic fascia and complete mobilization of the urethra might be necessary [10].

Patients who have BOO after a retropubic bladder neck suspension can be approached transvaginally. This approach offers complete urethral mobilization from surrounding fibrotic tissues, along with possible Martius flap interposition to prevent the formation of recurrent periurethral fibrosis. Complete retropubic freeing after a Marshall-Marchetti-Krantz procedure or a very tight Burch might necessitate a suprameatal incision to liberate the urethra completely from the back of the symphysis pubis. The retropubic approach can also be considered to cut the suspension sutures if the re-operation is contemplated early before permanent fibrosis settles in. However, after a few months, releasing the sutures alone will usually not result in much voiding improvement and the bladder neck and urethra must be fully separated from the pubis. This approach can be more hazardous for bladder injury and retropubic bleeding. Omental interposition between the freed bladder neck and proximal urethra and the back of the pubic bone can be considered to avoid recurrent scarring. For more detail on the exact surgical techniques of urethrolysis, see [2].

While patients can improve many years after the primary procedure to relieve the obstruction, recent data suggest that delayed treatment of this type of obstruction can lead to irreversible bladder dysfunction [11].

Intrinsic

Urethral fibrosis or stricture: Most urethral strictures or fibrosis in women are iatrogenic and can result from previous urethral or periurethral surgery, or in some cases from previous urethral dilatation. These pathologies might be identified during cystoscopy or in some cases after a lateral cystogram or MRI [12]. Depending on the location and length of the stricture or fibrosis, treatment might involve cystoscopy and

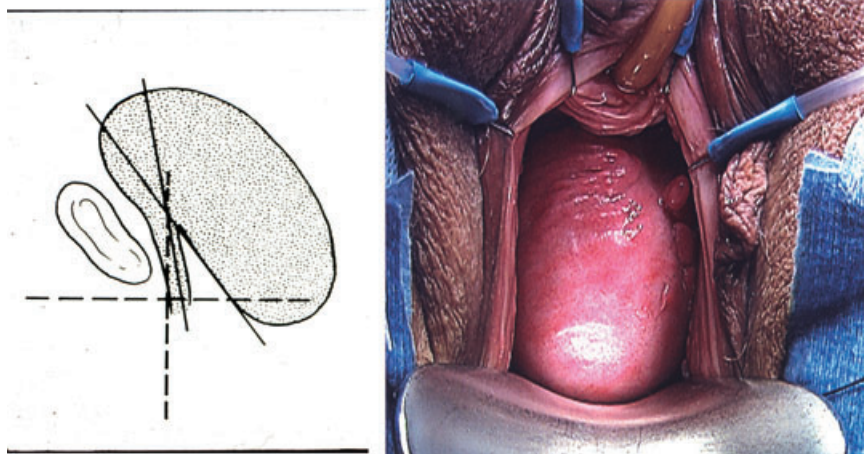


FIG. 2.

A successful urethral support but no attention to the existing laxity of the upper anterior vaginal wall and vault may result, years later, in BOO, by gradual kinking of the urethro-vesical angle. Urodynamic testing with prolapse reduction can improve the flow and/or detrusor pressure.

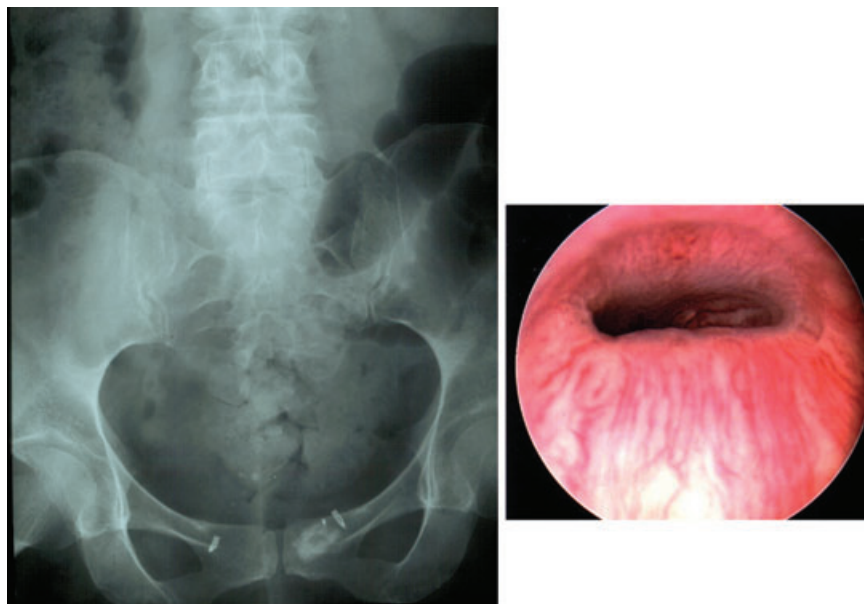


FIG. 3.

Severe urethral lumen distortion and flattening from a bone-anchor sling, resulting in voiding difficulties after surgery and pain with CIC in the mid-urethral region.

judicious dilatation, transurethral incision, and/or referral to specialized centres for vaginal flap interposition or complete urethral reconstruction [13]. Urethral dilatation should not be done indiscriminately. Only women who are truly obstructed and are felt to have an anatomical reason to necessitate urethral dilatation should have it. The previous routine, where all women with voiding symptoms or UTIs were dilated, should no longer be standard practice. When appropriate, the urethra can be dilated in the office setting, usually with premedication with an oral anxiolytic and narcotic, but for better tolerance and possibly enhanced effectiveness (to dilate more aggressively) this procedure can also be done under brief anaesthesia in an outpatient setting.

Diverticulum: In some cases a circumferential urethral diverticulum can compress the urethral lumen such that BOO occurs (Fig. 7). Diverticulectomy with maintenance of an appropriate calibre of urethral lumen should cure the obstruction [14].

Skene's gland cyst or abscess: On occasion, a large or infected Skene's gland cyst can cause BOO. If infected it can accumulate purulent material and cause distal urethral or meatal obstruction. On examination the meatus is usually eccentrically shaped and there might be a perimeatal bulge which is tender (Fig. 8). Pressure on the fluctuant area might or might not express purulence from the Skene's gland duct, but will cause no purulence to extrude from the

urethral meatus. In most cases incision and drainage of the gland in the perimeatal area, along with marsupialization to prevent recurrence, will be an adequate treatment. The urethra itself does not need to be entered.

Excessive bulking agent: Short-term BOO after injection with a bulking agent is quite common. Usually symptoms resolve within hours to days, but occasionally after injection with large amounts of a bulking agent, usually at more than one session [15], or after injection with too much of a permanent material, long-lasting obstruction can occur. In such cases transurethral resection of the obstructing material will usually alleviate the obstruction.

FIG. 4. Obstruction from a sling placed under tension in the proximal urethra. The urethral axis is kinked with a negative angle proximally. This patient had to void while standing and leaning forward with straining, to facilitate better emptying.

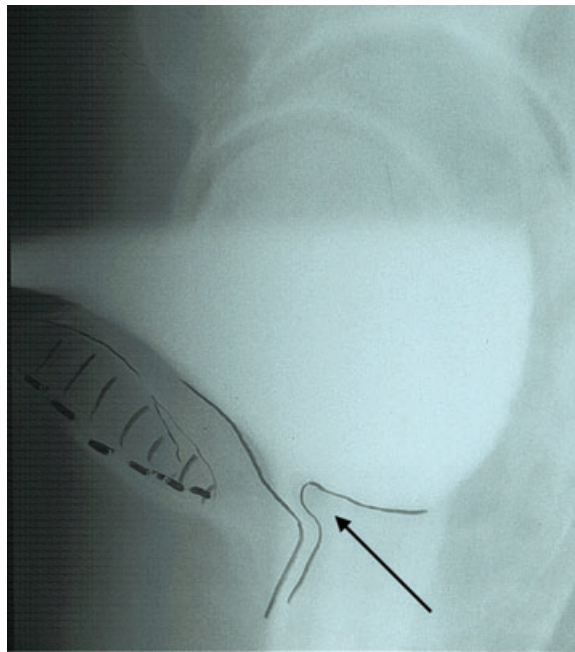
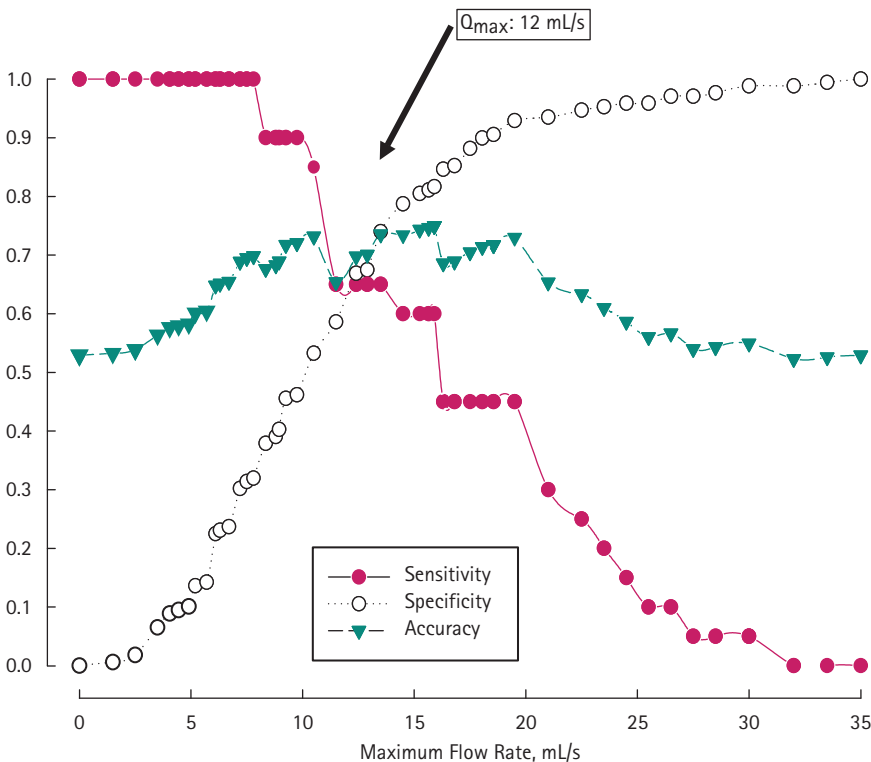


FIG. 5. Threshold pressure-flow criteria indicating that the combination of a flow at ≤ 12 mL/s for a detrusor pressure of ≥ 25 cmH₂O offers the highest sensitivity and specificity for BOO. Adapted from Defreitas et al. [8].



Luminal

An obstructing luminal structure can usually be diagnosed during cystoscopy. Those that are not attached to the urethra/bladder (stone,

foreign body) can be removed or broken transurethrally, while those that originate from the urethra or bladder (tumour, polyp) can usually be resected transurethrally. Urethral carcinomas can present with BOO. Definitive

treatment might require urethrectomy or even radical cysto-urethrectomy.

FUNCTIONAL

Primary bladder neck obstruction (PBNO)

PBNO has been identified in 9–16% of women with BOO [16,17] and is caused by a lack of relaxation of the smooth muscle at the bladder neck during voiding. The urodynamic features include a high voiding pressure, low flow rate, lack of significant electromyographic (EMG) activity and a non-funnelling appearance of the bladder neck on fluoroscopy (Fig. 9). Some patients can actually present with long-standing idiopathic retention and be dependent on clean intermittent catheterization (CIC). Once again, a high index of suspicion is necessary to make the correct diagnosis. Treatment options include CIC, α -blockers, transurethral incision of the bladder neck, and neuromodulation.

For those who do not desire an intervention or have already become comfortable with CIC, that may be an option. However, a trial with α -blockers should at least be considered; a recent study showed that half of women with PBNO responded well to α -blocker therapy [18]. In these patients the symptoms improved, flow rates increased, and the postvoid residual urine volume decreased significantly. α -blocker therapy should be tried before invasive therapy, as there are few side-effects and the efficacy of α -blocker therapy might suggest a good response to bladder neck incision.

Transurethral resection or incision of the bladder neck usually provides definitive treatment for a patient with PBNO. There are many published reports of its efficacy [18–20]; while some advocate resection of a portion of the bladder neck, most just incise it. One study assessed a technique wherein the bladder neck is incised at the 5 and 7 o'clock positions, after which the intervening tissue (from 5 to 7 o'clock) is resected. With a median follow-up of 3 years, six of seven patients considered themselves cured and only one had mild SUI under rare circumstances, and did not wear protective padding at 8 years after surgery [19]. Others have reported favourable results with just incision, with no resection. Most authors make two incisions, usually at the 5 and 7 o'clock positions, while some make only one, usually in the midline. A recent report reviewed the outcomes of 11 women with

FIG. 6. Synthetic midurethral sling dissected out and ready to be incised.

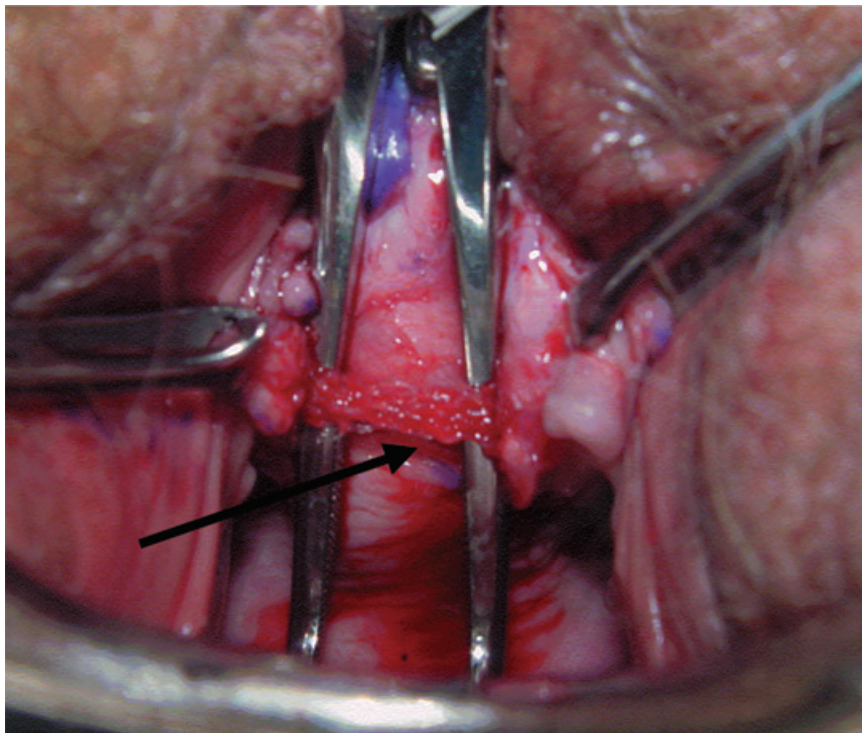


FIG. 8. Partially obstructing infected Skene's gland cyst. Note the perimeatal bulge next to a Foley catheter in urethra.

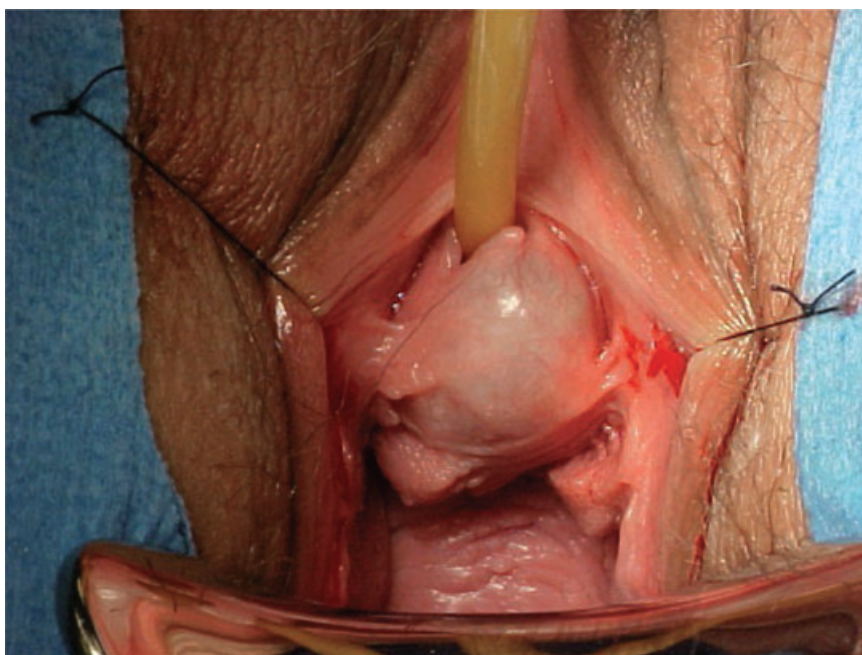
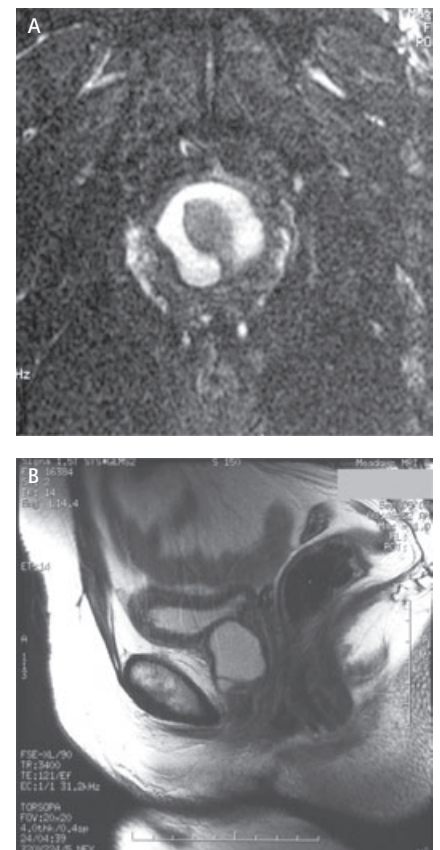


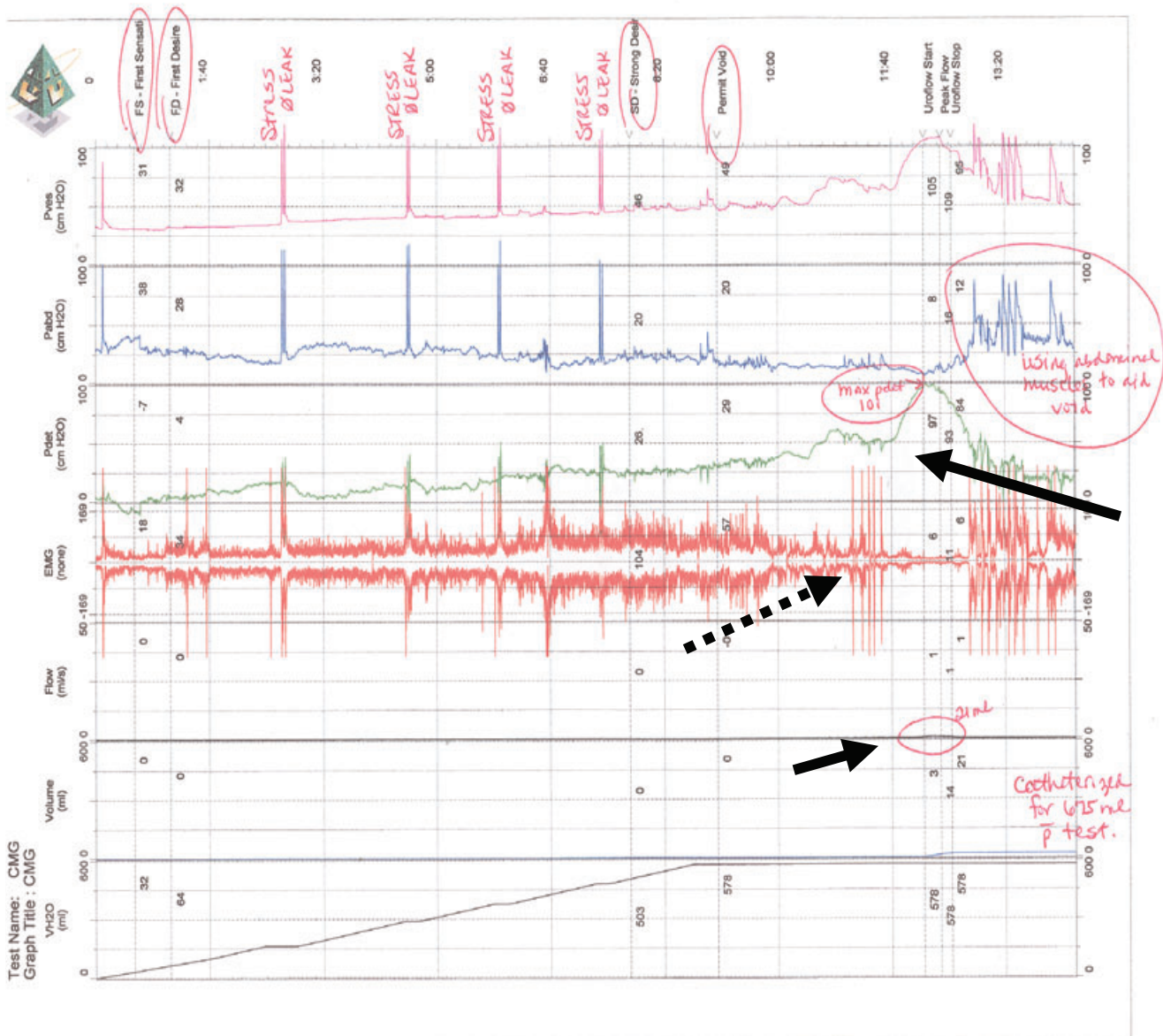
FIG. 7. (A) Pelvic MRI showing a completely circumferential urethral diverticulum in a 48-year-old woman presenting in urinary retention. (B) Note the extension of the diverticulum toward the bladder base on the sagittal view.



PBNO [20]. The bladder neck was incised transurethraly at the 5 and 7 o'clock positions deep into the serosal layer. A Foley catheter was left in place for 48 h; 10 of the 11 patients had symptom resolution or improvement, and one developed mild SUI. Some have suggested that the use of a paediatric resectoscope and Collins knife, and one bladder neck incision, might decrease the chance of SUI [18]. Ultimately, transurethral incision of the bladder neck appears to offer definitive, permanent treatment for women with PBNO, albeit with a chance of mild SUI.

Recently some have avoided a permanent solution, perhaps because of the risk of SUI, and have tried various neuromodulatory interventions. Injections with botulinum toxin into the bladder neck has been used, as has sacral neuromodulation, for PBNO in women. Currently there is a paucity of outcome data using these approaches.

FIG. 9. A urodynamic tracing of a woman with PBNO. Note the high detrusor pressure (long solid arrow), cessation of EMG activity (broken arrow) and poor flow (short solid arrow).



Pseudo-dyssynergia

Pseudo-dyssynergia, otherwise known as non-neurogenic, neurogenic bladder, Hinman's syndrome, external sphincter spasticity, as well as other pseudonyms, results from an external sphincter that does not relax during voiding in an otherwise neurologically intact woman. This 'obstructive' external sphincter can lead to both obstructive and irritative symptoms. Increased pelvic floor EMG activity during voiding is one of the hallmarks of this

syndrome. There are many theories as to its cause and it is probably an acquired (learned) disorder. First-line treatment consists of pelvic floor relaxation. This usually needs to be taught by a trained therapist, such that the patient re-learns how to void properly. Skeletal muscle blockers such as diazepam can be used as well, but the side-effects can limit their applicability. Finally, sacral neuromodulation has been used successfully in this group of patients but published outcome data are scarce.

Detrusor external sphincter dyssynergia

This occurs when the external sphincter contracts during a detrusor contraction in a neurologically impaired individual. It is common in patients with suprasacral spinal cord injuries and those with multiple sclerosis. A full discussion of its treatment is beyond the scope of this review, but the recent introduction of botulinum toxin injections into the external sphincter of these patient may revolutionise its treatment [21].

PREVENTION OF FEMALE BOO

The most common form of preventable BOO in women is after anti-incontinence surgery. Sutures that are misplaced or tied too tightly, or a sling that is pulled up too tightly, can lead to obstruction. Care should be taken to ensure that adequate laxity is present at the end of any anti-incontinence procedure; loose is usually better. Unfortunately, there is no 'magic formula' to determine the adequate tension, but experience and proper training help. However, a few basic points are clear. When performing a retropubic bladder neck suspension there is no need to pull the periurethral tissue all the way up to Cooper's ligament. A generous airspace can usually be left between them. With the bladder neck slings that are tied over the rectus fascia there should be enough room for two or three of the assistant's fingers to fit between the suture and fascia (with no tension). Also note that autologous fascia can contract a little from its original size and thus 'tighten' on its own. For patients having a traditional mid-urethral sling (up through the retropubic space) with urethral hypermobility, a spacing device like a 10 F Hegar dilator should be placed between the mesh and the urethra, and mild countertension should be applied when the mesh is positioned. Ultimately, a small air loop should be present when the mesh is in its final position. Finally, newer transobturator slings can be left flat or with a small air space between the mesh and urethra (a Mayo scissors may be used as a spacer). It appears that the 'flatter' position of these slings can lead to less urethral obstruction but further study of this is required.

Another less common, but problematic type of iatrogenic BOO in women is an iatrogenic urethral stricture or fibrosis. This can result from urethral surgery (diverticulectomy, urethral reconstruction) where the calibre of the lumen is severely compromised, or occasionally from urethral dilatation. When operating on the urethra care should be taken that the calibre of the lumen is at least large enough to accept a 14 F catheter with no difficulty.

CONCLUSION

Female BOO is probably a more common cause of voiding dysfunction in women than is commonly appreciated. A thorough evaluation, including urodynamics, should

be able to identify BOO and determine its cause. The most important part of its identification is a high index of suspicion. Once the cause is determined, appropriate treatment will lead to resolution of symptoms in most patients.

CONFLICT OF INTEREST

None declared.

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Correspondence: Howard B. Goldman, Section of Voiding Dysfunction and Female Urology, Glickman Urological Institute, The Cleveland Clinic, 9500 Euclid Ave/A100, Cleveland, OH 44195, USA.
e-mail: goldmah@ccf.org

Abbreviations: SUI, stress urinary incontinence; PBNO, primary bladder neck obstruction; EMG, electromyography; CIC, clean intermittent catheterization.