IJBPAS, February, 2026, 15(2): 665-679

ISSN: 2277–4998

International Journal of Biology, Pharmacy and Allied Sciences (IJBPAS)

'A Bridge Between Laboratory and Reader'

www.ijbpas.com

BENIGN PROSTATIC HYPERPLASIA: FROM ETIOLOGY TO TREATMENT – A PATHOPHYSIOLOGICAL PERSPECTIVE

HEMASRUTHI K, JASMITHA M, JYOTHIPRIYA N, KARUNAKARAN K AND YAIDIKAR L*

Seven Hills College of Pharmacy, Tirupati – 517561, Andhra Pradesh, India

***Corresponding Author: Dr. Lavanya Yaidikar: E Mail: drylavanya@gmail.com**

Received 15th Oct. 2024; Revised 5th Dec. 2024; Accepted 3rd Feb. 2025; Available online 1st Feb. 2026

https://doi.org/10.31032/IJBPAS/2026/15.2.9818

ABSTRACT

Benign Prostatic Hyperplasia (BPH) is one of the most prevalent medical conditions affecting the geriatric male population. The non-malignant enlargement of the prostate gland can lead to a range of clinical symptoms, including difficulty in voiding, urinary retention, and increased urinary frequency. The severity of symptoms often varies based on the degree of prostate enlargement. The International Prostate Symptom Score (IPSS) remains the gold standard for initial clinical assessment of BPH. However, recent advancements have introduced a variety of new diagnostic tools that enhance understanding and precision in diagnosing prostate hyperplasia. Over the past few years, BPH management has evolved significantly, with both medical and surgical interventions aimed at improving the patient's quality of life and reducing disease burden. Tailored pharmacological treatments, including alpha-blockers and 5-alpha-reductase inhibitors, along with minimally invasive surgical techniques, provide effective options to manage this condition, ultimately alleviating symptoms and improving long-term outcomes. This review highlights the importance of early diagnosis and personalized treatment approaches to effectively manage BPH and improve patient outcomes.

Keywords: Benign Prostatic Hyperplasia, Geriatric, International Prostatic Symptom Score

INTRODUCTION

Benign Prostatic Hyperplasia (BPH) is a common condition encountered in aging men and is a leading cause of lower urinary tract symptoms (LUTS). BPH refers to the nonmalignant growth of the prostate gland, primarily due to unregulated hyperplasia of epithelial and fibromuscular tissues in the transition zone (TZ) and periurethral area. This excessive tissue growth can lead to obstruction of the urethra, causing bothersome urinary symptoms. BPH is often referred to by other terms, including benign prostatic hypertrophy, an enlarged prostate, or simply BPH. As men age, the incidence of BPH increases, with nearly 50% of men over 50 and up to 90% of men over 80 being affected.

The pathophysiology of BPH involves hormonal changes, particularly the imbalance between testosterone and dihydrotestosterone (DHT), which stimulates the growth of prostate cells. The overproduction of DHT plays a critical role in the progression of BPH. Chronic inflammation, metabolic syndrome, and genetic predisposition have also been identified as contributing factors to the development of this condition.

Clinically, BPH is characterized by lower urinary tract symptoms such as urinary frequency, nocturia, weak urinary stream, and incomplete bladder emptying, which can significantly affect the quality of life. In some cases, untreated BPH can lead to complications such as acute urinary retention, recurrent urinary tract infections, bladder stones, and even renal insufficiency.

The diagnosis of BPH typically begins with the International Prostate Symptom Score (IPSS), a standardized tool to assess symptom severity. Additional diagnostic tests such as digital rectal examination (DRE), prostate-specific antigen (PSA) testing, and ultrasound may be utilized to evaluate the size of the prostate and rule out other conditions such as prostate cancer. In recent years, newer diagnostic modalities like MRI and advanced biomarkers have emerged, offering more precise and non-invasive ways to assess the condition.

The management of BPH has advanced significantly, ranging from conservative approaches, such as lifestyle changes and watchful waiting, to medical and surgical treatments. Pharmacological treatments, including alpha-blockers, 5-alpha-reductase inhibitors, and combination therapy, have become the cornerstone of medical management. These medications work by relaxing the smooth muscle of the

prostate or reducing its size. In more severe cases, surgical interventions like transurethral resection of the prostate (TURP), laser therapies, and minimally invasive techniques, such as prostatic artery embolization (PAE) or UroLift, provide effective options for relieving symptoms and improving urinary flow. As the population continues to age, BPH will remain a significant health issue. Early diagnosis and individualized treatment strategies are crucial in managing symptoms, preventing complications, and improving the overall quality of life for affected men **[1, 2]**.

ETIOLOGY

The etiology of Benign Prostatic Hyperplasia (BPH) is influenced by a wide range of risk factors, beyond the direct hormonal effects of testosterone on prostate tissue. It is well established that men who are castrated before puberty or those with androgen-related disorders do not develop BPH, highlighting the critical role of testicular androgens in the disease's development. Dihydrotestosterone (DHT), a potent androgen, plays a central role in promoting prostatic tissue growth and cellular proliferation by directly interacting with the prostatic epithelium and stromal cells.

Testosterone is converted into DHT by the enzyme 5-alpha-reductase type 2, which is highly active in the prostatic stromal cells. DHT accounts for approximately 90% of the total intraprostatic androgens, and it exerts its effects by influencing both stromal and adjacent epithelial cells. This process leads to an imbalance between cellular proliferation and apoptosis, resulting in prostatic enlargement. Despite the pivotal role of DHT in BPH, studies have shown no clear correlation between serum levels of testosterone or DHT and the development of symptomatic BPH. This suggests that local androgen metabolism within the prostate, rather than systemic hormone levels, may be more relevant in the progression of the disease. Other factors, including aging, inflammation, and genetic predisposition, may also contribute to the onset and severity of BPH, but hormonal regulation remains a key underlying mechanism **[2-4]**.

EPIDEMIOLOGY

The histological prevalence of BPH at autopsy is as high as 50% to 60% for males in their 60s, increasing to 80% to 90% of those older than 70 years of age. Age is a significant predictor of the development of BPH and subsequent LUTS. Fifty percent of men older than 50 years show evidence of BPH, and the association with the development of LUTS is shown to increase linearly with age. BPH increases each year

from 41% to 90%, and 50% of men between the ages of 51 and 60 years show the pathological features consistent with BPH. BPH occurs only in men; approximately 8 percent of men aged 31 to 40 have BPH. In men over age 80, more than 80 percent have BPH **[5]**.

The concept of obstruction by the enlarged prostate causing LUTS can be traced back to John hunter, one of the most influential British surgeons of the eighteenth century. In 1786, Hunter wrote. swelling of the prostate is the most common in the decline of life when diseased to alter its shape and size, it must obstruct the passage of urine". He continued to describe both the symptoms attributable directly to obstruction and those that urethral obstruction results in thickening of the wall of the bladder and irritability **[6]**.

ANATOMY OF PROSTATE

The prostate gland develops from the pelvic portion of the urogenital sinus, which is 10-12 weeks of the gestation. The prostate arises after the development of numerous endodermal buds, which initially proliferate throughout the entire length of the primitive urethra **[3]**. endodermal buds next invade the surrounding urogenital sinus mesenchyme, which is responsible for the development of the connective tissue and muscular constituents of the definitive prostate. The gland is well differentiated by the end of fourth month. Conversion to dihydrotestoterone is essential for the growth and development of the prostate. Prostate is supplied by prostatic branch of the inferior vesical artery, middle rectal artery and the internal pudendal arteries. Venous supply Venous drainage is by prostatic plexus of veins. Nerve supply Inferior hypogastric plexus conveys the sympathetic nerves; the preganglionic fibers are derived from the L1 and L2. The parasympathetic fibres derived from the pelvic splanchic nerves, the pregangionic fibers are from S2, S3 and S4 **[7]**.

PHYSIOLOGY

It is a male accessory sex gland that contributes considerable part of semen. Its secretion is milky and thin and contains acid phosphates ion, calcium citrate ion, fibrinolysin, prostaglandin and zinc. The growth and the function of the prostate is under the control of endocrine hormones i.e. androgens are essential for the prostate to achieve and maintain normal tissue mass, composition and secretary 6 function. The slightly alkaline prostatic fluid helps to neutralize the acidity of the other seminal fluids during the ejaculation and thus enhances the motility and fertilization of the

sperm with the ovum **[8]**.

PATHOPHYSIOLOGY

BPH arises due to the loss of homeostasis between prostatic cellular proliferation and apoptosis or cell death. This imbalance favors cellular proliferation without intervention. BPH, when prostatic urethra or bladder neck gets obstructed, more explicitly, microscopic BPH in the transitional zone of prostate gland may develop prostate enlargement or smooth muscle hyperplasia followed by bladder outlet obstruction **[7]**. Although BPH can increase prostate-specific antigen levels (PSA), it is not a risk factor for prostate cancer. BPH occurs primarily in the central/transitional portion of the prostate, while malignancies typically form in the prostatic periphery. Several pathophysiological (with emphasis on the androgen pathway) and the most relevant clinical aspects of BPH and BPE. The pathomechanisms leading to LUTS are much more complex than just BPH/BPE and involve several urodynamic patterns (e.g., detrusor overactivity/underactivity), changes within the urothelium and bladder ultrastructure, receptor status of the anticholinergic system, pelvic ischaemia and many more. LUTS appear which may be mild, moderate, or severe **[9]**.

HISTOPATHALOGY

Microscopic histological examination demonstrates that BPH is a hyperplastic process with increased cell numbers, including both glandular and stromal cellular proliferation. Hyperplasia occurs both in the periurethral and transition zones. Specifically, periurethral zones demonstrate hyperplastic stromal nodules, whereas glandular nodular proliferation is seen within the transition zone **[10]**.

SINGS AND SYMPTOMS

LUTS can be divided into storage (frequency, nocturia, urgency) and voiding disorders (weak or intermittent stream, straining to void [stranguria], hesitancy, prolonged micturition, incomplete emptying) and can help establish other causes of urinary problems such as urinary tract infections (UTIs), overactive bladder, or neurogenicity, in addition to determining the affected organ (bladder vs prostate) **[11]**.

When the prostate enlarges, it may constrict the flow of urine. Nerves within the prostate and bladder may also play a role in causing the following common symptoms Urinary frequency(Abnormally frequent urination), Urinary urgency(Urinary urgency is a sudden, uncontrollable need to pee), Nocturia(Needing to get up frequently at night to urinate),Hesitancy (Difficulty

initiating the urinary stream; interrupted, weak stream),Incomplete bladder emptying (The feeling of persistent residual urine, regardless of the frequency of urination), Straining (The need strain or push (Valsalva maneuver) to initiate and maintain urination in order to more fully empty the bladder), Decreased force of stream (The subjective loss of force of the urinary stream over time),Dribbling (The loss of small amounts of urine due to a poor urinary stream as well as weak urinary stream) **[12]**.

Men with BPH are likely to report symptoms of nocturia, poor stream, hesitancy, or prolonged micturition.Many men with BPH have no symptoms. In men with symptoms, the most common include needing to urinate frequently (during the day and night), a weak urine stream, and leaking or dribbling of urine. These symptoms are called lower urinary tract symptoms (LUTS) **[13]**.

PHYSICAL EXAMINATION

In the elective setting, the examination should include an abdominal examination (looking for palpable bladder/loin pain, lumps, hernias, or masses) and an examination of the external genitalia (meatal stenosis, testicular abnormalities, or phimosis). A neurological examination will help identify any neuropathy. The examination should include a digital rectal examination (DRE), making note of the particular size, shape, symmetry, nodularity, and consistency (smooth/hard) of the prostate. A smooth, enlarged prostate typically characterizes BPH **[14]**.

Further evaluation includes Urinalysis, Digital rectal examination, IPSS or American Urological Association symptom (AUA) symptom score, Postvoid residual volume (PVR to determine whether the bladder is emptying adequately), A frequency-volume chart or 24-hour voiding diary, Peak flow test, Laboratory evaluation for kidney function (BUN and creatinine) and diabetes (fasting glucose, Hgb A1c), PSA (Prostate-Specific Antigen) **[15]**.

Urinalysis

Urine specimen testing can help detect infection, microscopic hematuria, or metabolic disorders (glycosuria). Leukocytes and nitrites are common findings associated with infection, while the presence of proteinuria may suggest an underlying renal disorder. The AUA BPH Guidelines also recommend a urinalysis.

Blood Tests:

Blood tests, including BUN and creatinine, are useful to establish baseline renal function. They can help support the diagnosis of renal failure or acute kidney

injury in someone with chronic high-pressure or acute retention. A fasting glucose level or a Hgb A1c can identify diabetes.

24-Hour Voiding Diary:

A 24-hour urinary voiding diary, where the patient measures and records the time and volume voided for a complete day, can be extremely helpful in evaluating urinary disorders, particularly nocturia. High urinary volumes at night are more consistent with nocturnal polyuria, while small volumes suggest bladder overactivity. (See our companion StatPearls reference article on "Nocturia.")

Prostate-Specific Antigen (PSA):

Prostate-specific antigen testing is somewhat predictive of prostate volume. Benign prostates of 35 cc size will typically generate a PSA of 1.5 ng/mL. PSA testing is recommended where cancer is suspected (hard prostatic nodule, asymmetry, metastatic disease suspected) or a previous baseline PSA has been previously established. PSA testing is not a routine test done for BPH but is recommended before starting five alpha-reductase therapy or performing surgery to avoid missing a possible prostatic malignancy. (See our companion StatPearls reference articles on "Prostate Specific Antigen" and "Prostate Cancer Screening.")

Postvoid Residual Volume:

A postvoid residual urine volume is measured to determine how well the bladder empties after urination. This can be done with a bladder scan, formal bladder ultrasound, or a quick straight catheterization. The easiest way is to do a bladder scan if that is available. The normal PVR would be <100 to 150 mL, while >200 mL would be considered pathological. The measurement should be performed immediately after the patient voids but is still useful if done within 15 to 20 minutes. The postvoid residual measurement is an important and valuable determinant in evaluating and assessing BPH. It is also recommended by the AUA 2021 Guidelines on the Management of BPH before surgical intervention.

While urology offices and hospitals typically have dedicated bladder scanners, most primary care facilities do not. Many experts recommend bladder scanners for primary care offices to measure PVR and diagnose urinary retention in patients with suprapubic pain, incontinence, urinary difficulty, or symptoms of BPH. A bladder scan model specifically designed for primary care offices can also screen patients for abdominal aortic aneurysms. This makes it very cost-effective even for small primary care offices

Urinary Flow Studies (Flowmetry):

Urine flow studies (flowmetry) determine peak urinary flow rates. This can help establish whether there is objective evidence for urinary obstruction. The peak flow rate is the most significant measurement. A peak flow ≥ 13 cc/sec is considered acceptable. A flow test requires a volume of at least 150 cc to be considered valid. (If the peak flow ≥13 cc/sec, then the volume does not matter).

Optimally, the postvoid residual measurement is made immediately after the flowmetry study. Decreased peak flow unrelated to inadequate volume is typically due to either obstruction (BPH, detrusor sphincter dyssynergia, urethral stricture, or bladder neck contracture) or detrusor hypotonicity. Flowmetry is recommended by the AUA 2021 Guidelines on Management of BPH before surgical intervention.

Pressure/Flow Studies:

A pressure/flow study is recommended in cases of abnormal urination where the diagnosis is uncertain or the benefit of surgical intervention is unclear. Noninvasive studies such as PVR measurements and flowmetry are certainly sufficient in most cases, but only a pressure/flow study can reliably determine the adequacy of detrusor muscular contractility and the presence of bladder outlet obstruction. Most men with obstructive BPH will have a low urinary peak flow of <10 cc/sec with a normal or high detrusor voiding pressure. Pressure/flow studies are recommended by the AUA 2021 Guidelines on Management of BPH before surgical intervention.

Urodynamics:

Urodynamic studies are used to see how the bladder empties and fills. They include pressure/flow studies but also evaluate sphincteric function and possible neurogenicity. Urodynamics can help further assess patients where the diagnosis is not certain or where a neurogenic/overactive bladder is suspected (ie, neurological conditions that may affect the bladder, detrusor sphincter dyssynergia, equivocal flow studies, unclear diagnoses, spinal cord injuries, sacral disorders, etc). Urodynamics are not required in most patients with BPH and are only indicated where there is doubt about urinary function and obstruction, even after simpler studies.

Renal Ultrasound:

Renal ultrasound scans are used to look for evidence of hydronephrosis. They are only indicated in patients with high residual volumes, acute or chronic urinary retention, or unexplained renal impairment.

Other indications include suspicion of urinary tract stones or to investigate unexplained hematuria. If nephrolithiasis or bladder calculi are suspected, a KUB x-ray or a noncontrast CT of the abdomen and pelvis may be used (see Image. CT of Pelvis Showing Multiple Bladder Stones).

Cystoscopy:

Flexible cystoscopy should be used to investigate red-flag symptoms such as unexplained hematuria, possible bladder calculi, or suspected bladder cancer. It can also identify urethral strictures, evaluate median lobe enlargement, detect intravesical median lobe extensions and lobulations, determine the degree of obstruction in the prostatic urethra, and evaluate the bladder for stones and signs of damage. Cystoscopy provides a good estimate of prostate size and shape, can evaluate the degree of obstruction, and permits visualizing signs of bladder damage. It is strongly recommended before BPH surgical intervention. The bladder can be filled during the cystoscopy, allowing a urinary flow study and determining PVR **[15][16]**.

Before surgical intervention for BPH, measurement of prostatic size is recommended, as many procedures have prostatic volume or shape limitations. While this is typically done by cystoscopy, it may also be accomplished by abdominal or transrectal ultrasonography, CT scan, or MRI **[16]**.

DIAGNOSIS:

BPH has been diagnosed by the following features:

(A) Moderate to severe LUTS with International Prostate Symptom Score (IPSS) greater than 8,

(B) an enlarged prostate (total prostatic volume >30 mL), and

(C) maximum urinary flow rate less than 15 mL

American Urological Association Symptom Score,International Prostate Symptom Score (IPSS)

Both the AUA symptom score and IPSS have been verified and validated. They are used to identify significant LUTS, determine their type (obstructive or irritative), and assess their severity. They are useful when quantifying symptom severity, tracking symptom relief with therapy, and categorizing patients for treatment. The IPSS and AUA symptom scores categorize patients into 3 groups based on symptoms. The groups are mild (scores 0 to 9), moderate (scores 10 to 19), and severe (scores 20 to 35). A symptom score ≥ 10 suggests that BPH **[17]**.

RISK FACTORS:

Non-modifiable and modifiable risk factors also contribute to the development of BPH. These have been shown to include diabetes, diet, genetic factors, localized inflammation, obesity, and metabolic syndrome **[18]**.

TREATMENT:

Because BPH rarely causes serious complications, men usually have a choice between treating it than opting for watchful waiting Treatment Options: The primary goals of treatment for BPH are to improve urinary flow and to reduce symptoms. Many options are available. They include drug therapies, minimally invasive procedures, and major surgery **[19]**.

The main treatments are: Lifestyle changes, Medicine, Surgery and other procedures

LIFE STYLE CHANGES:

Fizzy drinks and drinks that contain alcohol, caffeine (such as tea, coffee or cola) and artificial sweeteners can irritate the bladder and make urinary symptoms worse. Drinking less fluid in the evening. Go to the toilet before long journeys or when you know you will not be able to reach a toilet easily. Double voiding involves waiting a few moments after you have finished peeing before trying to go again. It can help you empty your bladder properly. But take care not to strain or push. Eating more fibre (which is found in fruit, vegetables and wholegrain cereals) can help you avoid constipation, which can put pressure on your bladder and make the symptoms of an enlarged prostate worse **[19][20]**.

MEDICAL MANAGEMANT:

Nonselective Alpha-1 Blockers:

Doxazosin, prazosin, and terazosin reduce prostatic smooth muscle tone and, thus, have an immediate effect on urinary flow. Although these medications quickly improve BPH symptoms, International prostate symptom scores improve less than with surgery. Side effects occur such as dizziness, postural hypotension, fatigue, asthenia and retrograde ejaculation. Side effects can be minimized by bedtime administration and slow titration of the dosage. Alpha blockers can be used with other therapies as needed. Prazosin has the cost advantage of generic availability **[21]**.

Selective Alpha-1 Blocker

Tamsulosin is a highly selective alpha-1 adrenergic antagonist that was developed to avoid the side effects of non selective agents. Some patients who do not respond to non selective alpha blockers may respond to tamsulosin and, because of the selectivity, may have fewer side effects, including hypotension. Tamsulosin is initiated in a dosage of 0.4 mg once daily,

with a maximum dosage of 0.8 mg per day. Tamsulosin has no antihypertensive effect and is more expensive than non selective alpha blockers **[22]**.

5-Alpha Reductase Inhibitors

Finasteride slowly induces a 50 percent reduction in the serum dihydrotestosterone level. As a result, prostatic volume decreases by about 19 percent over three to six months of treatment. The treatment with finasteride led to significant improvements in urinary symptoms and flow rates. However, in the Prospect study, the improvements with finasteride were significantly less than those with any alpha blocker or surgery. Studies suggest that finasteride may work best in men with a large gland, whereas alpha blockers are effective across the range of prostate sizes. The incidence of side effects with finasteride is similar to that with placebo (4 to 5 percent). Adverse effects include decreased libido, ejaculatory disorder, and impotence. Finasteride decreases PSA levels by 40 to 50 percent. In a patient taking finasteride who has PSA screening, PSA levels should be doubled and then compared in the usual fashion to age-related norms. There is no change in sensitivity or specificity for the diagnosis of prostate cancer.

Antimuscarinics

Antimuscarinics like oxybutynin, solifenacin, tolterodine are commonly used for urinary frequency, urgency, and bladder overactivity symptoms. They are also useful for the symptomatic management of detrusor instability due to bladder outlet obstruction from BPH, which can result in increased urgency (overactive bladder) and frequency. Muscarinic receptor antagonists can help with these symptoms by blocking muscarinic receptors in the detrusor muscle. This reduces smooth muscle tone and can improve irritative symptoms in those with bladder overactivity. Examples include solifenacin, tolterodine, trospium, and oxybutynin.

Those who fail antimuscarinic treatment may be considered for mirabegron or vibegron use (beta-3 adrenoreceptor agonists), which also cause detrusor relaxation and relieve symptoms of overactivity with no cholinergic or mental side effects **[23]**.

Surgical management:

Surgical management of BPH has broadened significantly over the years, with the development of further minimally invasive techniques. Current recommended procedures include TURP and newer techniques, such as laser vaporization and holmium laser enucleation, which have largely replaced open prostatectomy.

Minimally invasive surgical options such as paclitaxel-coated prostatic balloon dilation, prostatic urethral internal lateral suturing (prostatic urethral lift), transurethral microwave thermotherapy, and water vapor or steam infusion therapy are also available and FDA-approved. In rare cases, prostatic artery embolization can be considered **[24][25]**.

CONCLUSION:

Even though benign prostatic hyperplasia is one of the most common disorders amongst the geriatric population, the surgical treatment has grown tremendously compared to the medical treatment. The medical treatment provides only the symptomatic relief rather than curing the disease itself. Since most of the geriatric would be having an allied comorbidities like diabetes, hypertension which would not allow everyone to undergo the surgical procedures and hence medical treatment becomes accessible and hence lot of research needs to be focused in this arena **[26-29]**.

REFERENCES:

[1] Park Ch, Chang HS, Oh BR, Hung KM, Sul CK, Chung SK, et al. Efficacy of low dose tamsulosin on lower urinary tract symptoms of benign prostatic hyperplasia. J Clin Drug Invest 2004; 24 Supp1: 41-47. http://dx.doi.org/10.2165/00044011-200424010-00005

[2] Marberger M, Horkaway R, Rosette JD. Optimising the medical management of benign prostatic hyperplasia. J Eururol 2004; 45: 411-19.

[3] Tanaka Y, Naoya M, Itoh N, Sato Y, Takahashi A, Ogura H, Furuya S et al. Urodynamic effects of terazosin treatment for Japanese patients with symptomatic benign prostatic hyperplasia. J Urol 2002; 167: 2492-95. http://dx.doi.org/10.1097/00005392-200206000-00033

[4] Hofner K, Jones U. Alfuzosin a clinically uroselective alpha-1A blocker. World Jurol 2002; 19: 405-12. PMid:12022709 5. Chapter in a book: Wein AJ, Kavoussi LR, Novick AC, Partin AC. In: Gartner FG editor. Campbells-Walsh urology. 9th ed: Saunders Elsevier; 2007. p. 465-78.

[5] Roehrborn CG. Benign prostatic hyperplasia: an overview. Rev Urol. 2005;7 Suppl 9(Suppl 9):S3-S14. [PMC free article] [PubMed]

[6] Abrams P. LUTS, BPH, BPE, BPO: A Plea for the Logical Use of Correct

Terms. Rev Urol. 1999 Spring;1(2):65. [PMC free article] [PubMed]

[7] Silverman WM. "Alphabet soup" and the prostate: LUTS, BPH, BPE, and BOO. J Am Osteopath Assoc. 2004 Feb;104(2 Suppl 2):S1-4. [PubMed]

[8] Abrams P. New words for old: lower urinary tract symptoms for "prostatism". BMJ. 1994 Apr 09;308(6934):929-30. [PMC free article] [PubMed]

[9] Roehrborn CG. Pathology of benign prostatic hyperplasia. Int J Impot Res. 2008 Dec;20 Suppl 3:S11-8. [PubMed]

[10] Parsons JK. Benign Prostatic Hyperplasia and Male Lower Urinary Tract Symptoms: Epidemiology and Risk Factors. Curr Bladder Dysfunct Rep. 2010 Dec;5(4):212-218. [PMC free article] [PubMed]

[11] Chughtai B, Forde JC, Thomas DD, Laor L, Hossack T, Woo HH, Te AE, Kaplan SA. Benign prostatic hyperplasia. Nat Rev Dis Primers. 2016 May 05;2:16031. [PubMed]

[12] Chapter in a book: Glass J, Mundy A. The perineum and pelvis. In: Susan S, Harold E, Jeremiah CH, David J, Andrew W, Collins P et al editor. Grey's anatomy. The anatomical basis ofclinical practice. 13th ed. Elsevier Churchill livingstone; 2005. p. 893-41.

[13] Chapter in a book: Moore KL. The perineum and pelvis. In Gardner N editor. Clinically oriented anatomy. 1 st ed. Williams and Wilkins; 1985. p. 298-392.

[14] Chapter in a book: Moore KL, Arthur FD. Pelvis and perineum. In: Moore KL, Arthur FD editor. Clinically oriented anatomy. 4 thed. Lippincott Williams and Wilkins; 1999. p. 362-467.

[15] Chapter in a book: Dutta AK. Male genital system.In: Dutta AK editor. Essentials of human anatomy; thorax and abdomen. 4 th ed. Current books international; 1997. p. 555-76.

[16] Chapter in a book: Guyton AC, Hall JE. Reproduction and hormonal functions of the male (and function of the pineal gland). In: Guyton AC, Hall JE editor. Textbook of medical physiology. 11th ed. Elsevier; 2000. p. 589-15.

[17] Keeth DW, Andriole GL. Medical therapy for benign proststic

hyperplasia. J Advances in clinical medicine 2002; 164: 11-15.

[18] Chapter in a book: Harsh Mohan. The male reproductive system and prostate. In: Harsh Mohan editor. Text book of pathology. 5 th ed. Jaypee brothers; 2005. p. 728-47.

[19] Damjanov I, Linder J. Male reproductive system. In Damjanov I, Linder J. Andersons pathology. Tenth edition. Mosby-year book; 1996. p. 2166- 31. 26. Chapple CR. Pharmacology therapy of benign prostatic hyperplasia / lower urinary symptoms an overview for practicing clinician. BJU 2004; 94: 738-44. http://dx.doi.org/10.1111/j.1464-410X.2004.05022.x PMid:15329091

[20] Mostafa MM, Patil N, Khalil M, Elgammal MA, Mahdy A. Is Holmium Laser Enucleation of Prostate equally effective in management of benign prostatic hyperplasia patients with either voiding or storage lower urinary tract symptoms? A comparative study. Arch Ital Urol Androl. 2022 Jun 29;94(2):174-179. [PubMed]

[21] Lin L, Teng J, Huang CJ, Zhang X, Xu D. Holmium laser enucleation of the prostate versus transurethral resection of the prostate: a systematic review and meta-analysis of randomized controlled trials. J Endourol. 2013 May;27(5):604-11. [PubMed]

[22] Awosika AO, Farrar MC, Jacobs TF. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Nov 18, 2023. Paclitaxel. [PubMed]

[23] Abra L, Zain MA, Siddiqui WJ. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Aug 7, 2023. Angioplasty. [PubMed]

[24] Weaver BA. How Taxol/paclitaxel kills cancer cells. Mol Biol Cell. 2014 Sep 15;25(18):2677-81. [PMC free article] [PubMed]

[25] Rten M, Torsello GB, Schönefeld E, Stahlhoff S. Critical appraisal of paclitaxel balloon angioplasty for femoral-popliteal arterial disease. Vasc Health Risk Manag. 2016;12:341-56. [PMC free article] [PubMed]

[26] Habra L, Zain MA, Siddiqui WJ. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Aug 7, 2023. Coronary Stents. [PubMed.

[27] Majeed H, Chowdhury YS. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Sep 14, 2022. Percutaneous Transluminal Angioplasty and Balloon Catheters. [PubMed]

[28] Valdes PJ, Akbar H, Kahloon RA, Diaz MA. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Jun 26, 2023. Intracoronary Stents. [PubMed]

[29] Kaplan SA, Pichardo M, Rijo E, Espino G, Lay RR, Estrella R. One-year outcomes after treatment with a drug-coated balloon catheter system for lower urinary tract symptoms related to benign prostatic hyperplasia. Prostate Cancer Prostatic Dis. 2021 Dec;24(4):1073-1079. [PMC free article] [PubMed]