

Benign prostatic hyperplasia (BPH), also called benign enlargement of the prostate (BEP or BPE), is a non-cancerous increase in size of the prostate. It involves hyperplasia of prostatic stromal and epithelial cells (*Cunningham et al., 2013*).

BPH is a common problem among the aging men. It is reported that BPH occurs in 15% to 60% of men aged more than 40 years, and its prevalence increases markedly with age (*Parsons et al., 2008*).

BPH may contribute to an array of urinary voiding difficulties that can range from bothersome to significantly impacting quality of life among older men (*Roehrborn et al., 2011*). Treatment choices for BPH causing bladder outlet obstruction have been increasing. There are numerous options including watchful waiting, pharmacological therapy, minimally invasive therapy, transurethral resection (TURP), and open prostatectomy (*Olesovsky C and kapoor A. , 2016*). The conventional standard monopolar TURP is still considered the gold standard surgical procedure for lower urinary tract symptoms (LUTS) caused by BPH (*El-Tabey et al., 2015*). The complications rate associated with M- TURP is ranged between 7% to 43% which are mainly bleeding, transurethral-resection syndrome, urinary incontinence, retrograde ejaculation, infection and erectile dysfunction Moreover, the mortality rate associated with M-TURP is 0.2 % (*Tawfiek, 2009*).

Technical modification of TURP with incorporation of bipolar technology (B - TURP) has two advantages: first, patients can better tolerate sodium chloride solution and thus eliminate the risk of TUR syndrome. Second, the high - frequency current used minimizes tissue denaturation. The resectoscope and electrode act as the neutral electrode that completes the circuit without need to the patient's plate (*Abou-Taleb et al., 2016*).

Recently, transurethral enucleation of the prostate (TUEP) has been an alternative approach for treating BPE, which can remove prostatic tissue anatomically similar to transvesical prostatectomy, but in a minimally invasive endoscopic manner. Many kinds of device have been adopted in TUEP. Holmium laser, thulium laser, and diode laser have been frequently used in TUEP based on their properties of high vaporization and excellent coagulation, but their application is limited because of expensive treatment cost, long enucleating time, and long learning curve. Plasmakinetic device with loop electrode has also been performed in TUEP. It is as efficacious as laser, is inferior to laser in coagulation and safety, whose application is restricted in patients with a large prostate (*Zhang et al., 2015*).

To compare between transurethral enucleation of the prostate using plasma vaporization and bipolar transurethral resection regarding to efficacy, operative time, post-operative bleeding, hospital stay, catheterization duration and early complications.

The normal prostate weighs 18-22 grams; measures 3 cm in length, 4 cm in width, and 2 cm in depth and is traversed by the prostatic urethra. Although ovoid, the prostate is referred to as having anterior, posterior, and lateral surfaces, with a narrow apex inferiorly and a broad base superiorly that is continuous with the base of the bladder (*James, 2007*).

Anatomic relationships:

Posterolaterally:

Microscopic bands of smooth muscle extend from the posterior surface of the capsule to fuse with Denonvillier's fascia. Loose areolar tissue defines a thin plane between Denonvillier's fascia and the rectum (*James, 2007*).

Anteriorly and anterolaterally:

The capsule blends with the visceral continuation of endopelvic fascia. Towards the apex, the puboprostatic ligaments extend anteriorly to fix the prostate to the pubic bone. The superficial branch of the dorsal vein lies outside this fascia in the retropubic fat and pierces it to drain into the dorsal vein complex (*James, 2007*).

The deep dorsal vein is an unpaired vein in the dorsal midline of the penis. It drains into the periprostatic plexus and then into the internal pudendal veins.

Laterally:

The prostate is cradled by the pubococcygeal portion of levator ani and is directly related to its overlying endopelvic fascia (*Myers, 1994*).

Superiorly:

The prostate is continuous with the neck of the bladder. The urethra enters the upper aspect of the prostate near its anterior border (*Harold, 2006*).

Inferiorly:

The apex of the prostate is continuous with the striated urethral sphincter. Histologically, normal prostatic glands can be found to extend into the striated muscle with no intervening fibromuscular stroma or capsule (*Epstein, 1989*).

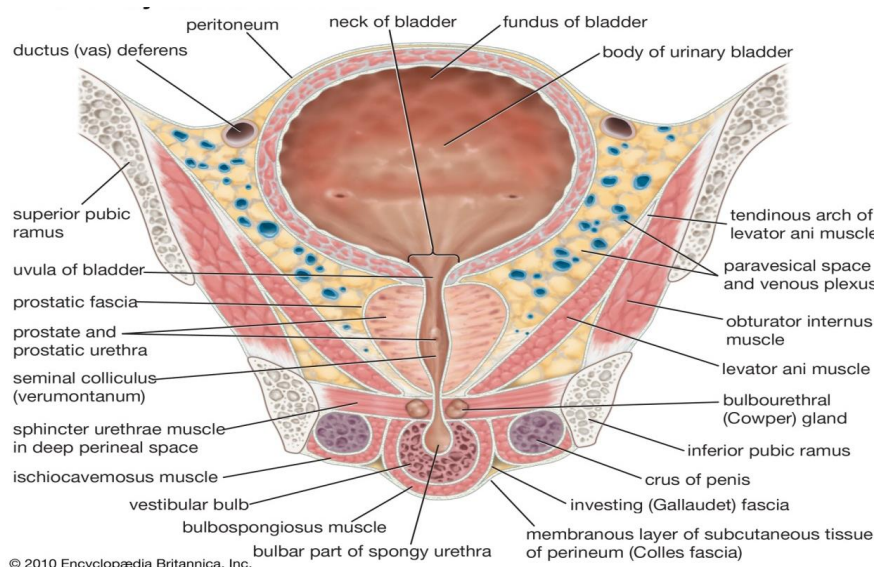


Figure (1): Anatomic relationships of the prostate (*Encyclopædia Britannica, inc. 2015*).

Structure:

The prostate is composed of approximately 70% glandular elements and 30% fibromuscular stroma. The stroma is continuous with the capsule and is composed of collagen and abundant smooth muscle. It encircles and invests the glands of the prostate and contracts during ejaculation to express prostatic secretions into the urethra (*James, 2007*).

Zonal anatomy of the prostate:

The glandular elements of the prostate have been divided into discrete zones, distinguished by the location of their ducts in the urethra, by their differing pathologic lesions, and, in some cases, by their embryologic origin.

1- The transition zone:

At the angle dividing the preprostatic and prostatic urethra, the ducts of the transition zone arise and pass beneath the preprostatic sphincter to travel on its lateral and posterior sides. Normally, the transition zone accounts for 5% to 10% of the glandular tissue of the prostate. A discrete fibro muscular band of tissue separates the transition zone from the remaining glandular compartments and may be visualized at Transrectal ultrasonography of the prostate. The transition zone commonly gives rise to benign prostatic hyperplasia, which expands to compress the fibro muscular band into a surgical capsule seen at enucleation of an adenoma. It is estimated that 20% of

adenocarcinomas of the prostate originate in this zone (*James, 2007*).

•- *The central zone*

The ducts of the central zone arise circumferentially around the openings of the ejaculatory ducts. This zone constitutes 25% of the glandular tissue of the prostate and expands in a cone shape around the ejaculatory ducts to the base of the bladder. In keeping with this suggestion, only 1% to 5% of adenocarcinomas arise in the central zone, although it may be infiltrated by cancers from adjacent zones (*McNeal, 1988*).

•- *The peripheral zone:*

The peripheral zone makes up the bulk of the prostatic glandular tissue (70%) and covers the posterior and lateral aspects of the gland. Its ducts drain into the prostatic sinus along the entire length of the (postsphincteric) prostatic urethra. Seventy percent of prostatic cancers arise in this zone, and it is the zone most commonly affected by chronic prostatitis (*McNeal, 1988*).

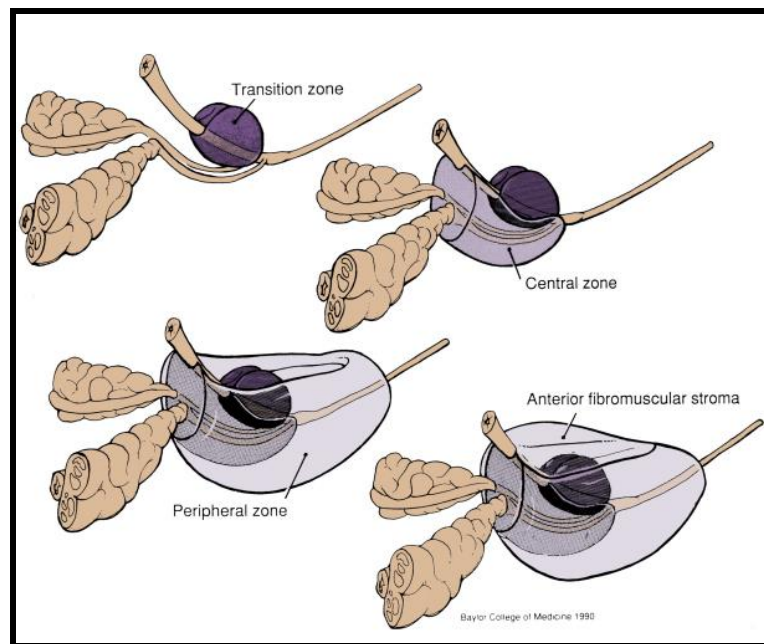


Figure (2): Zonal anatomy of the prostate (*McNeal, 1988*).

Lobes of the prostate:

Clinically, the prostate is often spoken of as having two lateral lobes, separated by a central sulcus that is palpable on rectal examination, and a middle lobe, which may project into the bladder in older men. These lobes do not correspond to histologically defined structures in the normal prostate but are usually related to pathologic enlargement of the transition zone laterally and the periurethral glands centrally (*James, 2007*).

The prostatic capsules:

These are normally two, pathologically three, in number:

- 1- **The true capsule:** a thin fibrous sheath that surrounds the gland.

- ↯- **The false capsule:** condensed extra peritoneal fascia which continues into the fascia surrounding the bladder and with the fascia of Denonvilliers posteriorly. Between layers 1 and 2 lies the prostatic venous plexus.
- ↯- **The pathological capsule:** when benign adenomatous hyperplasia of the prostate takes place, the normal peripheral part of the gland becomes compressed into a capsule around this enlarging prostatic tissue (*Harold, 2006*).

Histology of the prostate:

In general, the glands of the prostate are tubuloalveolar with relatively simple branching and are lined with simple cuboidal or columnar epithelium. Scattered neuroendocrine cells, of unknown function, are found between the secretory cells. Beneath the epithelial cells, flattened basal cells line each acinus and are believed to be stem cells for the secretory epithelium. Each acinus is surrounded by a thin layer of stromal smooth muscle and connective tissue (*James, 2007*).

Vascular supply:

Most commonly, the arterial supply to the prostate arises from the inferior vesical artery. As it approaches the gland, the artery (often several) divides into two main branches. The urethral arteries penetrate the prostatovesical junction posterolaterally and travel inward, perpendicular to the urethra. They approach the bladder neck in the 1- to 5-o'clock and 7- to

11-o'clock positions, with the largest branches located posteriorly. They then turn caudally, parallel to the urethra, to supply it, the periurethral glands, and the transition zone. Thus, in benign prostatic hyperplasia, these arteries provide the principal blood supply of the adenoma. When these glands are resected or enucleated, the most significant bleeding is commonly encountered at the bladder neck, particularly at the 4- and 8-o'clock positions. The capsular artery is the second main branch of the prostatic artery. This artery gives off a few small branches that pass anteriorly to ramify on the prostatic capsule. The bulk of this artery runs posterolateral to the prostate with the cavernous nerves (neurovascular bundles) and ends at the pelvic diaphragm. The capsular branches pierce the prostate at right angles and follow the reticular bands of stroma to supply the glandular tissue (*Flocks, 1937*).

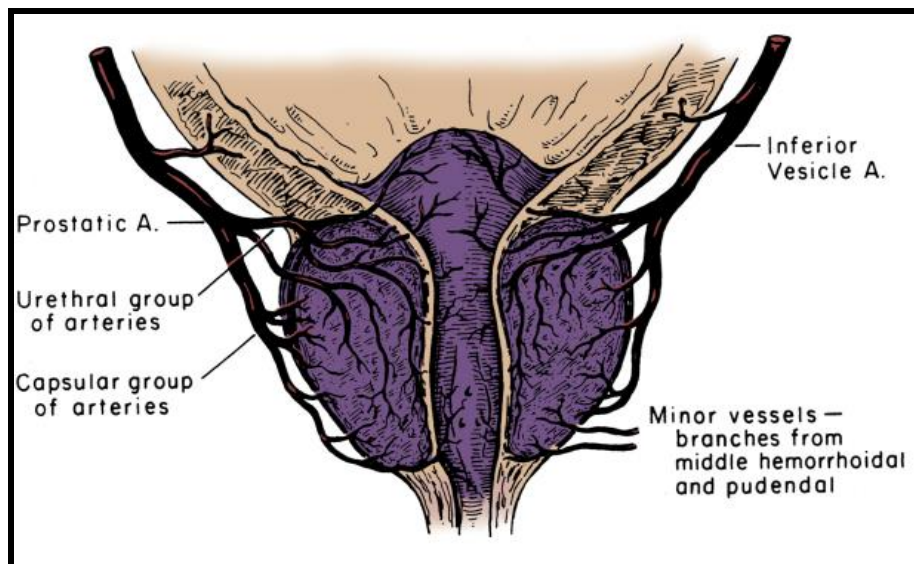


Figure (3): Arterial supply of the prostate (*Flocks, 1937*)

Venous drainage:

Venous drainage of the prostate is abundant through the periprostatic plexus (*Burnett, 1995*).

Lymphatic drainage:

Lymphatic drainage is primarily to the obturator and internal iliac nodes. A small portion of drainage may initially pass through the presacral group, or less commonly, the external iliac nodes (*Burnett, 1995*).

Nerve supply:

Sympathetic and parasympathetic innervation from the pelvic plexus travels to the prostate through the cavernous nerves. Nerves follow branches of the capsular artery to ramify in the glandular and stromal elements. Parasympathetic nerves end at the acini and promote secretion; sympathetic fibers cause contraction of the smooth muscle of the capsule and stroma. α -Adrenergic blockade diminishes prostate stromal and preprostatic sphincter tone and improves urinary flow rates in men affected with benign prostatic hypertrophy; this emphasizes that this disease affects both the stroma and the epithelium. Peptidergic and nitric oxide synthase-containing neurons also have been found in the prostate and may affect smooth muscle relaxation (*Burnett, 1995*).

The term *benign prostatic hyperplasia* (BPH) describes a proliferative process of the cellular elements of the prostate, an enlarged prostate, or the voiding dysfunction resulting from prostatic enlargement and bladder outlet obstruction. Histologically, BPH describes a proliferative process of both the stromal and epithelial elements of the prostate gland. BPH arises in the periurethral and transition zones of the prostate (*Herbert, 2004*).

The enlarged gland has been proposed to contribute to the overall LUTS complex via direct bladder outlet obstruction from enlarged tissue (static component), and from increased smooth muscle tone and resistance within the enlarged gland (dynamic component). Voiding symptoms have often been attributed to the physical presence of BOO. Detrusor over activity is thought to be a contributor to the storage symptoms seen in LUTS (*Reynard, 2004*).

Hyperplasia:

In a given organ, the number of cells, and thus the volume of the organ, is dependent upon the equilibrium between cell proliferation and cell death. An organ can enlarge not only by an increase in cell proliferation but also by a decrease in cell death (*Claus, 2016*).

Although androgens and growth factors stimulate cell proliferation in experimental models, the relative role of cell

proliferation in human BPH is questioned because there is no clear evidence of an active proliferative process. Although it is possible that the early phases of BPH are associated with a rapid proliferation of cells (*Kyprianou et al., 1996*).

The Role of Androgens:

Although androgens do not cause BPH, the development of BPH requires the presence of testicular androgens during prostate development, puberty, and aging. Patients castrated prior to puberty or who are affected by a variety of genetic diseases that impair androgen action or production does not develop BPH. It is also known that prostatic levels of dihydrotestosterone (DHT) as well as the androgen receptor (AR) remain high with aging despite the fact that peripheral levels of testosterone are decreasing. Moreover, androgen withdrawal leads to partial involution of established BPH (*Roehrborn, 2008*).

Assuming normal ranges, there is no clear relationship between the concentration of circulating androgens and prostate size in aging men (*Roberts et al., 2004*).

Despite the importance of androgens in normal prostatic development and secretory physiology, there is no evidence that either testosterone or DHT serves as the direct mitogen for growth of the prostate in older men. However, many growth factors and their receptors are regulated by androgens. Thus, the action of testosterone and DHT in the prostate is mediated

indirectly through autocrine and paracrine pathways (*Claus, 2016*).

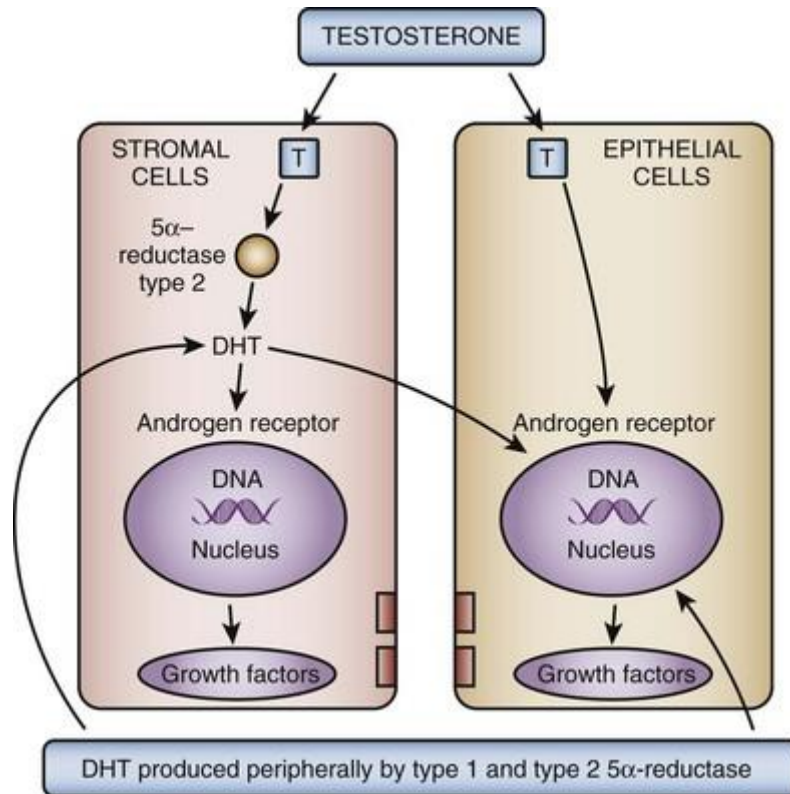


Figure (4): Testosterone as a stimulant mitogen in BPH proliferation (*Claus and Roehrborn, 2016*).

Dihydrotestosterone and Steroid 5 α -Reductase:

Intraprostatic DHT concentrations are maintained but not elevated in BPH. Initial studies of resected prostatic tissue suggested that prostatic DHT levels were higher in the hyperplastic gland than in normal control tissues (*AUA Guideline management of BPH, 2010*).

Two steroid 5 α -reductase enzymes have been discovered, each encoded by a separate gene. Type 1 5 α -reductase, the predominant enzyme in extraprostatic tissues, such as skin and liver. Type 2 5 α -reductase is the predominant in prostatic tissue and is critical to normal development of the prostate and hyperplastic growth later in life (*Claus and Roehrborn, 2016*).

These data demonstrate that the stromal cell plays a central role in androgen-dependent prostatic growth and that the type 2 5 α -reductase enzyme within the stromal cell is the key androgenic amplification step (*McConnell, 1995*).

The Role of Estrogens:

There are at least two forms of the estrogen receptor. Estrogen receptor α is expressed by prostate stromal cells, and estrogen receptor β is expressed by prostate epithelial cells (*Prins et al., 2001*).

Estrogens, mainly 17- β -estradiol also elicit effects on the prostate, mediated mainly through intraprostatic estrogen receptors— α and β (ER- α , ER- β) (*Kuiper et al., 1996*). Estrogens are also thought to play a role in the etiology of BPH, perhaps by rendering prostate cells more susceptible to the action of testosterone and DHT (*Wong YC and Wang Yz, 2000*). Despite this evidence for androgenic and estrogenic involvements in BPH development, some researchers are of the opinion that since not all patients benefit from hormonal