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# Management of Benign Prostatic Hyperplasia

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## Keywords

lower urinary tract symptoms, adrenergic alpha-antagonists, 5-alpha reductase inhibitors, phosphodiesterase type 5 inhibitors, transurethral resection of prostate

## Abstract

Benign prostatic hyperplasia (BPH) and associated lower urinary tract symptoms (LUTS) commonly affect older men. Age-related changes associated with metabolic disturbances, changes in hormone balance, and chronic inflammation may cause BPH development. The diagnosis of BPH hinges on a thorough medical history and focused physical examination, with attention to other conditions that may be causing LUTS. Digital rectal examination and urinalysis should be performed. Other testing may be considered depending on presentation of symptoms, including prostate-specific antigen, serum creatinine, urine cytology, imaging, cystourethroscopy, post-void residual, and pressure-flow studies. Many medical and surgical treatment options exist. Surgery should be reserved for patients who either have failed medical management or have complications from BPH, such as recurrent urinary tract infections, refractory urinary retention, bladder stones, or renal insufficiency as a result of obstructive uropathy.

## INTRODUCTION

Benign prostatic hyperplasia (BPH) occurs commonly in older men, with historical reports of up to 50% prevalence in men over the age of 50 years and increasing incidence with increasing age (1, 2). BPH anatomically compresses the urethra, causing increased bladder outlet resistance. This results in lower urinary tract symptoms (LUTS), which include symptoms of difficult voiding (hesitancy, straining, weak stream, sensation of incomplete emptying) and irritable voiding (frequency, urgency, urge incontinence) (3). Over the last 20 years, advances in medical treatments for BPH/LUTS have led to a corresponding decrease in utilization of surgical interventions for this disorder (4). As a result, understanding how to diagnose and treat BPH/LUTS has become increasingly important for primary care physicians and providers, particularly considering the strong relationship between LUTS, metabolic syndrome, and other cardiovascular risk factors (5, 6).

## PATHOGENESIS

BPH is a result of hyperplasia of both epithelial and stromal tissues and predominantly affects the transition zone of the prostate gland (7, 8). **Figure 1** illustrates the zonal anatomy of the prostate. Hyperplastic nodules cumulatively compress the urethra, causing mechanical obstruction to urinary outflow and bladder detrusor muscle irritability as a result of increased resistance. Although the exact mechanism for the development of BPH is unknown, metabolic, hormonal, and inflammatory mechanisms have been proposed. Basic understanding of the hypothesized metabolic and hormonal mechanisms for BPH is important, as medical treatments for BPH are directed at these pathways.

Metabolic mechanisms are based on the observation that cardiovascular risk factors in men are associated with BPH and LUTS. The effect of systemic atherosclerotic vascular damage on the pelvic vasculature supplying the bladder and prostate is thought to be the common link between cardiovascular risk factors, including metabolic syndrome, and BPH/LUTS (9–11). Preclinical models have revealed various mediators of tissue ischemia caused by atherosclerosis. Hypoxic tissue conditions have been shown to cause cultured human prostatic stromal cells to produce increased levels of growth factors, which lead to prostate epithelial hyperplasia (12). Additionally, improved blood flow and tissue perfusion that occurs with nitric oxide–generating medications have been found to promote smooth muscle relaxation in the prostate, reduce nonvoiding bladder

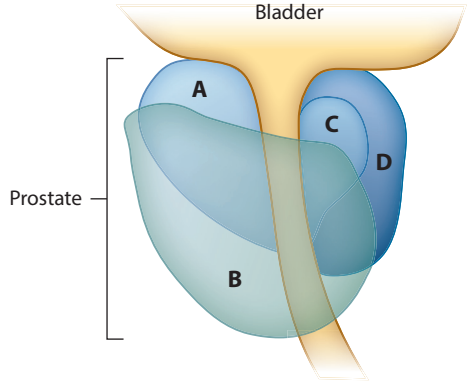


**Figure 1**

Prostate anatomy by zone. A, central zone; B, peripheral zone; C, transition zone; D, fibromuscular zone.

detrusor contractions, and have an antiproliferative effect on lower urinary tract cells cultured from men with BPH (13, 14). Components of metabolic syndrome have also been demonstrated to cause overactivation of the autonomic nervous system, which may contribute to LUTS and BPH as a result of alpha-adrenoceptor-mediated smooth muscle contraction of the bladder neck and prostate, as well as to the promotion of prostate growth (15, 16).

Androgens, namely testosterone (T) and dihydrotestosterone (DHT), play an important role in the development of the normal prostate as well as BPH and prostate cancer. T is produced by the testes and adrenal glands and is converted to DHT by the enzyme 5-alpha reductase. DHT has much greater affinity for the androgen receptor than T has. Both T and DHT drive prostate growth; however, serum and tissue concentrations of T and DHT are not clearly elevated in men with BPH compared to normal controls (17). Some investigators have suggested that estrogen and the increasing estrogen-to-androgen ratio that occurs with normal aging may also play a role in the pathogenesis of BPH (18).

Chronic inflammation may also contribute to BPH development. Preclinical models have demonstrated that proinflammatory stimulators drive prostatic epithelial cell growth (19). Clinical studies have found inflammatory infiltrates in the histologic examination of BPH tissues (20), and that the presence of intraprostatic inflammation was associated with LUTS severity and risk of developing acute urinary retention (AUR) (21, 22). The chronic inflammation associated with BPH is believed to be a result of autoimmune activation against prostatic tissue, which drives further cytokine production, resulting in prostate stromal growth (23).

**T:** testosterone

**DHT:** dihydrotestosterone

**AUR:** acute urinary retention

**AUA:** American Urological Association

**UTI:** urinary tract infection

## DIAGNOSIS

### History

The medical history is a critical component in the accurate diagnosis of BPH. Type and severity of symptoms must be assessed, but more importantly, LUTS resulting from BPH must be distinguished from LUTS resulting from another pathologic process.

The American Urological Association (AUA) symptom score is a validated questionnaire that is used to objectively measure LUTS. Seven symptoms—incomplete emptying, frequency, intermittency, urgency, weak stream, straining, and nocturia—are scored from 0 to 5. Total scores from 0 to 7 are considered mild, scores from 7 to 19 are considered moderate, and scores of 20 to 35 are considered severe. The reduction in quality of life due to LUTS is also qualitatively assessed and included (3). Dysuria may be a secondary indicator of BPH, as bladder outlet obstruction may lead to urinary stasis and resultant urinary tract infection (UTI).

Medications that the patient may be taking for other conditions may contribute to or exacerbate LUTS. Alpha-adrenergic agonists (e.g., decongestants with pseudoephedrine) can increase bladder outlet resistance by causing bladder neck and prostatic smooth muscle contraction. Anticholinergic medications and calcium channel blockers can prevent complete bladder emptying by promoting bladder relaxation. Diuretics (e.g., furosemide or thiazides) can cause urinary frequency when taken during the day and nocturia when taken at bedtime.

Although BPH is the most likely cause of LUTS in men over age 50, other medical problems may cause LUTS as well. Congestive heart failure may cause significant nocturia due to mobilization of lower-extremity edema in the evening. Diabetes can cause urinary frequency due to diuresis as well as irritative symptoms due to autonomic neuropathy of the bladder (24, 25). Neurologic disorders may be associated with neurogenic bladder, causing LUTS as a result of reduced bladder contractility or impaired communication between the bladder and urethral sphincter mechanism. Examples of disorders resulting in neurogenic bladder include Parkinson's disease,

multiple sclerosis, brain tumors, any spinal cord disorder (e.g., syringomyelia, spinal cord injury, spina bifida), and any peripheral nerve disease (e.g., diabetes, alcoholism, vitamin B12 deficiency).

Anatomic abnormalities in the lower urinary tract, such as urethral strictures or bladder neck contractures, may result in LUTS as well. Typically these patients have a prior history of lower urinary tract manipulation or instrumentation, penile or perineal trauma, or recurrent sexually transmitted infections. History of hematuria may be associated with these conditions but may also be a sign of lower urinary tract malignancy. Although unlikely to present with LUTS, bladder and prostate cancer should be considered in patients with rapid-onset LUTS (as opposed to the slowly progressive LUTS associated with BPH) or severe LUTS refractory to medical management.

## Physical Examination

For patients with LUTS and suspected BPH, digital rectal examination (DRE) and a focused neurologic examination should be performed. Although DRE underestimates prostate volume, DRE estimates are appropriately correlated to prostate size (26). An enlarged, smooth, and symmetric prostate are DRE findings consistent with BPH. Other findings, such as asymmetry, firmness, hard areas, or discrete nodules, should raise an index of suspicion for prostate cancer. Additionally, poor anal sphincter tone found on DRE may suggest an underlying neurologic diagnosis. A focused neurologic examination should be performed in order to evaluate for neurogenic causes of LUTS. This should include assessment of lower-extremity strength, sensation, and reflexes as well as perineal sensation.

## Laboratory Testing

**Urinalysis.** Urinalysis should be performed in men with LUTS to evaluate for glucosuria, pyuria, and hematuria. As mentioned above, diabetes may contribute to LUTS and should be distinguished from BPH. Pyuria is suggestive of UTI, which should be further evaluated with urine culture and treated with antibiotics. Furthermore, significant BPH causing recurrent UTI may require more aggressive treatment. The finding of hematuria (including asymptomatic and microscopic hematuria) requires additional testing to exclude genitourinary malignancies (27).

**Serum creatinine.** Serum creatinine measurement may be considered during the initial evaluation of patients with LUTS to ensure that renal insufficiency has not occurred as a result of obstructive uropathy. Although obstructive uropathy is a potentially serious complication of long-standing and untreated BPH, elevations in serum creatinine are much more commonly a result of medical renal disease. Patients with obstructive uropathy require early urinary decompression with urethral or suprapubic catheterization, stabilization during postobstructive diuresis, monitoring of kidney function recovery, and consideration of surgical BPH intervention.

**Prostate-specific antigen.** Elevations in prostate-specific antigen (PSA) poorly discriminate between BPH and prostate cancer and may be the result of both (28). Historical evidence for this connection exists: 83% of patients diagnosed with prostate cancer were found to have histologic evidence of BPH, and 10–15% of men receiving surgical intervention for BPH were incidentally diagnosed with prostate cancer (29).

Especially considering the recent controversy surrounding prostate cancer screening with PSA (30), serum PSA measurement in the evaluation of BPH should not be a requirement. This is especially true for patients where early diagnosis of prostate cancer would provide no benefit (generally men with less than ten-year life expectancy). Benefits of PSA measurement for patients

with BPH nevertheless do exist. Baseline PSA measurements prior to and after intervention may provide a surrogate for efficacy of treatment, as PSA and prostate volume have been found to be strongly correlated (31). A serum PSA level of ≥1.5 generally correlates with prostatic enlargement (size ≥30 cc) (32). Additionally, PSA measurements have been found to be strong predictors for the risk of developing AUR and the need for surgical intervention for BPH (33).

**PVR:** post-void residual

Modifications to the PSA assay have been developed to better distinguish BPH from prostate cancer. Higher PSA density (serum PSA divided by prostate volume measured by transrectal ultrasound) and PSA velocity (annualized rate of rise in PSA) have been demonstrated to correlate with a diagnosis of prostate cancer (34, 35). Free to total PSA ratios were used in the past to help differentiate PSA elevations due to BPH and prostate cancer, with lower free PSA percentage associated with an increased likelihood of prostate cancer (36). By combining other forms of PSA and other human kallikreins, the prostate health index (total PSA, free PSA, [−2] proPSA) and the 4K score (total PSA, free PSA, intact PSA, kallikreins-related peptidase 2) have also demonstrated improvements in distinguishing rises in PSA due to clinically significant prostate cancer (37, 38).

These modifications to PSA testing and newer PSA-related tests likely fall within the purview of urologists rather than the primary care physician's initial evaluation of LUTS. However, if serum PSA measurement in a patient presenting with LUTS is considered, it is important to have a thoughtful discussion with the patient regarding the role of PSA in the diagnosis of prostate cancer and the potential risks and benefits associated with diagnosis and treatment of PSA-detected prostate cancer.

## Other Testing

**Urine cytology.** Urine cytology is not recommended for routine evaluation of LUTS secondary to BPH but may be considered for patients who present with irritative symptoms refractory to medical treatment, have risk factors for bladder cancer (e.g., smoking, occupational exposures to industrial chemicals), and have a history of microscopic or gross hematuria.

**Transrectal ultrasound.** Although transrectal ultrasound provides a more accurate measurement of prostate volume than does DRE, this is not recommended during the initial evaluation of LUTS related to BPH. As discussed below, prostate size does impact medication choices during medical management of BPH; however, an exact measurement of prostate volume is not necessary. Surgical decision making can be affected by prostate size, so transrectal ultrasound may be considered prior to surgical intervention.

**Post-void residual.** Post-void residual (PVR) is a measure of fluid remaining in the bladder immediately after completion of urination and can be measured by ultrasound or by catheterization. Although it seems intuitively useful, studies have demonstrated that PVR is inconsistent for men with BPH (39). More importantly, no correlation has been found between PVR and severity of LUTS (40), and PVR provides little prognostic value in the progression of BPH (41).

**Cystourethroscopy.** Although endoscopic evaluation of the lower urinary tract has been found to correlate well to the degree of outlet obstruction on pressure-flow studies (42), cystourethroscopy for all men presenting with LUTS would be expensive and only rarely alter management (43). Cystourethroscopy should be reserved for patients who have a specific indication for lower urinary tract imaging, such as a history of hematuria, positive urinalysis for hematuria, positive urine cytology, or history of trauma, urethritis, or reason to suspect an anatomic abnormality.

**Urodynamic studies.** Maximum urinary flow rates below 15 ml per second are suggestive of bladder outlet obstruction from BPH; however, a patient with a poorly contractile bladder or neurogenic bladder would demonstrate similarly reduced flow rates. As a result, response to medical or surgical therapy has been shown to be independent of baseline maximum urinary flow rate (44).

Urodynamic studies (simultaneous measurement of bladder pressure with a small catheter and urinary flow rate) can help to discern reduction in urinary flow rates due to obstruction, as these patients will have elevated voiding pressures (45). Unfortunately, urodynamic studies have also been found to correlate poorly to symptom severity (46). Thus, urodynamic studies should be reserved for complicated cases of LUTS, such as instances when components of the clinical evaluation are in conflict, when neurologic disease is present, or when little to no benefit occurs with treatment.

## TREATMENT

### Watchful Waiting

As treatment of BPH-related LUTS is aimed at improvement of quality of life, watchful waiting is recommended by both the AUA and the European Association of Urology for patients with mild symptoms (AUA symptom score <8) or moderate-to-severe symptoms with minimal impairment in quality of life (47, 48). Watchful waiting should include education, modification of lifestyle factors (e.g., weight loss, increase in physical activity, and reduction of caffeine and alcohol intake), and yearly re-evaluation. Patients should be counseled appropriately on their risk of AUR, which is increased for those with moderate-to-severe symptoms, diminished urinary flow rates, larger prostate volumes, increasing serum PSA, and older age (33, 49, 50).

Watchful waiting is inappropriate for patients with complications of bladder outlet obstruction related to BPH—such as renal insufficiency due to obstructive uropathy, recurrent UTI, bladder stones, and refractory urinary retention (failed at least one voiding trial after catheter removal).

### Medical Management

**Table 1** summarizes the medications approved by the Food and Drug Administration (FDA) for the treatment of BPH.

**Alpha blockers.** Alpha-adrenergic antagonists, or alpha blockers, relieve LUTS by reducing smooth muscle tone in the prostate and bladder neck (15). Alpha blockers have been demonstrated to significantly improve symptom scores (both irritative and obstructive symptoms), quality of life, and urinary flow rates, but they do not reduce the risk of AUR or risk of requiring BPH-related surgery (51, 52). Common side effects include dizziness, fatigue, orthostatic hypotension, nasal congestion, and retrograde ejaculation. Alpha-1A-adrenergic receptors predominate in the lower urinary tract, while alpha-1B and alpha-1D receptors are found in blood vessels, the central nervous system, and nasal passages. As a result, alpha-1A-selective alpha blockers (tamsulosin and silodosin) have reduced systemic side effects compared to nonselective alpha blockers (alfuzosin, doxazosin, prazosin, terazosin), but they register higher rates of retrograde ejaculation. Nonselective alpha blockers have historically been titrated up to their maximal dosage based on orthostatic blood pressure in order to minimize adverse medication effects. However, the alpha-1A-selective alpha blockers do not require dose titration and are given once daily. Serum PSA is not affected by alpha blockers, as their mechanism of action does not affect prostate volume.

**Table 1 FDA-approved medications for the treatment of benign prostatic hyperplasia**

| Alpha blockers (nonselective) | Alpha blockers (alpha-1A selective) | 5-Alpha reductase inhibitors | Phospho-diesterase type 5 inhibitors | Combination products |
|---|---|---|---|---|
| Alfuzosin extended-release 10 mg daily | Tamsulosin 0.4 mg daily | Dutasteride 0.5 mg daily | Tadalafil 5 mg daily | Dutasteride 0.5 mg/ tamsulosin 0.4 mg daily |
| Doxazosin immediate-release 1 mg daily (titrate up to 8 mg daily) | Silodosin 8 mg daily | Finasteride 5 mg daily | | |
| Doxazosin extended-release 4 mg daily (titrate up to 8 mg daily) | | | | |
| Prazosin 1 mg twice to three times daily (titrate up to 15 mg total daily) | | | | |
| Terazosin 1 mg daily (titrate 2 mg, 5 mg, then 10 mg daily) | | | | |

**5-Alpha reductase inhibitors.** 5-Alpha reductase inhibitors (5-ARI) block the conversion of T to DHT by inhibition of type I (peripheral) and/or type II (lower genitourinary tract) 5-alpha reductase. Finasteride inhibits type II 5-alpha reductase, whereas dutasteride inhibits types I and II 5-alpha reductase. Reduction in DHT levels results in prostate volume reduction of 20–25% and decreases in serum PSA of ~50% after one year. For men with LUTS and enlarged prostates (typically defined as >30 g), 5-ARI use has been demonstrated to significantly improve symptoms, improve urinary flow rate, reduce the risk of AUR, and reduce the risk of requiring BPH-related surgery (31, 53). In a head-to-head comparison, dutasteride and finasteride performed similarly well in reducing prostate volume, improving symptoms, and improving urinary flow rates at 12 months (54). Although uncommon (<5% incidence), the sexual side effects of 5-ARI can be significantly bothersome, including impotence, decreased libido, decreased ejaculate volume, and gynecomastia.

**Phosphodiesterase type 5 inhibitors.** Phosphodiesterase type 5 inhibitors (PDE5-I) are well known to be effective in the treatment of erectile dysfunction. PDE5-I increase nitric oxide signaling in genitourinary tract tissues, which causes calcium-dependent relaxation of endothelial smooth muscle and increased blood flow. By the same mechanism of action, PDE5-I have been found to improve BPH based on preclinical studies (13, 14). Five randomized placebo-controlled trials have demonstrated the efficacy of PDE5-I (sildenafil, tadalafil, and vardenafil) on LUTS related to BPH, with significant improvements in AUA symptom scores, but no effect on urinary flow rate or PVR (55–59). Currently, daily tadalafil is the only PDE5-I that is approved by the FDA for treatment of BPH. Although these medications are expensive, patients with concomitant erectile dysfunction may derive the most benefit from PDE5-I treatment for their LUTS related to BPH.

**Combination therapy.** Treatment of LUTS related to BPH with a combination of the above-described medications is common in practice. Two studies have shown alpha blocker and 5-ARI treatment to be synergistic for men with demonstrable prostatic enlargement. The Medical Therapy of Prostatic Symptoms (MTOPS) study found that finasteride and doxazosin significantly reduced the risk of AUA symptom score rise, AUR, urinary incontinence, renal insufficiency, or recurrent UTI compared to either drug alone (32). The Combination of Avodart and Tamsulosin

(CombAT) study found that dutasteride and tamsulosin significantly reduced the risk of BPH clinical progression compared to either drug alone (60). The Veterans Affairs (VA) Cooperative Studies Benign Prostatic Hyperplasia Study Group found that a terazosin and finasteride combination was no more effective than terazosin alone (61). However, the VA Cooperative study had no threshold for prostatic enlargement in their study population. Secondary analyses of the MTOPS study found that men with a prostate volume of >25 g benefited from the added 5-ARI, and the CombAT study included only men with a prostate volume of ≥30 g.

Alpha blocker and PDE5-I combination treatment has been studied in randomized trials in men with concomitant erectile dysfunction and found to provide greater benefit than either therapy alone for LUTS related to BPH. In studies of alfuzosin with tadalafil, tamsulosin with sildenafil, and tamsulosin with vardenafil, combination therapy was found to provide greater improvements in AUA symptom scores as well as urinary flow rates than alpha blockers alone (62–64). The statistical significance of the improvement in qualitative and quantitative outcomes with combination therapy differed across studies and may be related to their small sample sizes.

**Supplements.** Numerous alternative medicines have been proposed for the treatment of LUTS related to BPH, including saw palmetto, stinging nettle, pumpkin seed, and African star grass. None of these supplements have demonstrated a significant benefit for patients with BPH. One supplement, saw palmetto, has been studied in randomized trials and found to provide no benefit compared to placebo in AUA symptom score or urinary flow rates for men with BPH (65, 66).

## Surgical Management

Surgical intervention for LUTS related to BPH should be considered for patients who have failed medical management or who have complications from bladder outlet obstruction due to BPH—such as renal insufficiency due to obstructive uropathy, recurrent UTI, bladder stones, and refractory urinary retention (failed at least one trial of void). For patients with moderate-to-severe LUTS, surgical intervention may be considered as the initial treatment based on coexisting medical conditions and patient preference.

Baseline urinary flow rates and PVR should be considered prior to surgical management, both to improve counseling and as a comparison to post-treatment measurements to assess effectiveness of intervention. In patients who failed medical management and whose urinary flow rates and PVR are not indicative of obstruction, pressure-flow studies may be useful. Cystourethroscopy and transrectal ultrasound may be beneficial in surgical planning as prostate size and anatomic configuration may alter treatment modality.

**Transurethral resection of prostate.** The gold standard for surgical treatment of BPH remains transurethral resection of prostate (TURP), which has demonstrated significant symptom improvement (67–69). Electrocautery devices are used endoscopically to resect prostate tissue per urethra. Patients require inpatient admission after TURP for monitoring of hemostasis and for monitoring of possible transurethral resection syndrome. The risk of this syndrome has been mitigated in recent years with the advent of bipolar resection devices that are compatible with normal saline irrigation. The success of TURP is durable with a <1% risk per year of requiring a repeat procedure. Adverse events that are seen with some frequency include failure to void postoperatively, requiring catheter replacement; hemorrhage, requiring a blood transfusion; and UTI (69). As portions of the bladder neck and internal sphincter are resected, retrograde ejaculation is expected following TURP, and patients should be counseled about this. Aside from retrograde

ejaculation, sexual dysfunction is rare. Urinary incontinence due to inadvertent distal resection into the external sphincter is rare but can be a devastating complication.

**Transurethral incision of prostate.** At 12 months postoperatively, transurethral incision of prostate (TUIP) has been found to be just as effective as TURP in symptomatic improvement (70). TUIP is performed endoscopically by making one or more longitudinal incisions into the prostate with electrocautery. TUIP has several advantages over TURP, including lower complication rates, fewer blood transfusions, decreased risk of retrograde ejaculation, shorter operative time, and shorter hospital stay. In the long term, TUIP is likely not as durable as TURP given that reoperation rates were found notably higher for TUIP in two meta-analyses (9.3% versus 5.5% and 15.9% versus 2.6% in References 70 and 71, respectively).

**Simple prostatectomy.** Simple prostatectomy is an extirpative surgery performed via a lower abdominal incision or laparoscopic approach. It is typically reserved for patients with a prostate size of >100 g. Significant reductions in operative time can be achieved compared to TURP, especially with larger prostates. However, the postoperative lengths of stay and complication rates are both substantially higher than for transurethral surgery. Following simple prostatectomy, blood transfusions were required in 8% of patients, and reoperation due to bleeding was required in 4% of cases (72). Blood loss has been shown to be significantly reduced with a laparoscopic or robotic-assisted approach compared to traditional open simple prostatectomy (73).

**Transurethral laser vaporization, ablation, and enucleation.** Improving technology has increased laser utilization in transurethral prostate surgery. Significant reduction in blood loss with laser vaporization and laser ablation of the prostate has allowed these procedures to be performed on an outpatient basis (74). Photoselective vaporization of the prostate (PVP) is performed with the potassium-titanyl-phosphate (KTP) laser, which has been demonstrated to provide equally effective reduction in AUA symptom score and improvement in urinary flow rate when compared to TURP (75). Transurethral ablation and enucleation of the prostate are typically performed with the holmium:yttrium aluminum garnet (Ho:YAG) laser. Holmium laser ablation of the prostate (HoLAP) is performed in a similar fashion to PVP, with similar postoperative expectations and patient management. Both HoLAP and PVP are more suitable for patients with a prostate size of <100 g, whereas holmium laser enucleation of the prostate (HoLEP) can be performed for patients with prostates of any size. The surgical technique of HoLEP is similar to simple prostatectomy, with removal of entire lobes of the prostate. In studies of patients with prostates larger than 100 g, HoLEP significantly reduced blood loss and hospital stay compared to simple prostatectomy, while providing equivalent reductions in AUA symptom score and improvement in urinary flow rates (76).

**Device therapy.** In attempts to reduce treatment-related morbidity, new devices for the treatment of BPH refractory to medical management continue to be introduced. Transurethral microwave thermotherapy (TUMT) is performed in an office setting with a transurethral probe that heats and causes coagulation necrosis of prostate tissue. Early studies demonstrated inferior outcomes with TUMT compared to TURP (77), but more recent studies suggest that both symptomatic and urinary flow rate improvement compare well to TURP (78). This may be attributable to improvements in device technology as well as increased experience with the procedure.

Transurethral needle ablation (TUNA) of the prostate is another office-based device that requires endoscopic visualization to confirm needle placement. TUNA has demonstrated efficacy

**Table 2 Surgical and device treatments for benign prostatic hyperplasia**

| Surgical treatments | Patient selection | Δ AUAss | Δ $Q_{max}$ (ml/s) | Complications | Durability |
|---|---|---|---|---|---|
| TURP (70)<br>TURP (75) | $V_{prostate}$ 20–30 g<br>AUAss ≥ 12<br>$Q_{max}$ < 15 ml/s<br>$V_{prostate}$ ≤ 100 g | 63–88% of patients with improvement<br>17.5 at 1 year | +8.2 to 15.1 at 1 year<br>+14.2 at 1 year | Blood transfusion (25%), retrograde ejaculation (73%)<br>Blood transfusion (10%), incontinence (3%) | 6% of patients required reoperation<br>2% of patients required repeat procedure to remove more tissue |
| TUIP (70) | $V_{prostate}$ 20–30 g | 63–85% of patients with improvement | +4.5 to 11.1 at 1 year | Blood transfusion (1%), retrograde ejaculation (21%) | 9% of patients required reoperation |
| Open simple prostatectomy (72) | Mean $V_{prostate}$ 96 g | Not reported | +13 ml/s | Blood transfusion (8%), mortality (0.2%) | Not applicable |
| Laparoscopic simple prostatectomy (73) | Mean $V_{prostate}$ 122 g | −16.7 | +16.5 ml/s | Blood transfusion (3%) | Not applicable |
| PVP (KTP laser) (75) | AUAss ≥ 12<br>$Q_{max}$ < 15 ml/s<br>$V_{prostate}$ ≤ 100 g | −14.2 at 1 year | +13.5 at 1 year | Blood transfusion (7%), incontinence (3%) | 3% of patients required repeat procedure to remove more tissue |
| HoLEP (76) | AUAss ≥12<br>$Q_{max}$ ≤12 ml/s<br>$V_{prostate}$ <100 g | −19.4 at 3 years | +16.0 at 3 years | Urethral stricture (4%), bladder neck contracture (3%) | 1% of patients with BPH recurrence |
| **Device therapies** | | | | | |
| TUMT (78) | AUAss ≥ 13<br>$Q_{max}$ < 13 ml/s<br>Mean $V_{prostate}$ 49 g | −13.6 at 5 years | +3.8 at 5 years | Hematuria (6%), urethral injury (3%), voiding symptoms (4%), incontinence (1%) | 10% required additional therapy<br>7% underwent TURP or TUIP within 5 years |
| TUNA (79) | AUAss ≥ 18<br>$Q_{max}$ 5–15 ml/s<br>$V_{prostate}$ < 90 g | −12.2 at 4 years | +3.5 at 4 years | Not reported | 23% required additional therapy<br>13% underwent TURP within 4 years |
| HIFU (80) | AUAss ≥ 18<br>$Q_{max}$ ≤ 15 ml/s<br>$V_{prostate}$ ≤ 75 g | −11.1 for the 46% of patients who responded to treatment | +2.7 ml/s | 54% of patients did not respond to treatment | 44% of patients underwent TURP within 4 years |
| Urethral stent (81) | Moderate to severe symptoms<br>25% with retention<br>72% with $V_{prostate}$ ≤40 g | 62% of patients with ≥50% improvement | +4.0 (for nonretention patients) | Stent explantation in 13% of patients | At 24 months: symptom improvement and Δ$Q_{max}$ sustained |
| Prostate lift (82) | AUAss ≥ 13<br>$Q_{max}$ ≤ 12 ml/s<br>$V_{prostate}$ 30–80 g | −11.1 at 1 year | +4.4 at 1 year | Serious adverse events in 11% of patients | At 12 months: AUAss improved with time and Δ$Q_{max}$ sustained |

Abbreviations: AUA, American Urological Association; AUAss, AUA symptom score; HIFU, high-intensity focused ultrasound; HoLEP, holmium laser enucleation of the prostate; KTP, potassium-titanyl-phosphate laser; PVP, photoselective vaporization of the prostate; $Q_{max}$, maximum urinary flow rate; TUIP, transurethral incision of prostate; TUMT, transurethral microwave thermotherapy; TUNA, transurethral needle ablation; TURP, transurethral resection of prostate; $V_{prostate}$, prostate volume.

for LUTS related to BPH; however, 13% of patients required BPH-related surgery within five years of TUNA (79).

Transrectal high-intensity focused ultrasound (HIFU) has also been investigated in the treatment of BPH. Compared to other device therapies, HIFU was effective in symptomatic improvement in relatively few patients receiving therapy (46%), and a large proportion (44%) of patients receiving HIFU underwent TURP within four years of treatment (80).

Endoscopic placement of a urethral prosthesis to stent open the prostatic urethra has also been investigated in treatment of BPH. Although symptomatic and flow rate improvements were noted with urethral stents, complications requiring stent explantation occur in roughly 13% of cases, due to device migration or recurrent infections from colonization and encrustation of the stent (81).

Most recently, a prostatic urethral lift device has been approved for treatment of BPH. The office-based procedure requires endoscopic guidance for placement of the small permanent implants that provide compression of the prostatic lobes against the capsule. The prostatic urethral lift has demonstrated significant reductions in AUA symptom score and improvements in flow rate (82). As the internal sphincter mechanism and ejaculatory ducts are unaffected, patients do not experience retrograde ejaculation. Long-term outcomes and comparison of outcomes to other interventions are not yet available.

**Table 2** summarizes the outcomes and complications associated with the various surgical and device treatments for BPH. To date, device therapies have not gained widespread use because treatment outcomes are generally inferior to those of surgical intervention, and improvements in technology (e.g., bipolar resection devices, KTP and holmium lasers) have limited the morbidity associated with surgery.

## DISCLOSURE STATEMENT

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