### Introduction

Benign prostatic hyperplasia (BPH) is defined as the proliferation of prostatic stromal cells, which results in an enlarged prostate gland. As a result the prostatic urethra is compressed, which restricts the flow of urine from the bladder. This interference with urine flow may cause uncomfortable symptoms such as frequency, urgency, nocturia, intermittency, decreased stream, and hesitancy (*Kapoor*, 2012).

Benign prostatic hyperplasia, a histologic diagnosis, is a condition that occurs with aging; the prevalence increases from 25% among men 40 to 49 years of age to more than 80% among men 70 to 79 years of age (*Sarma and Wei, 2012*).

Many complications may be developed from BPH such as increased post-voiding residual, bladder diverticula or calculi, vesico-ureteral reflux, hydronephrosis, renal insufficiency, and urine retention (*Oelke et al.*, 2012).

Acute urine retention (AUR) is a common urological emergency that is characterized by sudden and painful inability to pass urine. The incidence of AUR in patients with BPH varies widely from 0.4% to 25 % (*Kara and Yazici*, 2014).

Management of AUR consists of immediate bladder decompression by catheterization usually followed by BPH-related surgery. The evidence that emergency surgery was associated with an increased mortality and a higher rate of

postoperative complications, and the potential morbidity associated with prolonged catheterization have led to an increased use of a trial without catheter (TWOC) (*McNeill*, 2006).

Trial without catheter (TWOC) is preferable compared with leaving the catheter in place, and a 23-28% success rate has been reported. Nevertheless, most patients still require TURP, either as an emergency or elective surgery (*Taube and Gajraj*, 1989).

Alpha-1 ( $\alpha$ 1)-blockers decrease smooth muscle tone in the prostate, thereby rapidly improving urinary symptoms and flow. Currently available  $\alpha$ 1-blockers include the selective  $\alpha$ 1-blockers terazosin, doxazosin and alfuzosin and the highly selective  $\alpha$ 1A-blocker tamsulosin and silodosin. These agents have comparable efficacy, and the major differences between these agents are their tolerability profiles (*Montorsi and Moncada*, 2006).

By decreasing the resistance,  $\alpha 1$ -blockers can help relieve AUR and improve the chances of successful TWOC (McNeill, 2001).

Longer catheter duration significantly increases the risk of complications such as urinary tract infections, urine leak and catheter obstruction and urethral stricture, all efforts should be made to try to minimize the duration of catheterization and so reduce co-morbidity and healthcare costs (*Fitzpatrick et al.*, 2011).

However, the optimum duration of therapy has not been fully assessed, and there is controversy regarding the length of time a catheter should remain in situ during the initial therapeutic phase (*Kara and Yazici*, 2014).

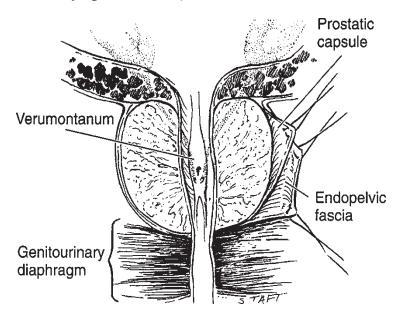
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# AIM OF THE WORK

The aim of this work is to compare between early and late removal of urinary catheter after acute urine retention in patients with Benign Prostatic Hyperplasia under Tamsulosin treatment.

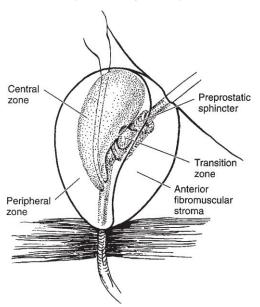
## **A**NATOMY

The prostate is a fibro-muscular and glandular organ lying just inferior to the bladder. The normal prostate weighs about 20 g and contains the prostatic urethra, which is about 2.5 cm in length. It is supported anteriorly by the pubo-prostatic ligaments and inferiorly by the uro-genital diaphragm. The prostate is perforated posteriorly by the ejaculatory ducts, which pass obliquely to empty through the verumontanum on the floor of the prostatic urethra just proximal to the striated external urinary sphincter (*Myers et al.*, 2010).



**Figure (1):** Section of the prostate gland shows the prostatic urethra, verumontanum, and crista urethralis, in addition to the opening of the prostatic utricle and the two ejaculatory ducts in the midline. Note that the prostate is surrounded by the prostatic capsule, which is covered by another prostatic sheath derived from the endopelvic fascia. The prostate is resting on the genitourinary diaphragm (*Tanagho and Lue*, 2013).

According to the classification of *Lowsley*, the prostate consists of five lobes: anterior, posterior, median, right lateral, and left lateral. This classification is often used in cystourethroscopic examinations. After a comprehensive analysis of 500 prostates, *McNeal* divides the prostate into four zones: peripheral zone, central zone (surrounds the ejaculatory ducts), transitional zone (surrounds the urethra), and anterior fibro-muscular zone. The segment of urethra that traverses the prostate gland is the prostatic urethra. It is lined by an inner longitudinal layer of muscle (continuous with a similar layer of the vesical wall). Incorporated within the prostate gland is an abundant amount of smooth musculature derived primarily from the external longitudinal bladder musculature. This musculature represents the true smooth involuntary sphincter of the posterior urethra in males (*McNeal*, 1981).



**Figure (2):** Anatomy of the prostate gland. Prostatic adenoma develops from the periurethral glands at the site of the median or lateral lobes. The posterior lobe, however, is prone to cancerous degeneration (*Tanagho and Lue*, 2013).

### Relationships

Although ovoid, the prostate is referred to as having anterior, posterior, and lateral surfaces, with a narrowed apex inferiorly and a broad base superiorly that is contiguous with the base of the bladder. It is enclosed by a capsule composed of collagen, elastin, and abundant smooth muscle. Posteriorly and laterally, this capsule has an average thickness of 0.5 mm, although it may be partially transgressed by normal glands. Microscopic bands of smooth muscle extend from the posterior surface of the capsule to fuse with Denonvilliers fascia. Loose areolar tissue defines a thin plane between Denonvilliers fascia and the rectum. On the anterior and anterolateral surfaces of the prostate, the capsule blends with the visceral continuation of endopelvic fascia. Toward the apex, the pubo-prostatic 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 (Walz et al., 2007).

Laterally, the prostate is cradled by the pubococcygeal portion of levator ani and is directly related to its overlying endopelvic fascia. Below the juncture of the parietal and visceral endopelvic fascia (arcus tendineus fascia pelvis), the pelvic fascia and prostate capsule separate and the space between them is filled by fatty areolar tissue and the lateral divisions of the dorsal vein complex. Although this is truly a parietal endopelvic fascia, it is commonly referred to as the "lateral prostatic fascia" (Myers, 1994).

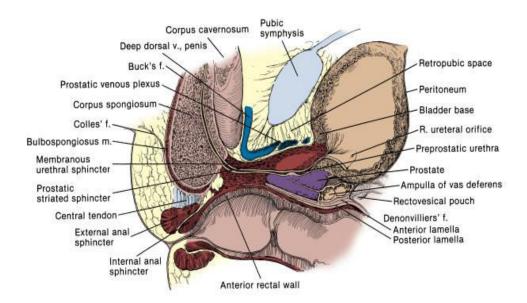


Figure (3): Sagittal section through the prostatic and membranous urethra, demonstrating the midline relations of the pelvic structures (*Hinman*, 1993).

# 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 hypertrophy, 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 (*Chung et al.*, 2012).

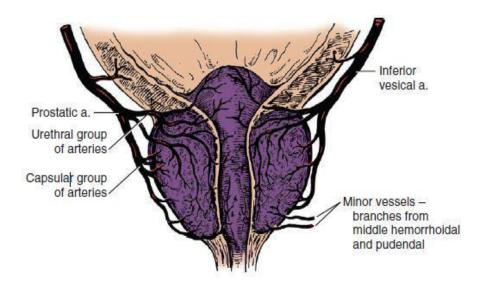


Figure (4): Arterial supply of the prostate (Chung et al., 2012).

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 tissues. Venous drainage of the prostate is abundant through the periprostatic plexus (*Chung et al.*, 2012).

The lymphatics from the prostate drain into the internal iliac (hypogastric), sacral, vesical, and external iliac lymph nodes (*Saokar et al.*, 2010).

## 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 (*Chung et al.*, 2012).

α1-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. Afferent neurons from the prostate travel through the pelvic plexuses to pelvic and thoracolumbar spinal centers. A prostatic block may be achieved by instilling local anesthetic into the pelvic plexuses (*Burnett et al.*, 1995).

### Histology

The prostate is composed of approximately 70% glandular elements and 30% fibromuscular stroma. The stroma is continuous with 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 (*McNeal*, 1984).

The urethra runs the length of the prostate and is usually closest to its anterior surface. It is lined by transitional epithelium, which may extend into the prostatic ducts. The urothelium is surrounded by an inner longitudinal and an outer

circular layer of smooth muscle. A urethral crest projects inward from the posterior midline, runs the length of the prostatic urethra, and disappears at the striated sphincter. To either side of this crest, a groove is formed (prostatic sinuses) into which all glandular elements drain. At its midpoint, the urethra turns approximately 35 degrees anteriorly, but this angulation can vary from 0 to 90 degrees. This angle divides the prostatic urethra into proximal (preprostatic) and distal (prostatic) segments that are functionally and anatomically discrete. In the proximal segment, the circular smooth muscle is thickened to form the involuntary internal urethral (preprostatic) sphincter. Small periurethral glands, lacking periglandular smooth muscle, extend between the fibers of the longitudinal smooth muscle to be enclosed by the preprostatic sphincter. Although these glands constitute less than 1% of the secretory elements of the prostate, they can contribute significantly to prostatic volume in older men as one of the sites of origin of benign prostatic hypertrophy (McNeal, 1988).

Beyond to the urethral angle, all major glandular elements of the prostate open into the prostatic urethra. The urethral crest widens and protrudes from the posterior wall as the verumontanum. The small slitlike orifice of the prostatic utricle is found at the apex of the verumontanum and may be visualized cystoscopically. The utricle is a 6-mm müllerian remnant in the form of a small sac that projects upward and backward into the substance of the prostate. In males with

ambiguous genitalia, it may form a large diverticulum that protrudes from the posterior side of the prostate. To either side of the utricular orifice, the two small openings of the ejaculatory ducts may be found. The ejaculatory ducts form at the juncture of the vas deferens and seminal vesicles and enter the prostate base, where it fuses with the bladder. They course nearly 2 cm through the prostate in line with the distal prostatic urethra and are surrounded by circular smooth muscle (*Chung et al.*, 2012).

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. Each acinus is surrounded by a thin layer of stromal smooth muscle and connective tissue. 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 fibromuscular 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 hypertrophy, which expands to compress the fibromuscular 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 (*Chung et al.*, 2012).

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. The glands are structurally and immunohistochemically distinct from the remaining prostatic glands (which branch directly from the urogenital sinus), which has led to the suggestion that they are of wolffian origin (McNeal, 1988).

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 (*Chung et al.*, 2012).

Up to one third of the prostatic mass may be attributed to the nonglandular anterior fibromuscular stroma. This region normally extends from the bladder neck to the striated sphincter, although considerable portions of it may be replaced by glandular tissue in adenomatous enlargement of the prostate. It is directly continuous with the prostatic capsule, anterior visceral fascia, and anterior portion of the preprostatic sphincter and is composed of elastin, collagen, and smooth and striated muscle. It is rarely invaded by carcinoma (*Chung et al.*, 2012).

## URINARY RETENTION IN BPH

bstruction of the lower urinary tract at or distal to the bladder neck can cause urinary retention. The obstruction may be intrinsic (e.g., prostatic enlargement, bladder stones, urethral stricture) or extrinsic (e.g., when a uterine or gastrointestinal mass compresses the bladder neck causing outlet obstruction). The most common obstructive cause is Benign Prostatic Hyperplasia (BPH) (*Rosenstein et al.*, 2004).

### Pathophysiology:

BPH causes bladder neck obstruction through two mechanisms: prostate enlargement and constriction of the prostatic urethra from excessive alpha-adrenergic tone in the stromal portion of the gland (*Guthrie et al.*, 2004).

The patho-physiology of Benign Prostatic Hyperplasia (BPH) includes both static and dynamic components.

Abnormal and excessive non-malignant growth of prostate tissue is a hallmark of the disease over time. The tissue growth starts impinging on the prostatic urethra and the bladder neck, leading to bladder outlet obstruction and may lead to urinary retention. This is the static component of prostatic hyperplasia (*Lepor et al.*, 2004).

Continued exposure of the prostate to Dihydrotestosterone (DHT) plays a central patho-physiologic role in this age-associated prostate growth and eventual hyperplasia. There is also an increase in the amount and tone of the smooth muscle

within the prostate. Stimulation of alpha1- adrenergic receptors on the bladder neck, prostatic capsule, and stromal tissue results in constriction of the urethral lumen which lead to BPH symptoms and up to urinary retention by another way. This is the dynamic component of prostatic hyperplasia (*Lepor et al.*, 2004).

#### The bladder's response to obstruction

Current evidence suggests that the bladder's response to obstruction is largely an adaptive one. However, it is also clear that many lower tract symptoms in men with BPH or prostate enlargement are related to obstruction-induced changes in bladder function rather than to outflow obstruction directly. Approximately one third of men continue to have a significant irritative or storage symptoms after surgical relief of obstruction (*Abrams et al.*, 1979).

Obstruction induced changes in the bladder are of two basic types. First, the changes that lead to 'detrusor instability' or decreased 'compliance' are clinically associated with symptoms of frequency and urgency. Second, the changes associated with decreased 'detrusor contractility' are associated with further deterioration in the force of the urinary stream, hesitancy, intermittency, increased residual urine and (in a minority of cases) detrusor failure. Acute urinary retention should not be viewed as an inevitable result of this process. Many patients presenting with acute urinary retention have more than adequate detrusor function, with evidence of a precipitating