Primary Bladder Neck Obstruction

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Primary bladder neck obstruction (PBNO) is a functional obstruction caused by abnormal opening of the bladder neck during the voiding phase of micturition. PBNO may present with a variety of symptoms including voiding symptoms (slow urinary stream, intermittent stream, incomplete emptying), storage symptoms (frequency, urgency, urgency incontinence, nocturia), and/or pelvic pain and discomfort. The diagnosis of PBNO can be made with videourodynamic testing, which demonstrates elevated voiding pressures with low flow, and fluoroscopic imaging demonstrating obstruction at the level of the bladder neck. Treatment options include conservative management with watchful waiting, pharmacologic management, and surgical intervention. In this article, we review the etiology, presentation, diagnosis, and treatment of PBNO in men, women, and children.

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KEY WORDS

Primary bladder neck obstruction • Lower urinary tract symptoms • Bladder outlet obstruction

Primary bladder neck obstruction (PBNO) is a functional obstruction of the bladder caused by abnormal opening of the bladder neck during the voiding phase of micturition. This occurs in the absence of other causes of anatomic obstruction or increased striated sphincter activity. PBNO was first described in men by Marion in 1933[1] and, in 1984, Diokono and colleagues[2] were the first to report PBNO as a clinical entity in women.

Etiology

The etiology of PBNO is not completely understood although there have been multiple theories proposed as the underlying pathophysiology. Marion[1] first postulated that PBNO stemmed from fibrous narrowing or hyperplasia at the bladder neck. Leadbetter[3] later theorized PBNO was a congenital obstruction caused by the fault of mesenchyme dissolution at the bladder neck or inclusion of abnormal quantities of non-muscular connective tissue leading to hypertrophic smooth muscle, fibrous contractures, and inflammatory changes. Similarly, Turner-Warwick and colleagues[4] proposed abnormal morphologic arrangement of the detrusor/trigonal musculature to be the cause of inefficient opening of the bladder neck.

More recently, experts have proposed a functional etiology of PBNO.[5] A normal volitional void begins with relaxation of the external sphincter; when pressure in the bladder equals that of the bladder neck normal voiding begins.[6] In patients with PBNO, the bladder neck pressure appears to remain elevated in comparison to intravesical pressure—some have suggested this may be due to excess striated muscle and/or muscle tone that extends from the external sphincter to the bladder neck,[7] whereas others have implicated increased sympathetic nervous system activity exerting an effect at the level of the bladder neck.[8]

Incidence and Prevalence

The true prevalence of PBNO is difficult to ascertain as most of the published epidemiologic studies focus on specific populations with obstruction and not the general population. As men age it becomes clinically difficult to distinguish PBNO from benign prostatic hyperplasia/benign prostatic obstruction (BPH/BPO) and it is possible that many men with PBNO are misdiagnosed with the later. Most of the prevalence studies for PBNO in men focus on younger populations where BPH is less of a confounding factor. In studies evaluating men under age 55 years with urodynamics for chronic voiding dysfunction, the prevalence of PBNO has been found to be between 33% and 45%.[9-11]

Data on the prevalence of PBNO in women is similarly limited. Most of the epidemiologic studies looking at the prevalence of PBNO in women do so in a subset of women presenting with symptoms of bladder outlet obstruction (BOO) and not the general population. It is estimated that 4.6% to 16% of women presenting with obstructive voiding have PBNO.[12-14]

The diagnosis of PBNO may be missed in children whose lower urinary tract symptoms (LUTS) are often presumed to stem from more common forms of dysfunctional voiding. In a retrospective study, 15% of neurologically normal children undergoing videourodynamic studies (VUDS) after failed response to first-line dysfunctional voiding treatment were found to have PBNO.[15] Another study of 650 children diagnosed with VUR who underwent UDS found fewer than 10% had PBNO.[16]

Presentation

Men and women with PBNO may present with a variety of symptoms including voiding symptoms (slow urinary stream, intermittent stream, incomplete emptying), storage symptoms (frequency, urgency, urgency incontinence, nocturia), and/or pelvic pain and discomfort. One study comparing the presentation of men and women with PBNO found both sexes reported voiding symptoms more frequently than storage symptoms, with men reporting pelvic pain more frequently than women (46% vs 15%).[17] Men with a pain-predominant presentation can often be misdiagnosed with chronic nonbacterial prostatitis or nonspecific pelvic pain.[18] On the contrary, a recent study found that 19% of all women referred for LUTS had some form of BOO, and that among women with all types of BOO, which included PBNO, the most common presenting symptom was urinary frequency.[19] Physical examination findings are generally unremarkable, with patients exhibiting a normal anal sphincter tone, lower extremity reflexes, and perianal sensation. Given the variable symptom presentation and low incidence of the condition, having a high clinical suspicion for PBNO is key to ensure the diagnosis is not overlooked.

Diagnosis

Uroflowmetry and post-void residual (PVR) volume are readily available screening tests in most urologic office settings to evaluate for urinary obstruction. Obstruction can be secondary to anatomic or functional causes (functional causes are sometimes categorized as neurologic and non-neurogenic; Table 1).[20] In men, prostate size should be assessed and in women a pelvic examination to assess for pelvic organ prolapse and other causes of urinary retention should be performed.

The diagnosis of PBNO is a VUD diagnosis. Pressure flow studies are utilized to diagnose obstruction and the addition of fluoroscopy allows for identification of obstruction specifically at the bladder neck, and, very often, incomplete bladder emptying is seen. Figures 1 and 2 demonstrate examples of VUD studies showing PBNO in men and women, respectively. Other causes of incomplete bladder emptying such as impaired bladder contractility, dysfunctional voiding, and Fowler's syndrome are not easily distinguished from PBNO when relying on tests such as physical examination, cystoscopy, noninvasive uroflowmetry, and PVR.[21] Some advocate defining PBNO based on fluoroscopic imaging regardless of pressure/flow criteria, assuming a void or attempt at voiding can be documented, as women with concurrent detrusor underactivity may not mount substantial detrusor pressures during an attempted void even in the presence of PBNO.[22]

There have been multiple attempts to define the urodynamic parameters of obstruction in women, with PBNO being just one potential cause of obstruction. Akikwala and colleagues[23] reviewed VUDS from 154 women with LUTS and compared five

TABLE 1

Etiologies of Obstructive Voiding

Anatomic	Functional
Bladder neck contracture	Non-neurogenic
Urethral stricture	Primary bladder neck obstruction[a]
Iatrogenic/ Prior anti-incontinence procedure	Dysfunctional voiding
Male specific	Fowler's syndrome
Benign prostatic obstruction	
Posterior urethral valves	Neurogenic[b]
Neoplastic: prostate cancer, urethral carcinoma	Sphincteric bradykinesia
Female specific	Detrusor-sphincter dyssynergia
Genitourinary prolapse	Spinal cord injury (suprasacral)
Neoplastic: urethral carcinoma, vaginal carcinoma, cervical carcinoma	Multiple sclerosis
Benign: urethral diverticulum, Skene's gland cyst/ abscess, Mullerian duct remnant, uterine leiomyoma	

[a]Primary bladder neck obstruction is most often thought of as a functional obstruction due to failure of the bladder neck to relax; however, those who theorize it may stem for hypertrophy of muscle may also classify it as an anatomic obstruction.
[b]Patient with a neurologic abnormality that can explain dysfunction.

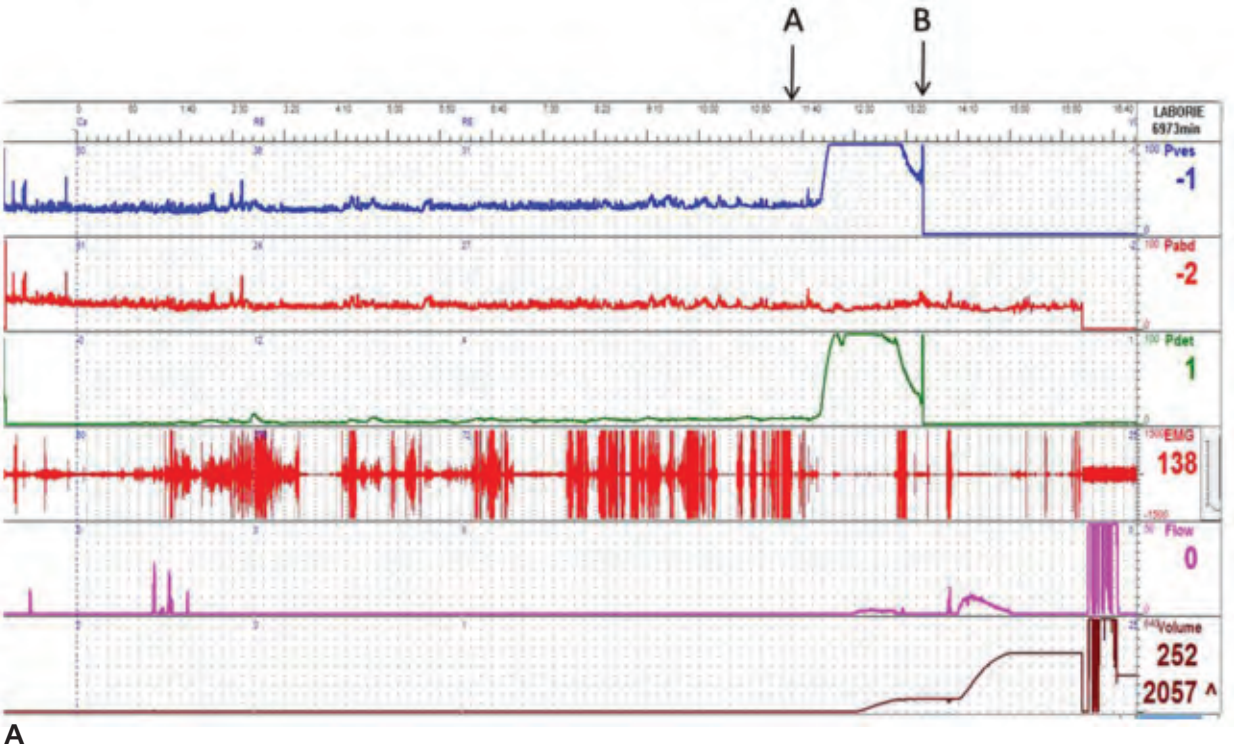
contemporary definitions of obstruction. They found that pressure flow cutoff values of ≤15 mL/s and detrusor pressure at maximal flow rate ($PdetQ_{max}$) ≥20 cm as proposed by Chassange and colleagues[24] and VUD criteria proposed by Nitti and colleagues[22] and defined as "radiographic evidence of obstruction between the bladder neck and distal urethra in the presence of a sustained detrusor contraction" correlated best with a clinical assessment of obstruction.

More recently, Solomon and colleagues[25] analyzed the VUD parameters of 535 women with LUTS and developed a nomogram for diagnosing BOO in women. They found that a cutoff of $PdetQ_{max}$ = $2*Q_{max}$ had a sensitivity of 0.94 and specificity of 0.93 for diagnosing obstruction. They also calculated a bladder outlet obstruction index (BOOI), defined as BOOI = $PdetQ_{max} - 2.2*Q_{max}$, and found BOOI <0 to be associated with a <10% probability of obstruction, BOOI >5 to be associated with a 50% chance of obstruction, and BOOI >18 to be associated with >90% chance of obstruction. Of note, only 9% of patients in this series had a functional obstruction, which should be considered when extrapolating findings to a patient with PBNO as they may have different voiding characteristics.

In men, nomograms used to classify obstruction in patients with BPH do not necessarily apply to PBNO. The Abrams-Griffiths nomogram was initially developed to help diagnose men with BOO and was later renamed BOOI, which is represented by the equation BOOI = Pdet @ $Q_{max} - 2\ Q_{max}$.[26] Based on their work, the International Continence Society subsequently published the ICS nomogram,[27] which divided men into categories according to their BOOI: BOOI > 40 = obstructed; BOOI 20−40 = equivocal; and BOOI < 20 = unobstructed. Of note, this nomogram is recommended for older men with LUTS suggestive of BPO. Although pressure-flow characteristics consistent with PBNO have been described in men, there is no universally accepted definition.

Yang and colleagues found that among 28 young men with PBNO, a sustained detrusor contraction during voiding with a Pdet ≥ 20 cm water, Q_{max} ≤15, and an obstructive flow pattern were associated with the diagnosis of PBNO. In men diagnosed with PBNO with a Q_{max} >15, an obstructive curve and a Pdet > 50 cm of water were also evident on pressure flow studies.[10]

Norlen and Blaivas looked at men with proximal urethral obstruction and found that these men had urodynamic findings with (1) a sustained detrusor contraction of adequate magnitude, (2) complete relaxation of the external urethral sphincter during detrusor contraction, (3) impaired flow, (4) radiographic or manometric evidence of obstruction at the level of the bladder neck, and (5) absence



A

B

Figure 1. Videourodynamic study of a man with primary bladder neck obstruction (PBNO). (A) Urodynamic tracing from a 48-year-old man with PBNO. At time point A he is granted permission to void and mounts a detrusor pressure of 112 cm water with a maximum urinary flow rate (Q_{max}) of 3mL/s. He is unable to empty with the catheter in place and at time point B the catheter is removed. Unintubated he has a Q_{max} of 11 mL/s with a voided volume of 410 mL and a post-void residual volume (PVR) of 97 mL. (B) Fluroscopic image obtained during attempted void with catheter in place demonstrating a closed bladder neck suggestive of PBNO.

of distal obstruction. They found it difficult to place a single numerical value on Pdet or uroflowmetry that was of adequate value to make the diagnosis, but in their series Pdet averaged 110 cm water (range, 30-200) and Q_{max} averaged 9.1 mL/s (range, 0-15 mL/s).[28]

In men and women with PBNO, electromyography (EMG) during micturition, or attempt at micturition, should demonstrate relaxation of the striated sphincter. Although increased EMG activity during micturition is the hallmark of dysfunctional voiding and/or detrusor external sphincter dyssenergia (in patient with a neurological lesion between the pons and the sacrum) and is not expected in PBNO, its presence should not in and of itself exclude the diagnosis of PBNO. This is because of false-positive results that can occur with surface EMG. Brucker and colleagues showed that 14.3% of women with PBNO with obstruction at the level of the bladder neck confirmed with fluoroscopy had increased EMG activity during micturition that was attributed to artifact of the surface electrode EMG (ie, wet pads, straining).[14]

In children, EMG lag time obtained by simultaneous uroflow/EMG has been advocated as a screening tool before proceeding with more invasive VUD testing. A time of greater 6 seconds between the onset of the EMG activity and the start of flow in symptomatic patients is highly correlated with the finding of PBNO on VUDS.[15]

Treatment of PBNO

Treatment options for men and women with PBNO include conservative management with watchful waiting, pharmacologic management, and surgical intervention.

Conservative Management

Watchful waiting is a reasonable treatment option for patients with minimal symptom bother, normal renal function, and low PVR. It is plausible that some older men with LUTS starting early in life and presumed to be caused by BPH may have long standing PBNO; however, longitudinal studies are lacking. Given there is little known about the natural history of PBNO and there exists the theoretical risk of worsening detrusor function in patients voiding against an obstructed bladder neck, we would advocate close follow-up with routine symptom check and non-invasive uroflowmetry. In any patient undergoing watchful waiting, worsening symptoms or a change in noninvasive tests should spur further evaluation and may lead to a change in management.[29]

Pharmacotherapy

The first-line pharmacologic treatment for PBNO in both men and women is usually considered to be α-blocker therapy. We know that stimulation of α-1-adrenergic receptors by norepinepherine via the sympathetic pathway increases tonic contraction of the bladder outlet.[30] α-Blockers are thought to work primarily by relaxing the smooth muscle of the bladder neck, although they may also work on the bladder through local or central mechanisms. Although there have been observational studies on the use of α-blockers in both men and women with PBNO, there are a lack of placebo-controlled trials for this indication their use is off-label.

Unlike α-blocker use in men with BPH, which has been used for decades with excellent results, α-blocker use in men with PBNO has more variable success. Yang and colleagues[10] found that in a group of 24 men younger than age 55 years with PBNO who were treated with 1 to 2 mg of doxazosin, 58% daily had a greater than 50% reduction in their International Prostate Symptom Score (IPSS). They found that men with higher mean detrusor pressures at Q_{max} and lower mean Q_{max} prior to treatment were more likely to respond. A recent prospective observational study of 39 men with PBNO looked at urodynamic parameters before and after initiation of α-blocker therapy and found that in addition to a 45% reduction in IPSS, treatment with 0.4 mg of tamsulosin daily lead to a statistically significant decrease in voiding pressures and BOOI and an increase in maximum flow rates.[31]

Although medication may be successful in some cases, many investigators have found long-term compliance to be an issue. Trockman and colleagues[32] found that although 67% of men treated with prazosin, 2 mg twice daily, or terazosin, 2 mg daily, reported improvement in symptoms, only 30% continued treatment for more than 1 year. Similarly, Nitti and colleagues found that although 58% of men treated with α-blockers had significant improvement in AUA Symptom Score, only 24% continued therapy for longer than 1 year.[9]

Success rates of α-blockers in women with PBNO are similar to those reported in men with a 70% improvement in subjective symptoms, flow rates, and PVRs.[33] A 2014 literature review looking at the use of α-blockers in women with LUTS and voiding dysfunction found most of the available literature to be limited due to small sample size, inconsistent study design, and short duration.[34] In our experience, we have anecdotally noted that women may have more difficulty tolerating the medication due to side effects such as fatigue and dizziness.

Few studies have been conducted on the use of α-blockers in children. The largest reviewed 51 children diagnosed with PBNO treated with α-blockers for a minimum of 1 year. Eighty-five percent of children reported significant symptom relief, a significant improvement measured on EMG lag time, and maximum and average uroflow rates; the medication was well tolerated. The authors note that although improvement in uroflow parameters was seen after 4 to 6 weeks, a significant number of patients did not experience symptomatic improvement until 6 months.[35]

Intradetrusor injection of OnabotulinumtoxinA (onaBoNT$_A$) causes muscle paralysis by preventing the release of presynaptic vesicles containing acetylcholine and is US Food and Drug Administration (FDA) approved in the urinary tract only for medication-refractory neurogenic and non-neurogenic detrusor overactivity. There have been some small studies looking at the use of onaBoNT$_A$ in the bladder neck and/or sphincter in patients with chronic urinary retention and difficulty urinating, although only a single study has looked specifically at its use in patients with PBNO. Sacco and colleagues[36] found that Q_{max}, PVR, and patient reported outcomes in 35 men with PBNO improved after onaBoNT$_A$ injection. IPSS decreased from 21.9 to 7.8 at 2 months and nearly 80% of participants reporting willingness to repeat the procedure. Further studies are needed on the use of onaBoNT$_A$ for PBNO; however, we know from its mechanism of action that its proposed use require re-injection at regular intervals given an average duration of action between 8 and 11 months.

Surgical Intervention

The traditional surgical management of PBNO in both men and women is endoscopic incision of the bladder neck. Although dilation has been used in cases of bladder neck narrowing due to stricture, PBNO is a functional obstruction for which the mainstay of treatment involves incision or resection of the obstructing tissue. Table 2 summarizes the major series describing surgical intervention for PBNO.

Men

Surgical treatment of PBNO involves unilateral or bilateral incision of the bladder neck to open the annular ring of obstructing tissue. The procedure is usually performed under general or spinal anesthesia. Most commonly this is done using a 24 or 26Fr resectoscope and a Collins knife or a laser. Incisions are typically made at the 5 or 7 o'clock positions, or in some cases in the 2 and 10 o'clock positions.

In men, the main concern with transurethral incision of the prostate (TUIP)/transurethral incision of the bladder neck (TUIBN) is the development of retrograde ejaculation. Retrograde ejaculation after TUIP/TUIBN has been reported in 27% to 100% of men undergoing bilateral bladder neck incision, but is much less likely to occur when performing a unilateral incision, reported in 0to 35% of patients.[18,28,32,37] Starting the incision near the ureteral orifice and carrying this downward about 0.5 to 1.0 cm proximal to the verumontanum, allowing for preservation of the supramontal prostate, has been shown in small series to preserve antegrade ejaculation in all patients.[38]

In a study of bilateral incision in men, Trockman and colleagues found that mean Q_{max} increased significantly from 8.2 to 26.7 mL/s and mean AUA symptom scores improved from 17.1 to 4.3.[32] To date there are no studies directly comparing outcomes of unilateral verus bilateral TUIP in men, although there have been multiple studies demonstrating success of unilateral incision with improvements of Q_{max} from 9.2 to 15.7 mL/s[18] with sustained improvement in symptom scores up to 1 year after surgery.[39]

Children may also benefit from bladder neck incision, although it is more controversial due to the consequence of retrograde ejaculation for younger patients. In response, superficial bladder neck incision (SBNI) limited to a unilateral 2 to 3 mm has been utilized. A study of long-term effects from this procedure found that of 40 men queried 15 or more years after SBNI either for primary PBNO or BNO secondary to PUV all had antegrade ejaculation, with 10.8% reporting reduced ejaculatory volume.[40]

Women

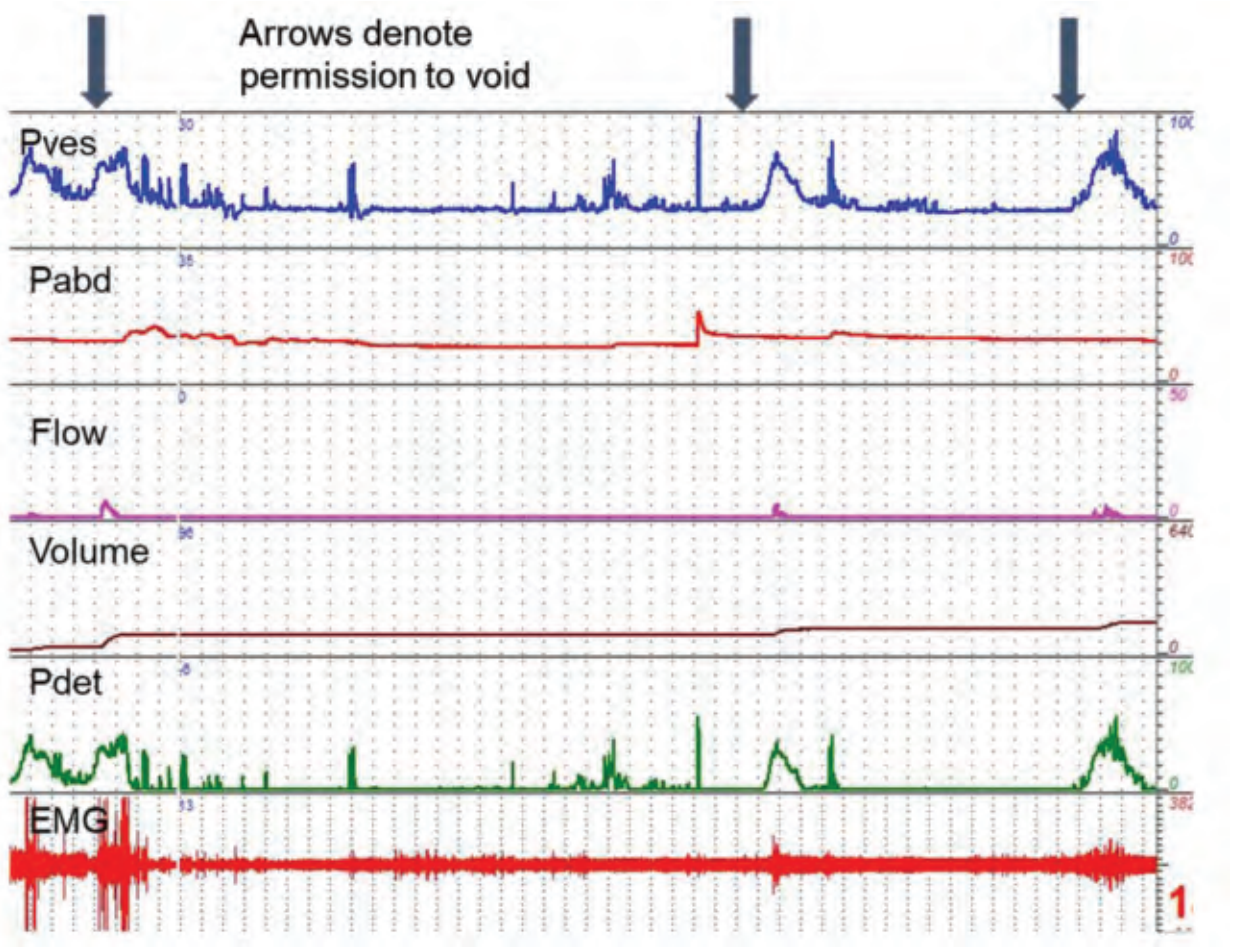
In women, TUIBN is performed in a similar fashion to me.The extent of the incision extends from just inside the bladder neck through the proximal third of the urethra, making sure to preserve continence by avoiding the external urethral sphincter. Turner-Warwick and colleagues[4] was the first to describe TUIBN in women in 1973. Their technique involved a single incision at 12 o'clock to prevent complications of stress incontinence and fistula. At present there is no standard endoscopic incision in women. Sites of incision have been proposed at 5 and 7 o'clock,[41] 2 and 10 o'clock,[42] and 4 and 8'clock done in a staged fashion with unilateral incision and a contra-lateral incision on an as needed basis.[43]

TABLE 2

Major Series of Surgical Intervention for Primary Bladder Neck Obstruction

Authors	Year	Study Population	Intervention	Outcome	Mean Follow-up
Kochakarn W, Lertsithichai P[39]	2003	Retrospective analysis of 35 men with PBNO	Unilateral transurethral incision of bladder neck	ΔIPSS 32.1 → 14.4 ($P < 0.001$) ΔQ_{max} 7.8 → 15.2 mL/s ($P < 0.001$) ΔQOL 5 → 1 ($P < 0.001$) Δ Sperm count 59.2 → 18.1 million/mL ($P < 0.001$)	1 y
Blaivas JG et al[12]	2004	Retrospective analysis of 7 women diagnosed with PBNO on VUDS	Transurethral resection of bladder neck and proximal urethra	ΔQavg 6 → 30 mL/s ($P < 0.03$) ΔVV 194 → 416 mL/s ($P < 0.06$) ΔPVR 680 → 173 ($P < 0.05$)	3 y
Yang SS et al[38]	2008	Prospective study of 33 men diagnosed with PBNO on VUDS	Transurethral incision of bladder neck with preservation of supramontanal tissue	Antegrade ejaculation preserved in all patients ΔIPSS 20.7 → 5.9 ($P < 0.01$) ΔQ_{max} 10.7 → 19.2 mL/s ($P < 0.01$) ΔQOL 4.2 → 2.3 ($P < 0.01$) ΔPVR 107 → 48 ($P < 0.01$)	2 y
Jin XB et al[45]	2012	Retrospective analysis of 30 women diagnosed with PBNO on VUDS	Transurethral incision of bladder neck at 3-, 6-, 9-, and 12-o'clock positions	ΔIPSS 23.3 → 5.9 ($P < 0.001$) ΔQ_{max} 7.6 → 17.5 mL/s ($P < 0.001$) ΔQOL 4.4 → 2.1 ($P < 0.001$) ΔPVR 185 → 29 ($P < 0.01$) Postoperatively 1 patient (3%) experienced SUI relieved with physical therapy	5 y
Zhang P et al[42]	2014	Retrospective analysis of 74 women diagnosed with PBNO on VUDS	Transurethral incision of bladder neck; most incisions used 5- and 7-o'clock positions; the last 21 patients had 2- and 10-o'clock incisions	ΔIPSS 22.9 → 7.9 ($P < 0.01$) ΔQ_{max} 9.8 → 18.8 mL/s ($P < 0.01$) ΔQOL 4.1 → 2.4 ($P < 0.01$) ΔPVR 115 → 23 ($P < 0.01$) Postoperative complications included hemorrhage, need for re-BNI, vesicovaginal fistula, SUI, and urethral stricture; no fistula was observed in patients with incision at 2- and 10-o'clock positions	2.3 y

BNI, bladder neck incision; IPSS, International Prostate Symptom Score; PBNO, primary bladder neck obstruction; PVR, post-void residual volume; Qavg, average flow rate; Q_{max}, maximum urinary flow rate; QOL, quality of life; SUI, stress urinary incontinence; VUDS, videourodynamic studies; VV, voided volume.



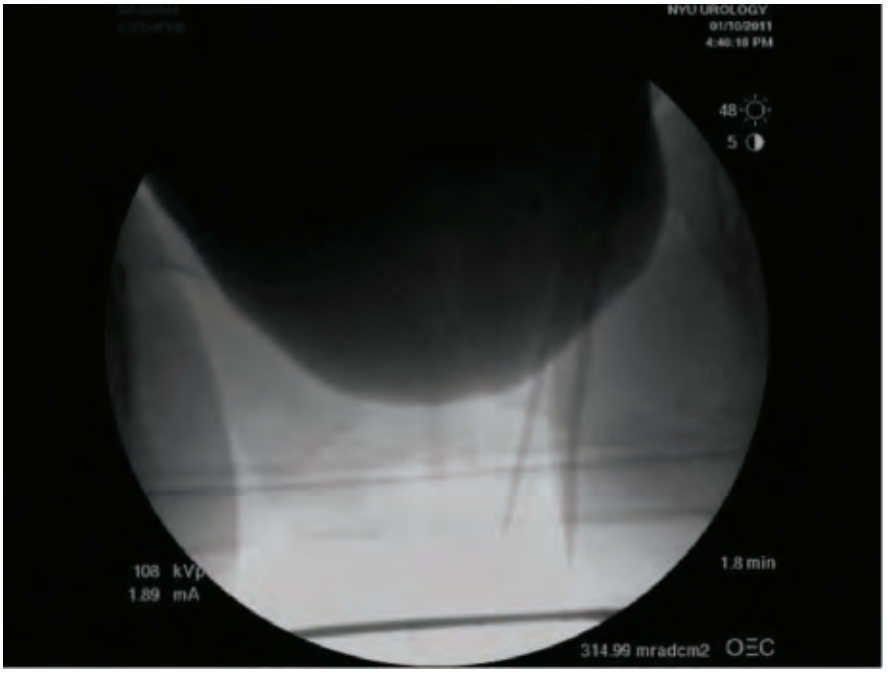


Figure 2. Videourodynamic study of a woman with primary bladder neck obstruction (PBNO). (A) Videourodynamic tracing of a 58-year-old woman with urinary frequency, urgency, and slow urinary stream who was diagnosed with PBNO. She attempted multiple voids during this study mounting a maximum urinary flow rate (Q_{max}) of 5 mL/s and a detrusor pressure (Pdet) at Q_{max} of 38 cm water. (B) Fluoroscopic image during attempted void demonstrating obstruction at the bladder neck.

Zhang and colleagues[42] reported the largest retrospective series of 84 women undergoing TUIBN; they initially used a 5 and 7 o'clock technique but transitioned to 2 and 10 o'clock after their first 63 patients due to a 5% risk of both vesicovaginal fistula and stress urinary incontinence identified during the initial technique. Advocates for the 5 and 7 o'clock technique have reported no risk of these complications when incising "just enough" to relieve the obstruction and do not believe it is necessary to cut to the fat to achieve a successful outcome that may put the patient at higher risk for fistula and incontinence.[44]

Zhang and colleagues[42] reported a successful outcome of TUIBN without serious complication in 84.5% of women at an average follow-up of 27.4 months. They found TUIBN in women to be associated with a decrease in IPSS score from 22.9 to 7.9 ($P < 0.01$) with an improvement in Q_{max} from 9.75 to 18.75 mL/s ($P < 0.01$). Another series of 30 women with 5 years of follow-up found improvement in IPSS score from 23.3 to 5.9 and Q_{max} from 7.61 to 17.53mL/s.[45]

Future Perspectives

PBNO is not well understood and there remains much to be learned about the natural history, etiology, and presentation of this disorder in both men and women. Clinicians should consider PBNO as a possible cause of symptoms in men and women presenting with LUTS, obstructive voiding symptoms, or pain. VUDS are required to diagnose PBNO although uniformly accepted diagnostic criteria are also needed.

Although there is a lack of data looking at disease progression and risk factors for developing detrusor underactivity and/or renal failure due to prolonged obstruction, early diagnosis and treatment of PBNO should help to ameliorate these sequelae. In order to fully evaluate the effects of both pharmacologic and surgical treatment, randomized, placebo-controlled trials are needed. ■

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MAIN POINTS

- Primary bladder neck obstruction (PBNO) is not well understood and there remains much to be learned about the natural history, etiology, and presentation of this disorder in both men and women.
- Clinicians should consider PBNO as a possible cause of symptoms in men and women presenting with LUTS, obstructive voiding symptoms, or pain. Videourodynamic studies (VUDS) are required to diagnose PBNO although uniformly accepted diagnostic criteria are also needed.
- Although there is a lack of data looking at disease progression and risk factors for developing detrusor underactivity and/or renal failure due to prolonged obstruction, early diagnosis and treatment of PBNO should help to ameliorate these sequelae. In order to fully evaluate the effects of both pharmacologic and surgical treatment, randomized, placebo-controlled trials are needed.

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