"COMPARISON OF PREOPERATIVE AND POSTOPERATIVE POST VOID RESIDUAL URINE VOLUME IN UROGENITAL PROLAPSE"

By

Dr. NITU KADAM

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In Partial Fulfillment
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M. S.
(OBSTETRICS AND GYNAECOLOGY)

Under the Guidance of

Dr. B. R. NILGAR MD
PROFESSOR

DEPARTMENT OF OBSTETRICS AND GYNAECOLOGY,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELGAUM, KARNATAKA

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DECLARATION

I hereby declare that this dissertation entitled **"COMPARISON OF PREOPERATIVE AND POSTOPERATIVE POST VOID RESIDUAL URINE VOLUME IN UROGENITAL PROLAPSE"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. B. R. NILGAR** $_{MD}$ Professor, Department of Obstetrics and Gynaecology, Jawaharlal Nehru Medical College, Nehru Nagar, Belgaum – 590 010.

Date:

Place: Belgaum (Dr. NITU KADAM)

KLE UNIVERSITY, BELGAUM,

KARNATAKA

CERTIFICATE

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Date:

Place: Belgaum

Dr. B. R. NILGAR MD
Professor,
Department of Obstetrics and Gynaecology,
J. N. Medical College,
Nehru Nagar, Belgaum – 590 010

KLE UNIVERSITY, BELGAUM,

KARNATAKA

ENDORSEMENT

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Dr. B. R. DESAI MD,
Professor and Head,
Department of Obstetrics and Gynaecology,
J. N. Medical College,
Nehru Nagar,
Belgaum – 590 010

Date:
Place: Belgaum

Dr. V. D. Patil MD, DCH
Principal,
J. N. Medical College,
Nehru Nagar,
Belgaum – 590 010

Date:
Place: Belgaum

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(Dr. NITU KADAM)

Place : Belgaum

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Date:

Place: Belgaum **Dr. NITU KADAM**

LIST OF ABBREVIATIONS USED

ATFP	-	Arcus tendineus fascia pelvis
LMP	-	Last menstrual period
POP-Q	-	Pelvic organ prolapse quantification
PVR	-	Post void residual
TVL	-	Total vaginal length
UTI	-	Urinary tract infection

ABSTRACT

Background and objectives

The genital prolapse causes bladder dysfunction because of the anatomical closeness of female genital organs to the pelvic urinary system. PVR is a key marker for the evaluation of the efficacy of bladder emptying particularly in women with pelvic organ prolapse and lower urinary tract dysfunction. Objectives of the present study were to compare pre and post operative post void residual urine volume and to know the relation of PVR to urinary symptoms and prolapse.

Methods

The present study was conducted in Department of Obstetrics and Gynaecology, KLES Dr Prabhakar Kore Hospital and Medical Research Center, Belgaum over a period of one year during October 2008 to September 2009 on 65 patients admitted with urogenital prolapse. Detailed history, general physical examination was done as per predesigned and pretested proforma. Grading for prolapse was done by POP-Q, Baden walker halfway. PVR was measured before and after operation.

Results

Patients were aged between 25 to 72 years. 45 patients were postmenopausal. Age has shown significant relation with the raised PVR > 50 ml ($p=0.007$). Out of 65 cases, 11 had second, 48 had third degree and 6 had procedentia according to Baden Walker system. Urge and stress incontinence were complained by 43% and 26% of patients respectively and increased

frequency and nocturia was complained by 68% and 65% of patients. Storage symptoms were not significantly associated with degree of prolapse or raised PVR. straining to void, incomplete emptying and has to reduce to void were present in 42, 46 and 47 patients respectively and showed significant association with degree of prolapse. Except incomplete emptying other two were associated with raised PVR.

Conclusions and interpretation

Raised PVR has shown significant association with increasing degree of prolapse as well as obstructive urinary symptoms like straining to void, incomplete emptying and has to reduce prolapse to void. Vaginal hysterectomy with anterior colporrhaphy was effective procedure in reducing elevated PVR in prolapse patients.

Key words:

Pelvic organ prolapse; Post void residual urine (PVR); Urinary symptoms; Vaginal hysterectomy with anterior colporrhaphy.

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

INTRODUCTION

The pelvic organ prolapse is the downward displacement of the structures that are normally located adjacent to vaginal vault. These conditions are commonly affecting a progressively large percentage of women as age advances. Though mortality is negligible, significantly morbidity is associated with prolapse. In areas of high parity and little or no access to health care, countless women suffer from problems associated with pelvic organ prolapse, with no real possibility of resolution. These conditions have a direct effect on urinary, gastrointestinal and sexual functions. And it can only be appreciated by those women burdened and living with these problems on a day today basis.

Treatment of pelvic organ prolapse and the associated symptoms constitutes a major subject in gynecology. Providing permanent relief from this classical malady, by restoring normal anatomy and maximum physiological functions always tests the ingenuity of gynecologists.[1]

Prolapse is associated with voiding difficulty, bladder outlet obstruction, occult stress incontinence and lower urinary tract symptoms.[2]

Voiding difficulty in the female is a condition in which the bladder fails to empty completely and easily after micturition. The symptoms of voiding dysfunction are an unreliable guide to diagnose the presence of voiding difficulty in women. The detection of voiding difficulties before pelvic operations gives a indication to the surgeon of a delay in resumption of spontaneous voiding and the likely need for prolonged catheterization.[3]

Urinary symptoms in voiding difficulties are frequency, stress incontinence, Urge incontinence, incomplete empyting, poor stream, straining to void.

Post-void residual volume (PVR) is the urine volume left in bladder at the completion of micturition. It is a key marker for the efficacy of bladder emptying. PVR measurement is essential, particularly in women with the symptoms of pelvic floor dysfunction, including those with lower urinary tract and pelvic organ prolapse symptoms.[4]

PVR Measurement techniques include:

1. Transvaginal ultrasonography.[5]
2. Transabdominal ultrasonography.[6]
3. Bladder catheterization.[7,8]

These different techniques might account for the differences in recommended upper limit of normal PVR. Another one key source of the possible variation is the delay in measuring PVR after micturition.[4]

Post-void residual urine volume has been examined for its relationship to voiding dysfunction. Elevated PVR values are associated with poor detrusor muscle contractility or increased urethral resistance, as well as abnormal uroflowmetry.[9]

Prolapse has a significant positive relationship with high PVR. The proposed mechanism of genital prolapse is the distortion or kinking effect of the prolapse on the urethra to create bladder overflow obstruction. And women with

high grade prolapse have increased urethral closure pressure and pressure or transmission ratios that decrease after the prolapse is reduced. Hence there is need for studies which demonstrate that prolapse surgery can reduce or eliminate any elevated PVR.[4]

PVR IS DISCUSSED UNDER FOLLOWING HEADINGS

1. Anatomical considerations in pelvic organ prolapse.
2. Functional anatomy of lower urinary tract.
3. Bladder neurophysiology.
4. Bladder dysfunction.
5. Anatomical changes in prolapse leading to bladder dysfunction.
6. Prolapse degrees classifications.
7. Bladder dysfunction symptoms.

ANATOMICAL CONSIDERATIONS IN PELVIC ORGAN PROLAPSE

1. Bony pelvis

Lordosis of the lumbo-sacral portion of the spines places the pelvic inlet in a position so that the posterior aspect of the pelvic inlet is approximately 60º above the anterior aspect.

The more vertical orientation of the pelvic inlet deflects force onto the symphysis pubis rather than pelvic outlet and urogenital hiatus.

Consequently the pelvic outlet is shielded from downward stresses in the anatomically normal situation.[1]

2. Muscles of pelvic diaphragm

The muscles of pelvic diaphragm form a basin or covering of the pelvic outlet and grouped together, called as levator ani or sling.

Within this diaphragm is the urogenital hiatus which is large enough to allow birth of child. (Figure 1)

The most medical portion of pelvic diaphragm is formed by puborectalis, the muscular boundary of urogenital hiatus. The Obstetric axis of the pelvis passes through the urogenital hiatus just medial to puborectalis.[1]

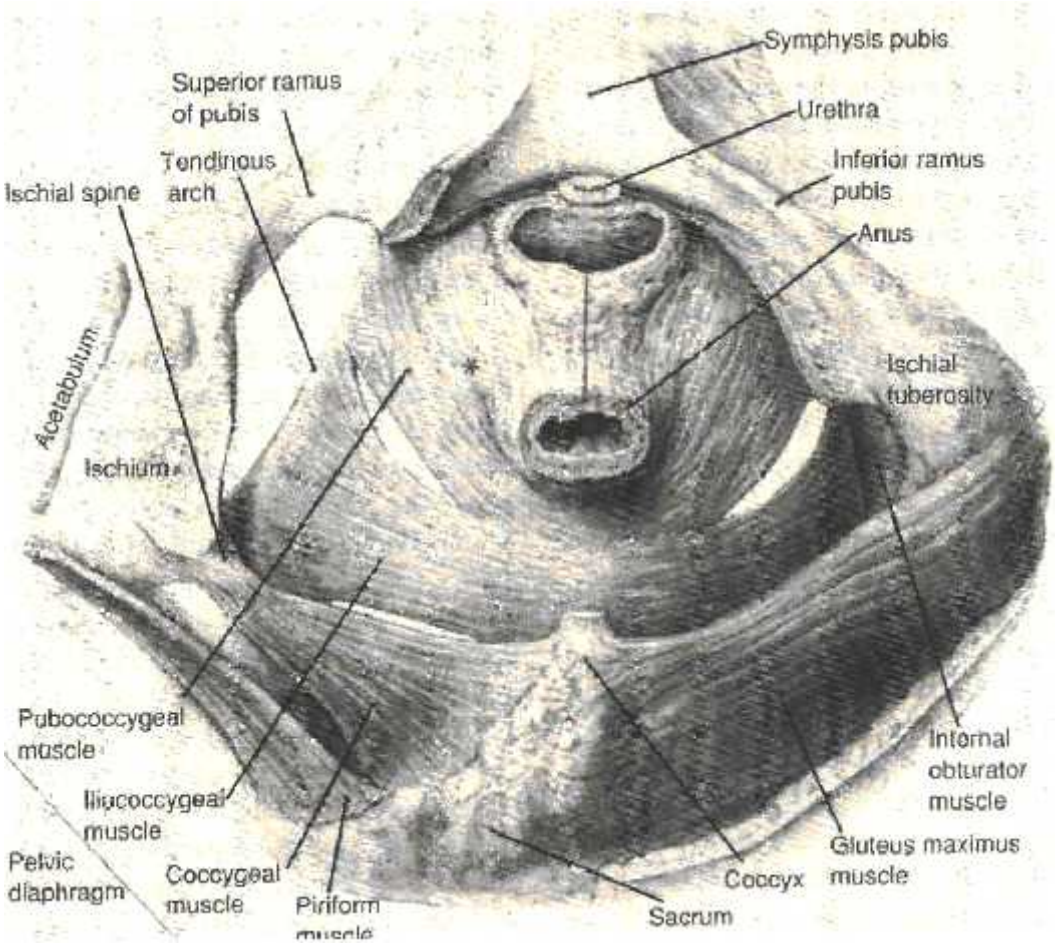


Figure 1: Levator ani muscles seen from below. That portion of the pubococcygeus which inserts into the rectum and forms a 'U' behind it is called puborectalis

In standing position, the puborectalis is horizontal and palpable as 2.0 to 2.5 cm band of voluntary muscle on distal one third of vagina. When well innervated it closes vaginal hiatus and forms the bulk of the pelvic diaphragm.

Pubococcygeous and ilieococcygeus muscles cover the posterior and lateral portions of the pelvic outlet respectively (Figure 1).

The insertion of ileococcygeus is a thickening of the pelvic side wall parietal fascia that extends from the ischial spine to a point on the public bone (pubic tubercle).

This line of insertion is known as arcus tendineus fascia pelvis (fascial arch) or white line. The white line serves the function of midvaginal lateral support**.** *Paravaginal and proximal pararectal defects are located immediately medial to the white line.*

The lateral supports of the anterior and posterior vaginal septa merge and they are not separate in the proximal half of the vagina.

Posterior to the iliococcygeus, the pelvic floor is covered by the coccygeus muscle and closely associated with sacrospinous ligament.

The most posterior portion is covered by piriformis muscle .The midline confluences of the levator ani forms a particularly strong band of connective tissue between coccyx and anus known as the levator plate or median raphe.

This plate is oriented horizontally in the standing position, vagina and rectum are the weakness of pubococcygeus and iliococcygeus muscles. And it may allow the levator plate to sag and descend permanently.[1,10]

3. Pudendal nerve

It is the important motor and sensory nerve of the pelvic floor and perineum. It descends from the area posterior to the ischial spine under the sacrospinous ligament into Alcock's canal.

Alcock's canal is located in the ishiorectal fossa immediately adjacent to the inferior fascia of the pelvic diaphragm. In this location, the pudendal nerve is subjected to stretch and pressure during the descent of a fetus through the pelvis. The muscles of pelvic diaphragm are also subjected to great pressure and stretch during labour.

Neuropathy of the pudendal nerve and myopathy of the levator muscles are believed to be active contributing factors in the development of pelvic organ prolapse.[1]

4. Endopelvic fascia

The connective tissues of the pelvis are collectively known as the endopelvic fascia. The irregular space between the pelvic diaphragm, the muscular pelvic sidewall and visceral peritoneum is the location of the endopelvic fascia which is further divided into:

4.1 Parietal fascia

4.2 Visceral fascia

4.3 Deep endopelvic connective tissue

Parietal fascia

- Obturator fascia: Superior to the arcus tendineus levator ani and below the linea termination.
- Levator ani fascia: This fascia is continuous across the pelvic floor, blending laterally with obturator fascia at arcus tendineus levator ani and centrally with the levator plate and the visceral fascia at urogenital hiatus.

- Coccygeus fascia (sacrospinous ligament): Extends from ischial spines laterally to sacrum medially. It is *alternative support* when *uterosacral ligament* is insufficient.
- Piriformis fascia: Thinnest and most posterior of parietal fascia.

Visceral fascia

- The visceral fascia covering vagina, uterus, bladder and rectum is loose and highly elastic. These structure allow for the high degree of physiologic distension necessitated by the function of the pelvic organs.

Deep endopelvic connective tissue includes

- Six Ligaments
- Two septa
- One ring

Six pericervical ligaments form the paracolpium

These ligaments are pubocervical, cardinal and uterosacral on both sides. Paracolpium results in the suspension of the cervix in the posterior pelvis and it leads to the consequent placement of the vagina directly over the levator plate and away from direct exposure to the urogenital hiatus. Therefore, pressure from above tends to close the vaginal vault and no tendency towards prolapse.[1]

Two Septa or Fascia

These condensations of fibroelastic connective tissue are in the close contact with vaginal epithelium and visceral fascia of the adjacent organs.

Between bladder and vagina is pubocervical septum. And between rectum and vagina is rectovaginal septum.

One ring

The pericervical ring is the single location where all deep endopelvic connective tissue support structures converge.

No complete anatomic method to reconstruct the proximal anterior vaginal support exists if cervix and its surrounding support tissue are absent. Some form of anatomic distortion (Shortening of the vagina or plication) is necessary to compensate for this defect.

DeLancy divided vaginal support into three levels.[1,10]

1. Proximal vaginal level I support: It is formed by the ligaments of Paracolpium (Figure 2). Damage to level I support results in

    - Utero-Vaginal prolapse
    - Post hysterectomy vaginal prolapse
    - Enterocele

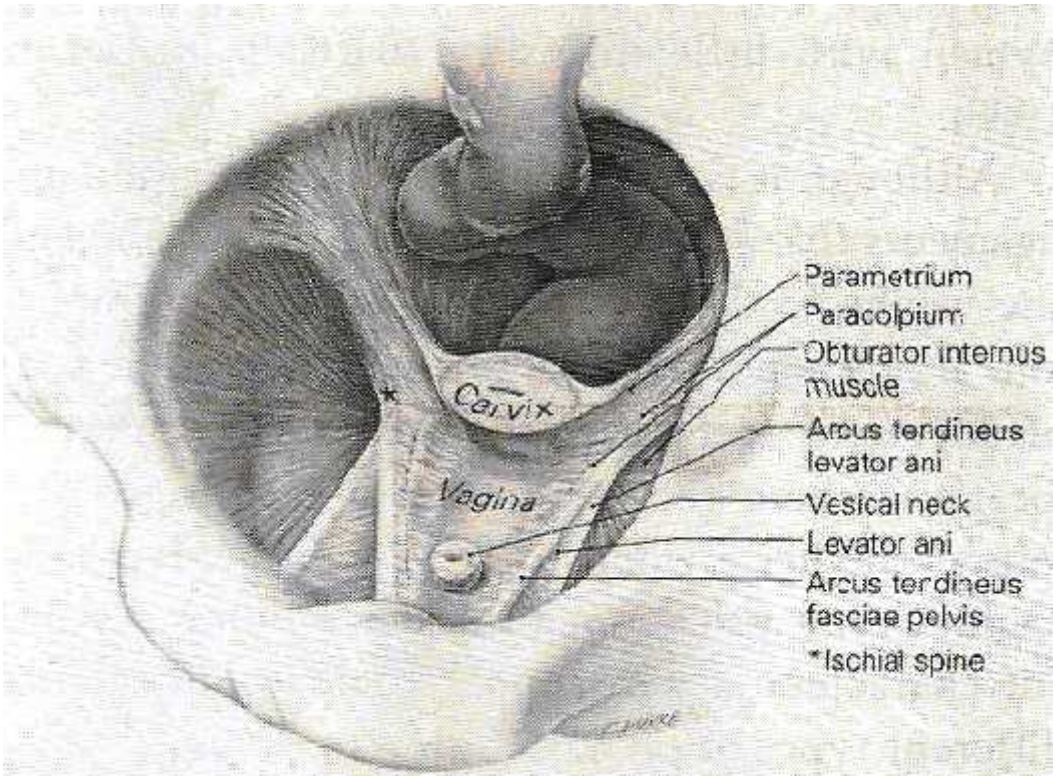


Figure 2: Supportive tissues of the cervix and upper vagina. Bladder has been removed above the vesical neck

Cause of level I support problems are at and above the level of ischial spines. Primary load bearing elements are uterosacral ligaments and to a lesser extent the cardinal ligaments.

2. Mid-vaginal level II support: It is due to lateral attachment of the fascial septa to the pelvic sidewalls; the septa attach to the arcus tendineus fascia pelvis and the arcus tendineus fascia rectovaginalis (Figure 3).

   Damage to this level result in paravaginal and pararectal defects, Cystocele and rectocele are due to central or lateral defects in level II support.

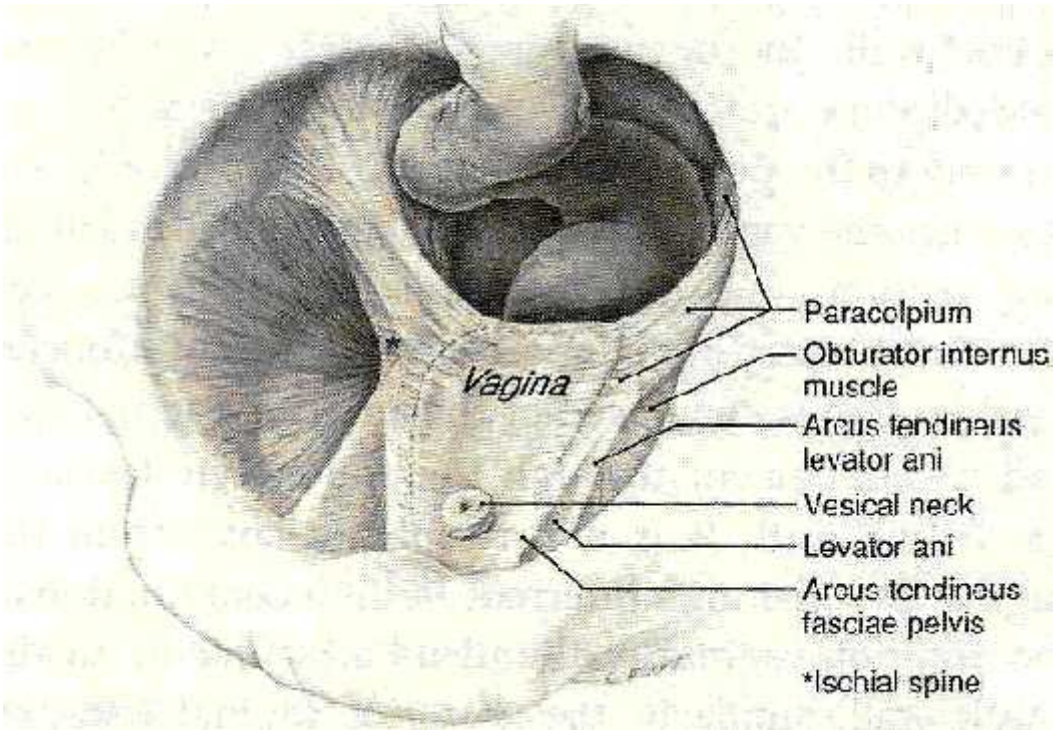


Figure 3: Vagina and supportive structures drawn from dissection of 56 year old cadaver after hysterectomy. The paracolpium extends along the lateral wall of the vagina.

3. Level III Support: It is attributed to fusion to the urogenital diaphragm anteriorly and to the proximal perineum posteriorly (Figure 4).

Damage at these sites result in the urinary incontinence anteriorly and in perineal body deficits posteriorly.[1,10]

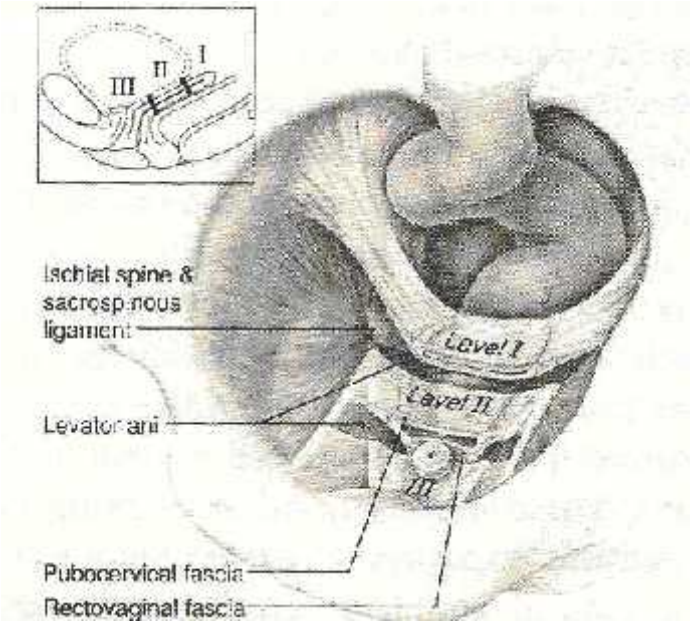


Figure 4: Level I (Suspension) and level II (Attachment). In level I the parcolpium suspends the vagina from the lateral pelvic walls. Fibres of level II vagina is attached to arcus tendineus fasciae pelvis and superior fascia of levator ani

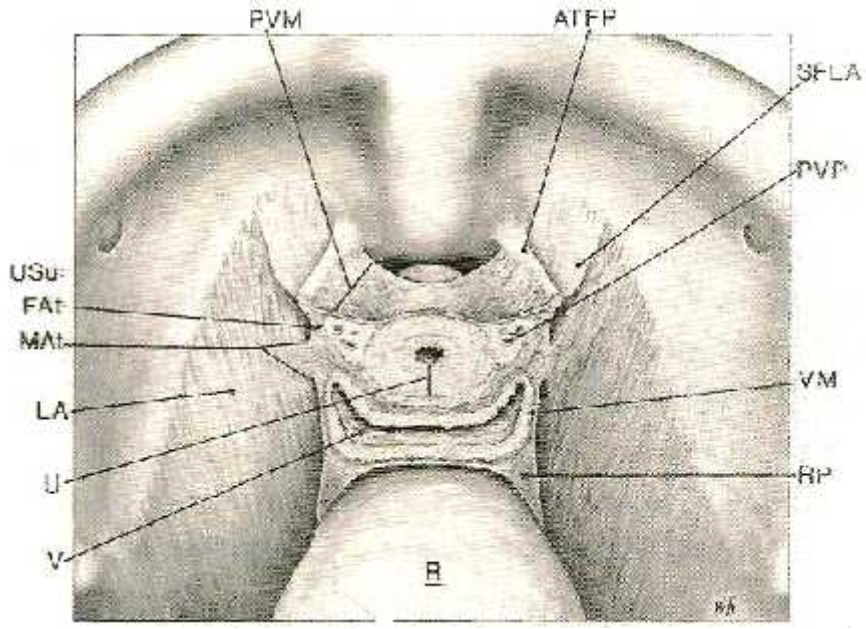


Figure 5: Relationship of the supportive tissues of the urethra (USu) to the pubovesical (PVM). Cross section of the urethra (U), vagina (V), arcus tendineus fasciae pelvis (ATFP), and superficial facia and levator ani (SFLA), the urethral supports (USu) attach the vaginal surface of urethra and vagina to levator ani muscles.

Para vaginal defects due to damage in the arcus tendineus fascia pelvis (ATFP) can occur in one of three different ways.

1. The entire ATFP may remain attached to the pelvic side wall with the pubocervical fascia breaking away from the arcus.

2. The ATFP may pull away from the side of the pelvis but remains attached to the pubocervical fascia.

3. The ATFP may split with a portion of it remaining attached to the side wall and a portion tearing away but remaining attached to pubocervical fascia.[1]

FUNCTIONAL ANATOMY OF THE LOWER URINARY TRACT

The lower urinary tract can be divided into the bladder and urethra. At the junction of these two, vesical neck is present. It is a hybrid structure, represents that part of the lower urinary tract where the urethral lumen transverses the bladder wall before becoming surrounded by the urethral wall.

The vesical neck is considered separately because of its functional differentiation from bladder and the urethra.[10,11]

Trigone

There is visible triangular area within the bladder known as vesical trigone.

The two ureteral orifices and the internal urinary meatus form its apices.

The base of the trigone triangle i.e. interureteric ridge forms useful landmark in cystoscopic identification.

This triangular elevation has a specialized group of smooth muscle fibers that lie within the detrusor and arise from a separate embryological primordium. They are continuous above with ureteral smooth muscle and below with the urethra and form a ring inside detrusor loops at the level of the internal urinary meatus.

The mucosa over trigone frequently undergo squamous metaplasia differs from the rest of bladder.[10,11]

Urethra

The urethra holds urine in the bladder therefore important structure for urinary continence.

It is a complex tubular viscous organ extending below the bladder.

In its upper third, it is clearly separable from adjacent vagina but its lower portion is fused with the wall of the vagina. In its substance, a number of elements are embedded which are important for lower urinary tract function (Figure 6).

Region of Urethra	**Paraurethral structures**
Intramural Urethra	Urethral lumen transverses the bladder wall
Mid Urethra	Sphincter urethral muscle, Pubovesical muscle
Perineal Membrane	Compressor urethral muscle, urethrovaginal sphincter muscle
Distal Urethra	Bulbocavernous muscle

Urogenital sphincter

The outer layer of urethra is formed by the muscle of the striated urogenital sphincter.

In upper two third fibers are circular in orientation. In distal part, they leave the confines of the urethra and encircle the vaginal wall as the vaginal sphinder.

It extends along the inferior public rami, above the perineal membrane (urogenital diaphragm) as compressor urethrae. Slow twitch muscle fibres, which are well suited to maintain the constant tone exhibited by this muscle.

In addition, *voluntary muscle activation increases urethral contraction during times when increased closure pressure is needed.*[11]

Urethral smooth muscles

These are continuous with trigone and detrusor. It has an inner longitudinal layer and this outer circular layer. The former is prominent of two. Urethral smooth muscle lies inside the striated urogenital sphincter and present in upper 2/3 of urethra (Figure 6).

The configuration of the circular muscle suggests a role in constricting the lumen and the longitudinal muscle, may help to shorten and funnel the urethra during voiding.[10]

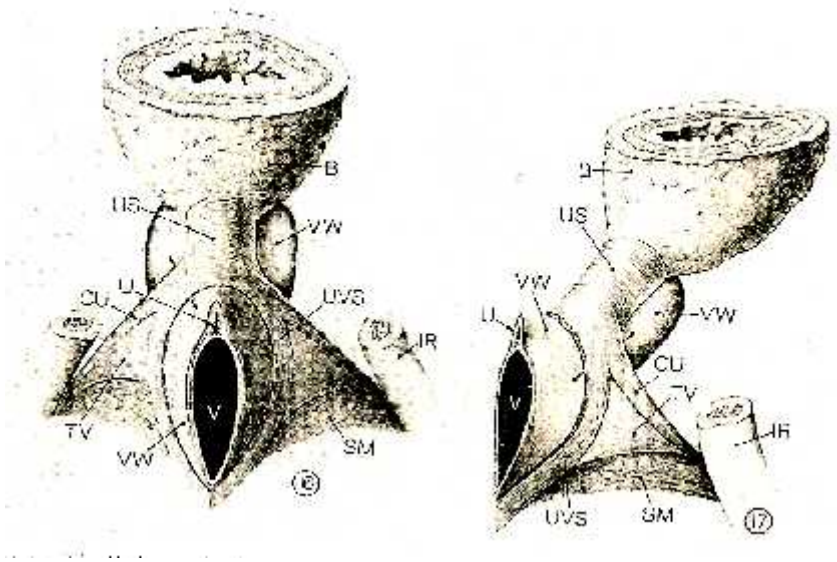


Figure 6: Striated urogenital sphincter muscle seen from below after removal of the perineal membrane and pubic bones including US, urethral sphincter; UVS, urethrovaginal sphincter; CU, compressor urethrae. Bladder (B), Ischiopubic ramus (IR), transverse vaginae (TV) muscle, smooth muscle (SM), urethra (U) and vagina (V), vaginal wall (VW).

Submucosal vasculature

Submucosal layer has a specialized type of arteriovenous anastomoses. They are formed in such a way that the flow of blood into large venules can be controlled to inflate or deflate them. This would assist in forming *a watertight closure of mucosal surface*.[11]

Mucosa

The mucosal lining of the urethra is continuous above with the transitional epithelium of the bladder and below with non-keratinizing squamous eithelium of vestibule. It is hormone sensitive.

A series of glands are found submucosa primarily along the dorsal (vaginal) surface of the urethra. The location of urethral diverticulum (which is derived from cystic dilatation of these glands) follows their distribution.

It stretches fascia, therefore fascia approximation after diverticular excision is required.[11]

Vesical Neck

It is a region where the detrusor musculature including the detrusor loop, surrounds the trigonal ring and the meatus. It has unique functional characteristics. Sympathetic denervation or damage of this area results in vesical neck remaining open during rest.

It is associated with stress incontinence and simple urethral suspension is often ineffective in curing this problem.

- The *extrinsic continence mechanism* or external sphincteric mechanism usually refers to that group of structures that respond when an individual is instructed to stop the urine stream.

  During the efforts of constricting the urethral lumen by the striated urogenital sphincter, an elevation of the vesical neck is caused by contraction of levator ani-muscle.

- The *intrinsic continence mechanism* consists of structures within vesical neck, which are not activated by contraction of the voluntary muscle. It is this system that fails in the patients whose vesical neck can be seen open at rest.[11]

Pelvic Floor

Pelvic floor consist of the components lying between pelvic peritoneum and vulval skin. It includes peritoneum, viscera, endopelvic fascia, levator ani muscles, and perineal membrane and external genital muscles.[11]

Perineal membrane and external genital muscles

In the anterior portion of the pelvis, below the pelvic diaphragm is a dense triangular membrane containing a central opening, called as perineal membrane (urogenital diaphragm).

This lies of level of the hymenal ring and attaches the urethra, vagina and perineal body to the ischiopubic rami.

Just above the perineal membrane, compressor urethrae and urethrovaginal sphincter muscles are present. The correct anatomy explains that pressures during a cough are greatest in the distal urethra, where the compressor urethrae and urethrovaginal sphincter can squeeze the lumen in anticipation of a cough.[11]

Position and mobility of the urethra

The position and mobility of the bladder and urethra are recognized as important for urinary continence. Upper portion of the urethra and vesical neck are normally mobile structure where as the distal urethra remains fixed in its position.

Urethral support is dynamic rather than static and urethral position is determined by both, attachments to bone and to the levator ani-muscle.

The Resting position of urethra is high within pelvis, Some 3 cm above the inferior surface of public bone. It is above the insertion of pubourethral ligament which attaches near the lower margin of the public bones.[11]

Maintenance of this position would be best explained by the constant muscular activity and levator ani. The upper 2/3 urethra is mobile and under voluntary control.

At the onset of micturation, relaxation of the levator ani muscle allows the urethra to descend and obliterates the posterior urethrovesical angle. Resumption of the normal tonic contraction of the levator ani muscle allows the urethra to its normal position.

The anterior vaginal wall and urethra arise from the urogenital sinus and intimately connected. *The support of the urethra depends upon the vaginal wall attachments and attachment of perineal tissue to the muscles and fascia of the pelvic wall.*[12]

On the either side the arcus tendineus fascia pelvis, a band of connective tissue attached laterally to lower 1/6 of Public bone.

The layer of tissue that provides urethral supports has two lateral attachments. These are fascial attachment and muscular attachment.

The fascial attachments of the urethral supports connect the peri- urethral tissues and anterior vaginal wall to the ATFP- Paravaginal fasical attachment. Pubourethralis muscle (part of levator ani) supports and maintains the resting tone in urethra.[11]

BLADDER NEUROPHYSIOLOGY

Normal micturation is coordinated on autonomic and somatic nervous system. Autonomic pathway (Figure 7);

1. Parasympathic pathway
2. Sympathetic pathway

Parasympathetic innervation of the lower urinary tract is by pelvic nerve. S1, S2, S3 parasympathetic nerves are cholinergic for bladder. It causes contraction of bladder wall and relaxes the sphincter vesicae.

Parasympathetic nerves from S1, S2, S3 comes to superior hypogastric plexus, then along with sympathetic post ganglionic fibres, relay with pelvic plexus.

Pelvic nerves form interconnected nerve stands in the pelvic fascia located lateral to rectum, internal genitalia and lower urinary tract. Branches from pelvic plexus pass laterally to the bladder and trigone and to the upper third of the vagina.

Due to anatomic proximity of these branches to the cardinal ligament and to the upper aspect of vagina, varying degrees of voiding dysfunction can result from surgical disruption of autonomic innervations and fascial support to the bladder and urethra.

Sympathetic enervation causes stimulation beta adrenergic receptors, located in bladder smooth muscles. It promotes bladder accommodation. And

stimulation of α adrenergic receptors cause urethral contraction and an increase in urethral closure pressure.[13]

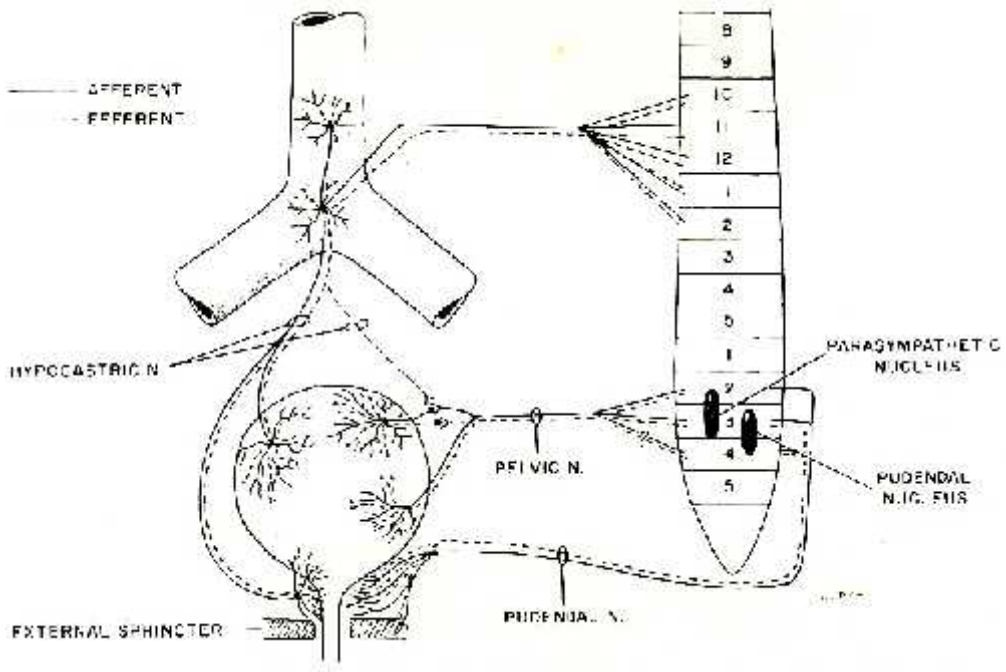


Figure 7: Parasympathetic and sympathetic pathways involved in micturition

Central mechanism

The brain stem centre is situated in the pontine reticular formation.

Pontine micturation centre: It is responsible for the detrusor contraction, Promoting striated sphincter relaxation causing efficient empting of bladder.

Cerebellum: It receives sensory input from the bladder and pelvic floor muscles. It coordinates bladder contraction with striated sphincter relaxation and maintains tone of the pelvic floor musculature.

Anterior frontal cortex and the anterior cingulated gyrus: It causes conscious inhibition of micturation reflex.

Pontine reticular formation

It is origin of the common pathway of efferent impulse, and exerts both facililatory and inhibitory effects on the spinal cord centre.

Conscious inhibition of the micturation reflux occurs in the medial wall of the anterior frontal cortex and the anterior cingulated gyrus.[13]

Micturation refluxes

First loop: Cerebral inhibitory control of the Pontine micturation centre which is responsible for inactivating the micturation reflux.

Second loop: Micturation reflex, afferent pathways from the lower urinary tract to Pons and efferent pathways to the bladder and urethra.

Third loop: Responsible for the coordination of detrusor, urethra and pelvic floor during filling and empting of the bladder.

Fourth loop: The voluntary control of the urethral sphincter function.[13]

Voiding reflux

Voiding reflux is mediated by spinobulbar circuit that passes through Pons and involves both activation of the parasympathetic input to bladder, and inhibition of sympathetic input and somatic input to the urethra.

Estrogen and progesterone receptors present in bladder and urethra, which affect autonomic receptor distribution.[13]

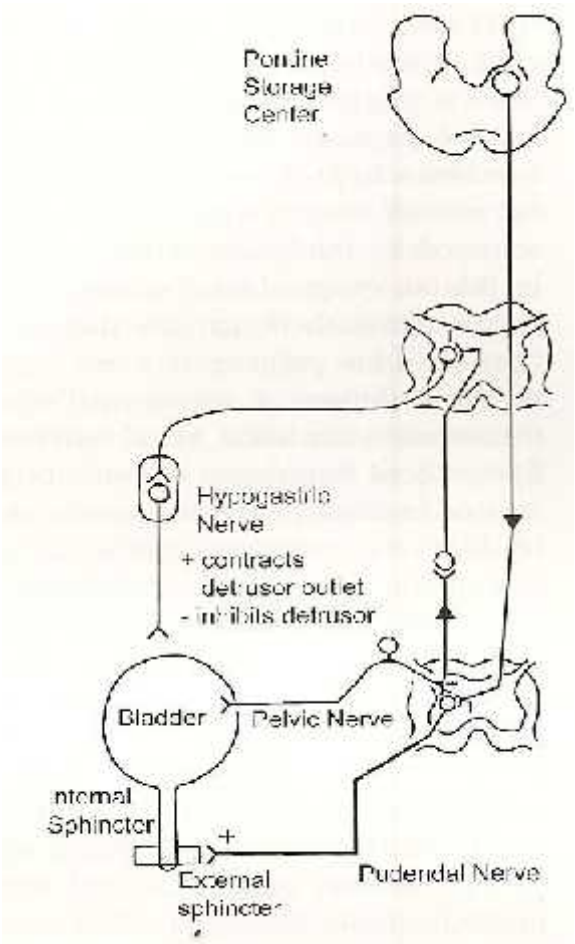


Figure 8: Guarding reflux (Filling phase)

Negative feedback mechanism

↓

Vesical sympathetic reflux

↓

Allows bladder filling

↓

Trigger to increase in sympathetic input to bladder

↓

Vesical afferent input during bladder filling

↓

Activates pudendal motor neurons

↓

Thus triggering sphincter contraction and contributing to urethral closure called as “guarding reflux”. (Figure 8)

Micturation reflux (Figure 9)

Area designated as the pontine micturation centre which is connected to the sacral micturation centre via spinal pathways in posterior and lateral columns. When neuronal connections are intact, micturation is accomplished by activation of the reflux which results in a coordinated interaction between the detrusor contraction and the sphincter relaxation.[13]

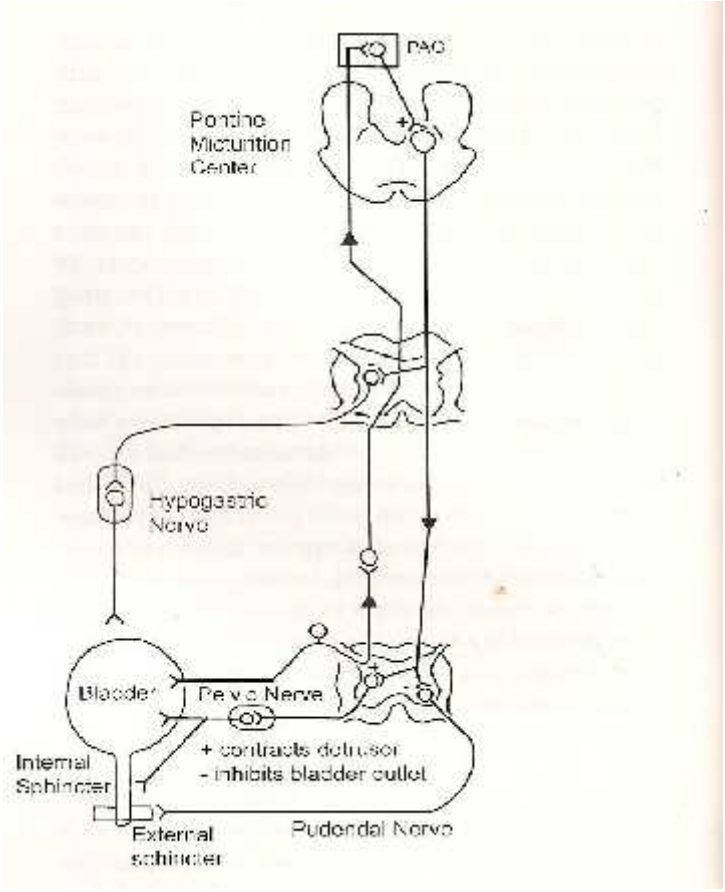


Figure 9: Micturation reflex

BLADDER DYSFUNCTION

Bladder dysfunction is divided in to:

- *Overactive bladder* represents unwanted increase detrusor pressure during urinary storage.
- *Underactive bladder* describes failure to generate sufficient destrusor contraction during bladder emptying.
- *Over active outlet* indicates obstruction during emptying.
- *Underactive outlet* suggests decrease resistance during storage.

Symptomatic presentation of bladder dysfunction

	Overactive	Underactive
Bladder	"urge" – Pressure too much Symptoms: Urgency, Urge incontinence, Frequency, Nocturia	"Retention"-Pressure too little. Symptoms: Hesitancy, Nocturia, Straining, overflow incontinence.
Outlet	Underactive	Overactive
	"stress symptoms"- Resistance too little Symptoms: Urinary loss with cough, sneeze, stress incontinence.	"Obstruction" –Resistance too much Symptoms: Hesitancy, staining, Incomplete emptying, Nocturia, Overflow incontinence.

Incontinence symptomatic classification:

1. Stress incontinence:

Symptom: Involuntory loss of urine with activity eg. cough, sneeze, laugh, lift,strain.

In the absence of true bladder contraction, the involuntary loss of urine results from increase in intraabdominal pressure which overcomes the resistance of the bladder outlet is called as stress incontinence.

The decrease in bladder outlet or urethral resistance may result from poor anatomical support of the bladder neck (urethral hyper mobility) or loss of urethral function (intrinsic sphincter deficiency).[14]

2. Urge incontinence

Symptom: The loss of urine with feeling of urgency, voiding before ability to toilet.

It is the involuntary loss of urine resulting from increase in bladder pressure secondary to true bladder contraction. An involuntary contraction may be result of detrusor hyperreflexia or detrusor instability.

Patients with other symptoms of motor urgency are frequency, urgency, nocturia. Sensory urgency is the sensation of urinary urgency without true contraction.[14]

3. Overflow

It is the involuntary loss of urine resulting from urinary retention. Such retention may result from inadequate bladder contractibility or outlet obstruction.

Urinary loss occurs when the increased bladder pressure overcomes the urethral resistance.[14]

4. Urgency and frequency without incontinence

Urinary urgency and frequency without incontinence may be product of abnormal sensation or bladder contractile activity.

5. Functional

Involuntary loss of urine resulting from deficiency to perform toileting functions secondary to physical and mental limitations.[14]

Causes of voidind difficulties and retention

1. Neurological

A. Upper motor neuron lesion
B. Lower motor neuron lesion
C. Autonomic lesion
D. Local pain reflex

Myelodysplasia, herniated disc, Diabetic neuropathy, multiple sclerosis and spinal cord injury etc. are the important neurological causes.[12]

2. Pharmacological causes

A. Tricyclic antidepressants
B. Anticolinergic agents
C. Alfa adrenergic agents
D. Ganglionic blocking agents
E. Epidural anesthesia

Epidural anesthesia is the most frequent pharmacological cause of voiding dysfunction. Anticholinergic drugs like Oxybutinine, tolterodine used for overactive bladder symptoms can give raise to voiding difficulties. Hence, it is vital to exclude residual urine before prescribing these medications.[12,14]

3. Inflammatory

Acute urethritis, cystitis, vulvovaginitis, acute anogenital infections cause inflammation.

Painful stimuli can cause voiding difficulty due to urethral edema or by urinary irritation to inflamed urethral mucosa as well as vaginal mucosa. Anogenital herpes infection is a recognized cause of voiding difficulty.[14]

4. The hypersensivitive female urethra

It presents with symptoms of frequency, urgency and nocturia. Urodynamic testing reveals a high incidence of obstructed voiding and a lower incidence of detrusor overactivity.

5. Obstructive causes

A. Distal urethral stenosis
B. Acute urethral edema of surgery
C. Foreign body
D. Pelvic mass – Retroverted gravid uterus, hematocolpos uterine fibroid, ovarian cyst and fecal impaction.
E. Cystocele
F. Uterine prolapse

Intrinsic causes of obstruction (Urethral stenosis, foreign bodies, calculi, scarring from instrumentation) are uncommen in women.

Extrinsic causes of obstruction include retroverted uterus, pelvic masses (uterine fibroids and ovarian cysts), chronic constipation and hematocolpos associated with retention and voiding difficulty. Distortion of urethra following genital prolapse is also recognized cause of voiding difficulty.[14]

6. Early postpartum voiding difficulty

Voiding difficulty has been noted in early postpartum period particularly after vaccum extraction. The risk factors are prolonged first and second stage of labour and birth weight more than 3800gms.

7. Psychogenic

There is a relationship between retention of urine and a stressful event such as a child birth, marital discord, surgical treatment or rape. Hysteria, depression and schizophrenia may be associated with this problem.

8. Endocrine

Hypothyroidism and diabetes mellitus can develop urinary retention as a result of peripheral neuropathy causing bladder atony.[14]

9. Detrusor myopathy

It leads to detrusor muscle cell degeneration, fatty replacement, hydropic cytoplasmic changes and perinuclear vacuoles. The inclusion bodies in detrusor act a cause of the primary detrusor myopathy causing urinary retention.[14]

10. Urethral sphincter hypertrophy

This is a primary disorder in which urethral sphincter fails to relax. Bladder hypo or acontractility appears secondary to it.

11. Overdistention injury

Overdistention of bladder causes voiding difficulty. Urinary retention can result in a hypo or acontractile bladder. Irreversible detrusor muscle damage due to lying down of collagen can occur as a result of overdistention. An increase in vesical pressure leads to weakening of the bladder wall due to ischemic changes.[14]

1. **Iatrogenic**
2. **Idiopathic**

Etiological presentation of bladder dysfunction

Bladder	**Overactive**	**Underactive**
	• Idiopathic • Neurogenic • Obstruction	• Distention-drugs • Neurogenic • Muscular
Outlet	**Underactive**	**Overactive**
	• Anatomic hypermobility • Intrinsic sphincter deficiency	• Iatrogenic surgery • Neurogenic • Anatomic atrophy

ANATOMICAL CHANGES IN PROLAPSE LEADING TO BLADDER DYSFUNCTION

Anterior vaginal prolapse cystocele is defined as pathologic descent of the anterior vaginal wall and overlying bladder base. Nichols and Randall described two types (Figure 10).

1. Distension
2. Displacement

Distension cystocele

It was produced by overstretching of the vaginal wall caused by the fetal head during childbirth. It is often related to long labor or the development pressure necrosis, or occasionally to a short precipitous labor in which distention was so rapid that the vagina was not given time for elastic adaptation.

Menopause, estrogen withdrawal, defective connective tissue disorders can be also factors.

It can be illustrated by absent vaginal rugal folds. Midline defects are involving isolated loss of integrity pubocervical ligaments.[15]

Displacement cystocele

This translocation cystocele is associated with vaginal descent which occurs due to overstretching or attenuation of the lateral connective tissue support to the vagina. The lateral attachments of vagina and cervix get stretched as they are pushed in front of baby's head. And finally, leads to pathologic detachment or

elongation of the antero-lateral vaginal supports, arcus tendineus fascia pelvis from white line.

It may occur unilaterally or bilaterally often coexist with some degree of distension cystocele with urethral hyper mobility and with apical prolapse.[15]

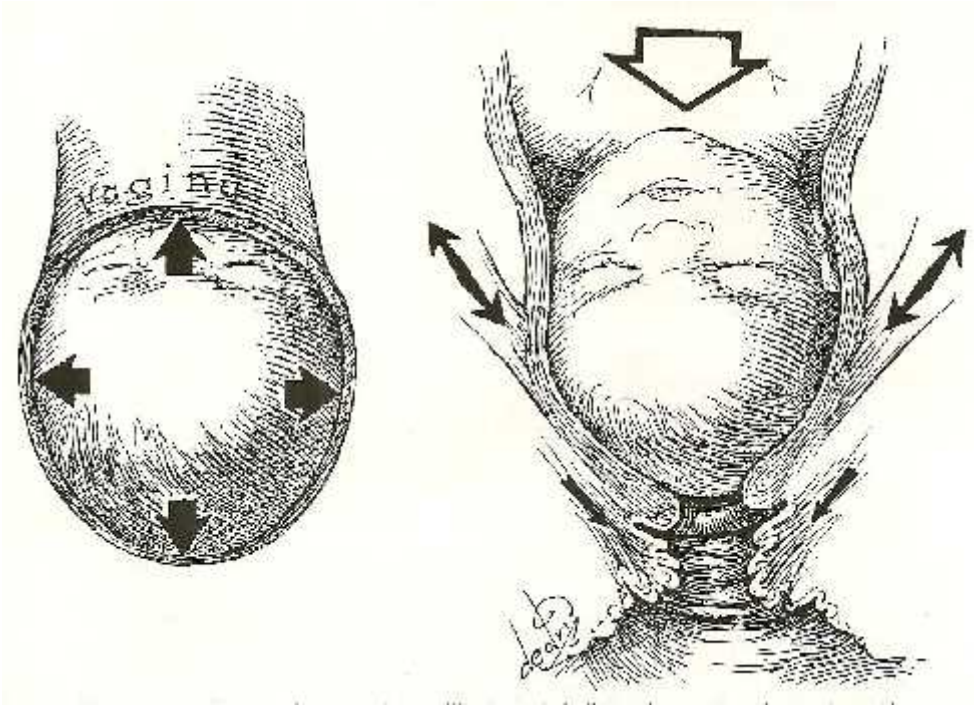


Figure 10: Distension cystocele and displacement cystocele etiology

Richardson explained as: (Figure 11)

Transverse defects: Represents separation of pubocervical fascia from its insertion around the cervix.

Midline defects: Represents the anterio-posterior separation between bladder and vagina.

Lateral defects: Represents the separation of arcus tendineus fascia pelvis from white line.

The vagina provides a potential space for both anterior and posterior vaginal prolapse.

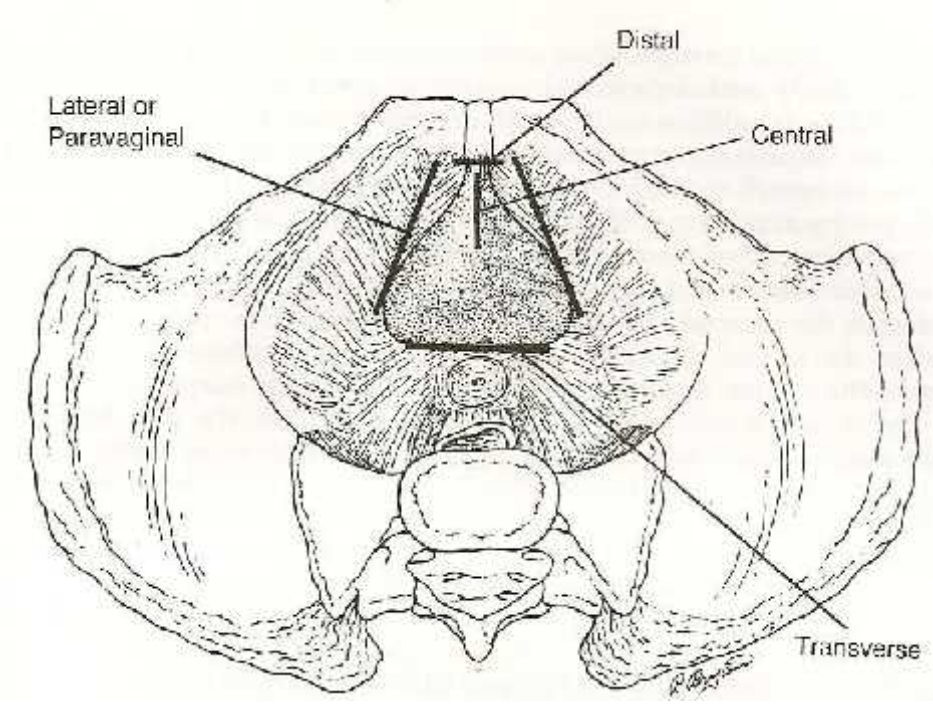


Figure 11: Location of pubocervical fascia defects that contribute to anterior vaginal wall prolapse.

The fascia of bladder and anterior vaginal wall fuses to form the pubocervical fascia. At the level of the proximal urethra and bladder neck (Periurethral fascia), and at the level of the bladder and fascial ring of the cervix (Supravaginal septum) are densely adherent to the vagina. But along the base of bladder, the vesicovaginal space can be developed between these two fascial planes.

The pubocervical fascia prevents herniation of the bladder and urethra into the vagina.

Herniation of the bladder base or urethra may indicate additional pubourethral ligaments and pubourethralis muscle deficiency.

The urethra is supported by pubourethral ligaments which are condensations of endopelvic fascia composed of collagen smooth muscle and cholinergic nerve fibers. The main support is form the posterior pubourethral ligaments which extend from the inferior portion of the OS pubis and attaches to the mid urethra and they prevent downward and posterior rotational displacements.[16]

The hammock of levator ani muscle is covered by endopelvic fascia (Pelvic diaphragm). It supports the bladder and urethra in their intra abdominal position. The pubourethralis, a division of this muscle, forms a sling around the proximal urethra as it passes through the pelvic diaphragm and aids in preventing posterior displacement of the proximal urethra and bladder neck. The urogenital diaphragm (perineal musculature and fascia) provides additional inferior support, especially during coughing and straining.[12,16]

The urethral axis and the trigone form an angle of less than 100^0 (Urethrotrigonal angle) and the urethral axis to vertical does not exceed $30^{0.}$ (Figure 12)

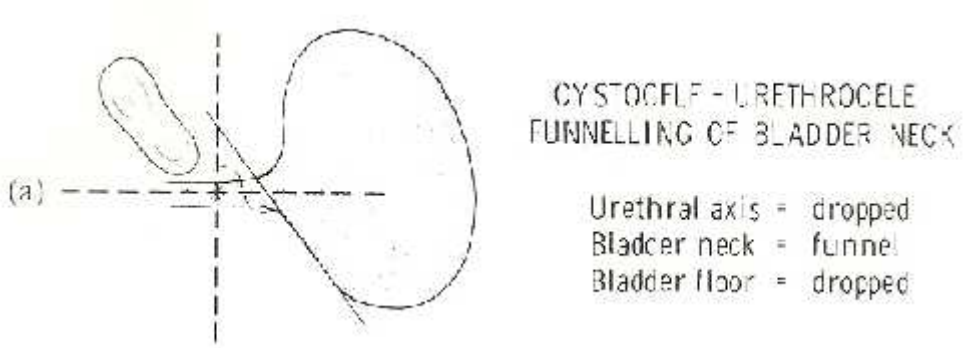


Figure 12: Cystocele – Urethrocele funneling of bladder neck

With straining the normal bladder base remains horizontal and may descends up to 1.5cm but not below the pubic symphysis. The urethra maintains its position so that the bladder neck appears as valve or kink and remains the first level of continence.[12]

The bladder neck and proximal urethra are maintained in an intra abdominal position and the mid urethra is supported within the pelvic floor.

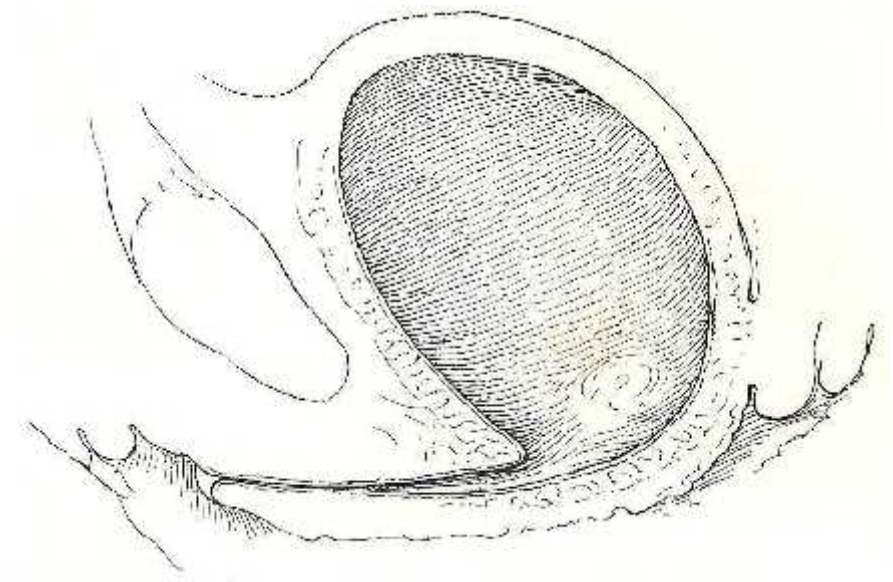


Figure 13: Rotational descent of the vesicourethral junction and funneling of the urethra are both present

An increased intra abdominal pressure is transmitted to the bladder and proximal urethra. The fixation of the bladder neck provides increased efficiency of the valvular mechanism, as urethrotrigonal angle remains less than 90 degrees. The pelvic floor undergoes reflex contraction and acts as a back board, providing a stable surface for urethral compression.[12]

The loss of anatomical support allows downwards and posterior rotational movements of the bladder neck and posterior urethra from their intra abdominal position. It prevents transmission of the raised pressure to the bladder base and continence area and places the urethra in a dependent position with respect to the bladder base. (Fig.13) The tensile forces of the full bladder open the bladder neck, and permit urine flow in to the proximal urethra. Structurally this motion is associated with loss of kinking mechanism. This explains stress urinary incontinence in prolapse patients.[12]

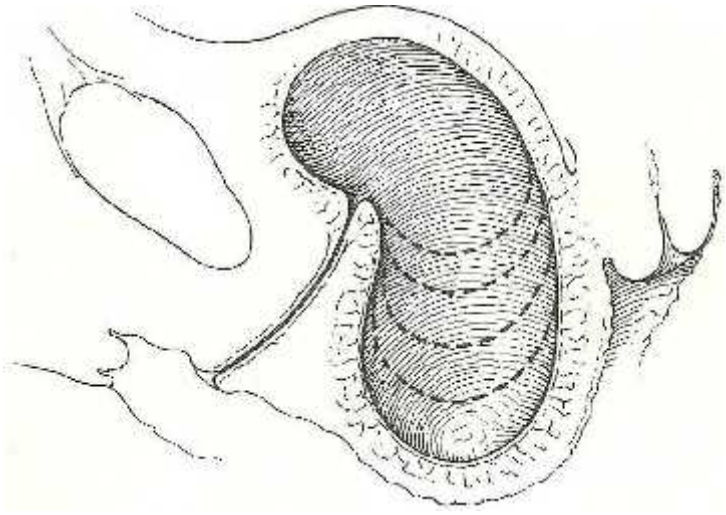


Figure 14: Varying degrees of posterior distention type cystocele are shown by the broken lines

The dislocation of the uterus and vagina as happens with prolapse, must affect the bladder and urethra because of the anatomical closeness of internal female genital organs to the pelvic urinary system.[12]

Hypotonia of the bladder may result, when a large cystocele permits chronic over distension of the bladder. A persistent amount of residual urine may provide the basis for over flow incontinence (Figure 14).[16]

This urine may also become infected from stagnation with chronic cystitis. Decreased blood flow to the bladder secondary to chronic over distension may also cause cystitis because of inflammation.[16]

Digital elevation of the bladder may be required to void in a patient with massive cystocele.

Dislocation of the urethrorvesical junction is usually coincident with massive eversion of the vagina because it stretches the pubourethral ligament portion of the urogenital diaphragm.[12]

Urethral or ureteral kining may occur, when they are anatomically displaced outside the pelvis,. The ureter obstruction of vary degree that there may be demonstrable hydronephrosis, oligouria and in advanced cases, anuria.

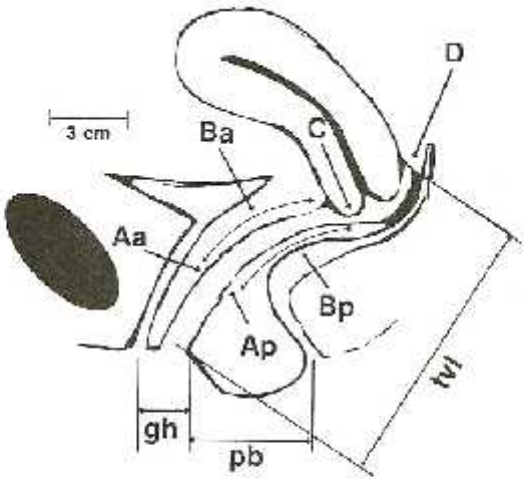
Vaginal lymphangiectasis coincident with massive prolapse may follow disturbances in circulation and produce chronic edema to cause another source of extra pelvic urethral compression.[12,16]

Baden walker Cystocele classification

- Grade 1 Cystocele: Bladder descent towards the introitus with strain
- Grade 2 Cystocele: Bladder up to introitus with strain
- Grade 3 Cystocele: bladder outside of introitus with strain
- Grade 4 Cystocele: Bladder outside of the introitus at rest

POP-Q STAGING[17]

Stage 0	No prolapse is demonstrated. Points Aa, Ap, Ba, and Bp are at -3cm and either point C or D is between –TVL (total vaginal length) cm and – (TVL -2)cm (i.e., the quantification value for point C or D is ≤ -(TVL -2)cm) represents stage 0.
Stage I	The criteria for stage 0 are not met, but the most distal portion of prolapse is > 1 cm above the level of the hymen (i.e., it quantification value is <-1cm)
Stage II	The most distal portion of prolapse is ≤ 1 cm proximal to or distal to the plane of the hymen (i.e., it's quantification value is ≥ -1 cm but ≤ +1 cm)
Stage III	The most distal portion of the prolapse is >1cm below the plane of the hymen but protrudes no further than 2cm less than total vaginal length in centimeters (i.e., its quantification value is > + 1cm but < + [TVL -2] cm)
Stage IV	Essentially, complete eversion of the total length of the lower genital tract is demonstrated. The distal portion of the prolapse protrudes to at least (TVL -2) cm (i.e., its quantification value is ≥ + [TVL -2] cm). In most instances, the leading edge of stage IV prolapse will be the cervix or vagina cuff scar.



Landmarks for quantitative pelvic examination. Aa – point A anterior; Ap = point A posterior; Ba – point B anterior; Bp point B posterior; C – cervix or vaginal cuff; D = posterior fornix (if cervix is present); gh = genital hiatus; pb – perineal body; tvl – total vaginal length (*From* Bump RC, Mattiasson A, Bø K, et al: The standardization of terminology of female pelvic floor dysfunction. Am J Obstet Gynecol 175:10–17, 1996; with permission.)

Figure 15: POP Q points

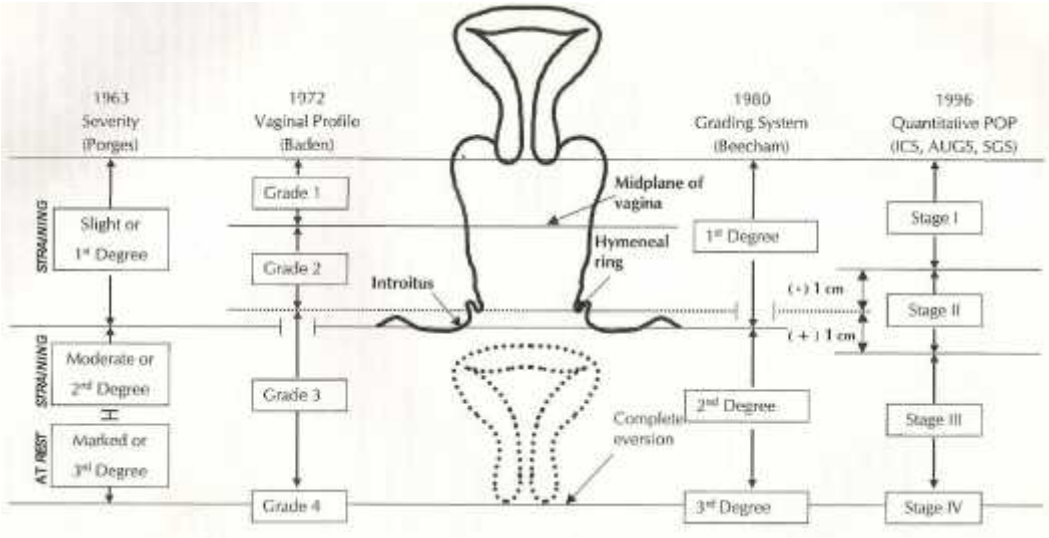


Figure 16: Comparison of ordinal classifications of pelvic organ prolapse

Baden Walker Halfway System for evaluation on Physical examination

- Grade 0: Normal position for each respective site
- Grade 1: Descent halfway to the hymen
- Grade 2: Descent to the hymen
- Grade 3: Descent halfway past the hymen
- Grade 4: maximum possible descent for each site

BLADDER DYSFUNCTION SYMPTOM

Bladder has two main functions: to store urine and then to empty it completely in socially acceptable time and place.

Bladder is involuntary organ but still functions under voluntary control.

Symptoms are mainly divided into

1. Storage disorder
2. Voiding difficulty (Incomplete emptying)

Storage disorder

Failure to store urine leads to incontinence.

Urinary frequency is the number of times per day the patient voids. More than seven voids per day suggest problem of frequency. But it is highly dependent on the habitat and fluid intake.[1]

Nocturia is defined as awakening from sleep with need to urinate. If it is more than once, it is abnormal and referred as nocturia. And it increases with advancing age commonly.[1]

Urge incontinence is a urine loss accompanied by a powerful desire to urinate. It is associated with UTI as well as altered sensations of bladder.

Stress urinary incontinence refers to involuntary loss of urine in conditions of increased intra-abdominal pressures like coughing or sneezing. It can be because of urethral or bladder neck incompetence.[1]

Symptoms of storage disorder

1. Stress incontinence
2. Urge incontinence
3. Mixed incontinence
4. Frequency
5. Nocturia

Voiding difficulty (Incomplete emptying)

Voiding phase disorders have been classified by the International Continence Society as those involving abnormal detrusor or urethral function during micturition.[1]

Voiding dysfunction in women is most commonly associated with detrusor contractility. Detrusor underactivity is defined as a detrusor contraction of inadequate magnitude, duration, or both to effect bladder emptying within normal time span.[18]

Inadequate voiding associated with normal detrusor activity results from a functional and mechanical obstruction, functional in cases of detrusor sphincter dyssynergia and mechanical in cases of urethral stricture or advanced pelvic organ prolapse.

Hesitancy refers to trouble in starting the stream of urine. Straining to void, poor and intermittent flow can all reflect either urethral or detrusor dysfunction. Post micturition dribbling can be due to urethral diverticulum.[1]

Symptoms of abnormal bladder emptying:

1. Hesitancy
2. Straining to void
3. Incomplete emptying
4. Poor flow
5. Intermittent stream
6. Post micturition dribbling
7. Acute urinary retention

Post void residual urine is associated with retention of urine in bladder after completion of micturition. Elevated PVR can be associated with storage disorder like urge urinary incontinence. Overflow incontinence can be associated with raised PVR as there is retention of urine with overflow which may be acute or chronic. Acute is painful and due to acute genital infection. Chronic is usually in elderly and often as result of neuropathy of diabetes or cerebrovascular artherosclerosis.[18]

Frequency and urgency are associated with bladder hyperreflexia most commonly. These symptoms can be due to interstitial cystitis which is result of chronic retention.[18]

Voiding difficulty has two phases; asymptomatic and symptomatic. Symptoms of voiding difficulty like poor stream, straining to void, and incomplete emptying appear in symptomatic stage of bladder decompensation. The urine flow rate is reduced; and both maximum voiding pressure and PVR are elevated. The causes for voiding difficulty include neurological, pharmacological

(anticholinergic drugs), acute inflammation, obstruction, endocrinological, or due to over distention of bladder, acontractile or hypotonic detrusor.[18]

OBJECTIVES

The objectives of the present study were;

- To compare preoperative and postoperative post void residual urine volume in urogenital prolapse patients undergoing anterior colporrhaphy with vaginal hysterectomy.
- To know the prevalence of urinary symptoms in prolapse.
- To study the correlation between PVR, urinary symptoms and prolapse.

REVIEW OF LITERATURE

Post void residual urine volume (PVR) is the amount of residual urine in the bladder after a voluntary void and is a key marker for the evaluation of the efficacy of bladder empyting, particularly in women with pelvic organ prolapse and lower urinary tract dysfunction.

PVR measurement is recommended in women with genital prolapse, to evaluate potential stress incontinence, overt urinary incontinence and empyting phase dysfunction.

Threshold values delineating what constitutes an abnormal PVR are poorly defined. Different studies have referred an abnormal PVR values ranging from 30 ml to 100 ml.[4,19] Physiological bladder empting can be affected by age, fluid intake, medications, restriction and voiding habits.

Pelvic organ prolapse is the protrusion of vaginal apex or walls towards or beyond the introitus. The presence of prolapse is due to a deficient musculo fascial vaginal support mechanism. Etiological factors causing the prolapse include connective tissue deficiencies, birth trauma and neurologic dysfunction.

Lower urinary tract symptoms are commonly associated with prolapse. Neuromuscular injury or connective tissue failures of the anterior vaginal supports may results in deficiencies of the urethral sphincter mechanism, abnormal trigonal function and mechanical occlusion of the urethra.[20]

The women with significant pelvic organ prolapse, particularly of the anterior wall, may have impaired bladder emptying. It may be due to kink in normal urethral mechanism, which will lead to voiding dysfunction.[9]

Voiding dysfunction can result in various lower urinary tract symptoms, including storage symptoms like frequency, urgency, urge incontinence and emptying symptoms like poor stream, hesitancy, need to strain and a feeling of incomplete emptying.[21]

Post void residual urine volume is examined for voiding dysfunction. Elevated PVR is associated with poor detrusor muscle contractility or increased urethral resistance, and abnormal uroflowmetry.[9]

Studies have concluded that overactive urinary symptoms are associated with urinary retention in women. The symptoms of voiding difficulty like hesitancy, weak stream, need for straining, and sensation of incomplete bladder emptying are associated with increased PVR. And it is possible to identify the patients with urinary symptoms who are at risk of elevated PVR based on history and physical examination.

Lower urinary tract symptoms are common in the women with genital prolapse. Voiding difficulty, bladder outlet obstruction and stress incontinence may coexist and associated with prolapse. Detrusor instability and urethral hypermobility also correlate with the degree of prolapse.

Urethral hypermobility and symptoms of voiding difficulty were more common with grade 3 and grade 4cystocele. Urodynamic studies also revealed

bladder outlet obstruction in 58% patients with the cystocele of grade 3 and grade 4 prolapse.[2]

In the study done by Stanton to determine the reliability of symptoms associated with voiding difficulty and to determine their cause, the symptoms of voiding dysfunction found to be unreliable. This lack of correlation between symptoms and bladder function explains that the patient's interpretation of bladder emptying is unreliable and emphasizes the need for a more objective urodynamic assessment.[3]

In one study done by Lowenstein et al. to explore the relationship between obstructive urinary symptoms and PVR in women with pelvic floor disorders for determining the efficacy of screening questions for obstructive voiding dysfunction as a diagnostic tool for urinary retention. They found that the obstructive voiding symptoms have low sensitivity and specificity to detect the elevated PVR in pelvic floor disorders. And also found that patients with advanced prolapse are more likely to have retention of urine; 10% of stage III /IV prolapse compared to only 3% with stage II prolapse.

The lack of association between obstructive voiding symptoms and urinary retention may be attributed to the fact that both prolapse and detrusor hypo contractility are chronic changes that happen gradually and are likely to be associated with neuropathy.[22]

Lukacz et al. have also stated that elevated post void residual cannot be predicted based on symptoms alone, however greater degree of prolapse is associated with incomplete bladder emptying.[19]

In the study done by B.T.Hayden, presenting symptoms of urge incontinence and voiding difficulty were significantly linked to high PVR. There was a strong inverse relation of a high PVR to the diagnosis of urodynamic stress incontinence. Increasing age and higher grades of prolapse were associated with high PVR levels.[4]

In contrast, Megan o. Schimpf and O' Sullivan found that anterior vaginal wall prolapse of stage 2 or greater was not associated with urge incontinence or voiding dysfunction. And women without prolapse were more likely to report stress incontinence.[9]

Anterior colporrhaphy has been in use for more than 100 years and still perhaps the most commonly performed operation for genital prolapse. The advantages of anterior colporrhaphy are, it is a vaginal operation, it is quicker to perform, may cause less postoperative morbidity, and correct prolapse at the same time.

Stanton and Cardozo found no increase in urgency, frequency or voiding difficulty following the procedure. And anterior colporraphy with vaginal hysterectomy is satisfactory operation for correction of prolapse.[18]

METHODOLOGY

The present study was conducted in Department of Obstetrics and Gynaecology, KLES Dr Prabhakar Kore Hospital and Medical Research Center, Belgaum over a period of one year during October 2008 to September 2009.

Type of study

The present one year study was observational descriptive cross sectional study.

Study period

This study was conducted over a period of one year during October 2008 to September 2009.

Source of data

Patients admitted with urogenital prolapse at KLES Dr Prabhakar Kore Hospital and Medical Research Center, Belgaum.

Sample size

Present study was conducted on 65 patients with urogenital prolapase.

Sampling procedure

The sample size was calculated based on 80% of the average number patients attending at KLES Dr Prabhakar Kore Hospital and Medical Research Center, Belgaum over last three years with urigenital prolapse.

Selection criteria

Inclusion Criteria

- All the patients with urogenital prolapse.

Exclusion criteria

- Patients not willing to give informed consent.
- Patients not available for followup after operation.

Ethical Clearance

The ethical clearance was obtained from Review Board of Jawaharlal Nehru Medical College, Belgaum.

Procedure

Patients attending with genitourinary prolapse were explained about the nature of the study and written and informed consent was obtained (Annexure-I). Detailed history, general physical examination was done as per predesigned and pretested proforma (Annexure-II) and they were subjected to;

- History taking and filling of the preformed questionnaire for urinary complaints.
- Menstrual and Obstetric history.
- Physical examination which included POP-Q.
- Determination of PVR preoperative and postoperative.
- Urine routine and microscopy.

- Renal function test.

Physical examination

- Cough stress test.
- Grading for prolapse by: POP-Q, Baden walker halfway.
- Presence or absence of;
    - Vaginal rugosity.
    - Decubitous ulcer.
    - Cystocele.
    - Urethrocele.

Statistical analysis

The data obtained was tabulated and analysed using chi-square test and a p value of less than 0.05 was considered significant.

RESULTS

In this study, sixty five cases of different degrees of genitourinary prolapse were studied. The age of the patients varied from 25 years to 72 years with mean age of 50 years. 45 patients were postmenopausal and the mean menopausal age was 12 years.

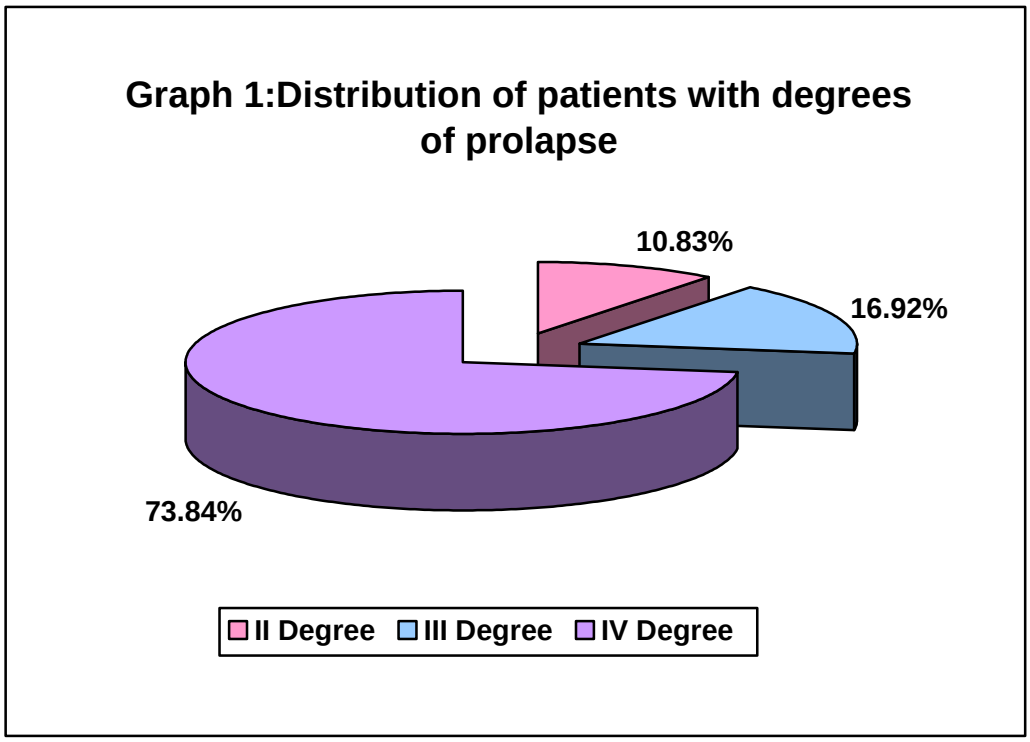
Table 1: Demographic features in relation to prolapse

Parameters	Years
Mean age	50.0 ± 14
Mean duration of prolapse	5.0 ± 6.5
Mean duration of menopause	12.0 ± 7.5

Duration of prolapse varied from 1 year to 30 years and degree of prolapse varied from 2nd degree prolapse to procedentia.

Table 2: Distribution of patients with degrees of prolapse

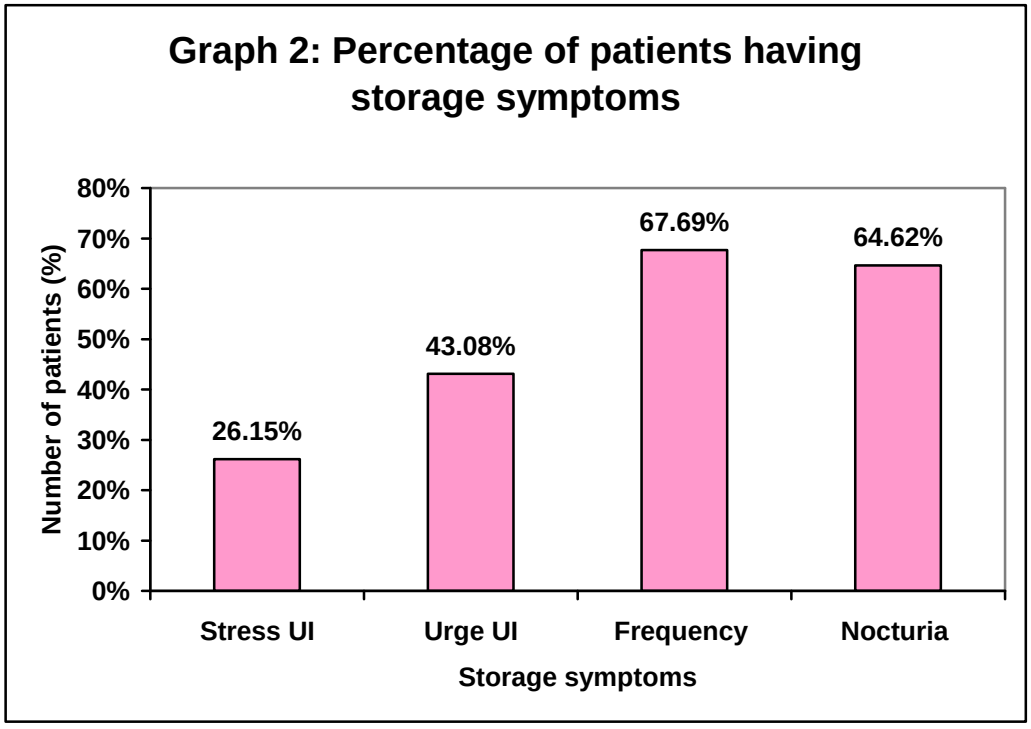
Degree of prolapse	Number of patients	Percentage
2nd Degree	06	10.83%
3rd Degree	11	16.92%
4th Degree	48	73.84%



Out of 65 cases, 11 were second degree, 48 were third degree and 6 were procedentia according to Baden Walker system (Graph 1). According to the POP-Q system, there was one patient with stage 1prolapse, nine patients with stage 2 prolapse, 28 patients with stage 3 prolpase and 27 patients were with stage 4 prolapse.

Table 3: Distribution of patients storage symptoms and its significance to degree of prolapse

Storage symptom	Frequency	Percentage	P value *
Stress incontinence	17	26%	0.212
Urge incontinence	28	43%	0.449
Increased frequency	44	68%	0.153
Nocturia	42	65%	0.250



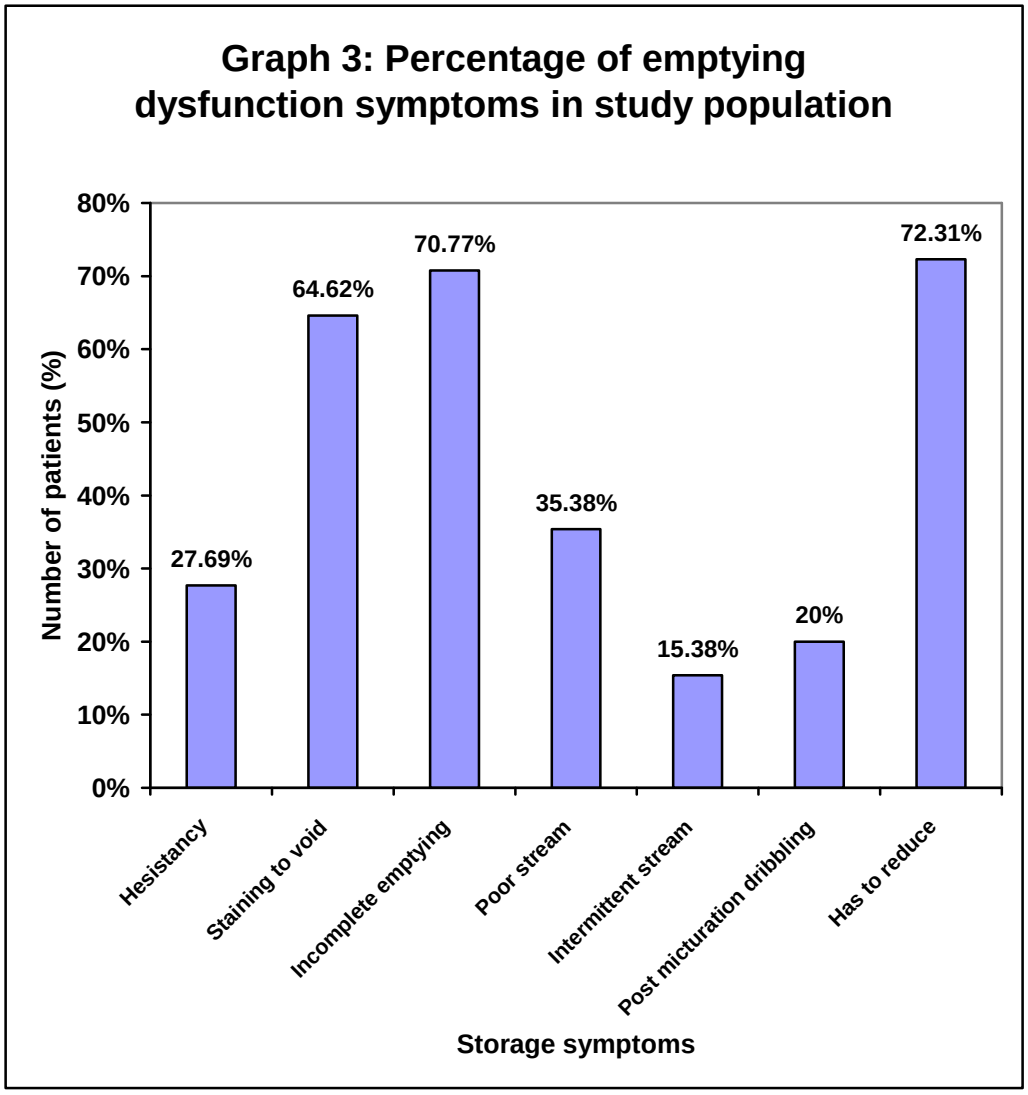
Storage symptoms were present in a large percentage of patients but they were not significantly associated with the degree of prolapse. Urge incontinence was complained by 43% of patients and stress incontinence was complained in

26% patients. 17 patients had complaints of stress urinary incontinence, but it was observed in only 9 patients (Graph 2).

Increased frequency of micturation and nocturia found in 68% and 65% patients, but they were not significantly associated with the degree of prolapse (Table 3).

Table 4: Percentage of emptying dysfunction symptoms in study population

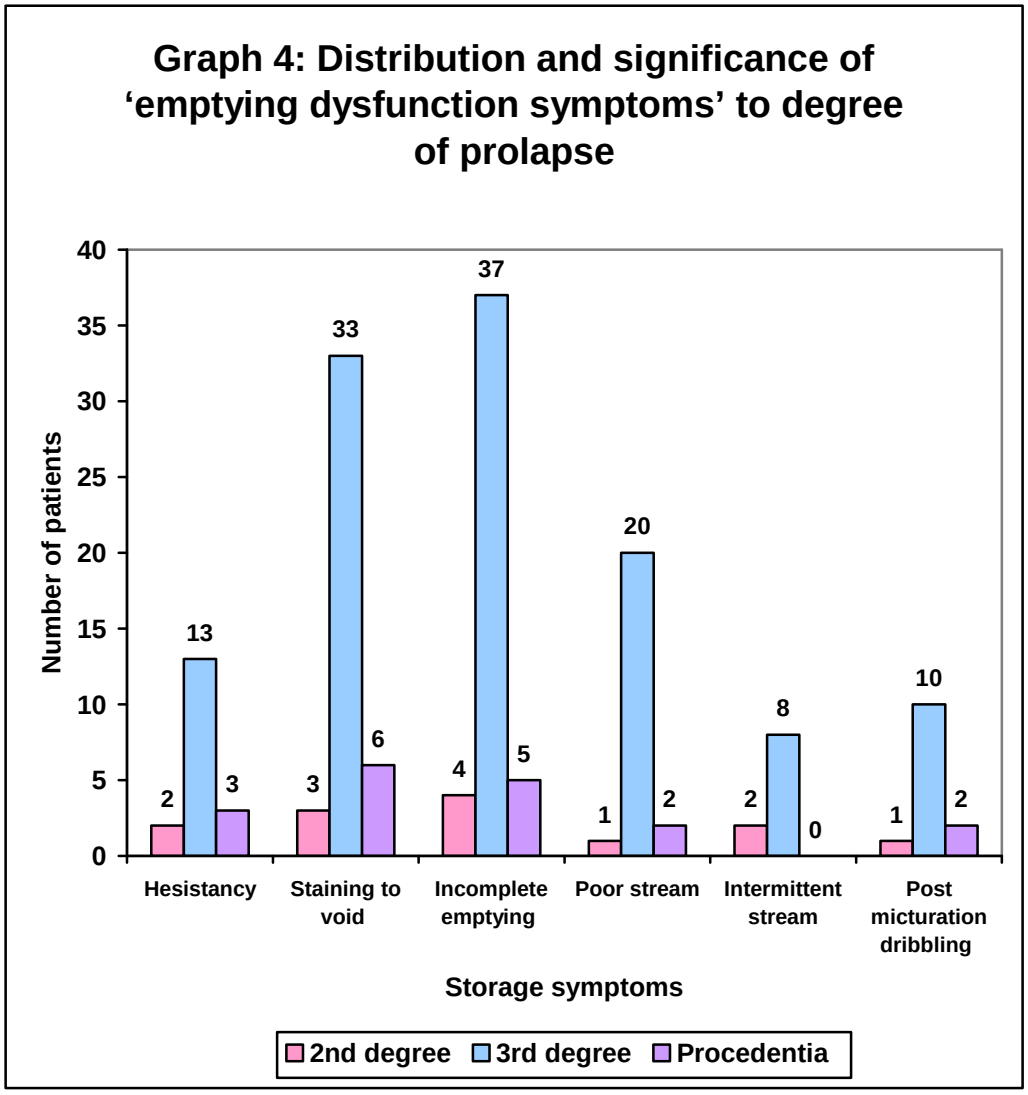
Emptying dysfunction symptoms	Number of Patients	Percentage
Hesitancy	18	27.69%
Straining to void	42	64.62%
Incomplete emptying	46	71.77%
Poor stream	23	35.38%
Intermittent stream	10	15.38%
Post micturation	13	20.00%
Has to reduce to void	47	72.31%



Hesitancy was present in 18 patients and it was not significantly associated with the degree of prolapse. Straining to void was reported by 42 patients, out of which 3 patients had second degree and 33 patients had third degree prolapse. All the six patients with procedentia had complaints of straining to void. P value was 0.006 which shows a significant relation with degree of prolapse (Table 5).

Table 5: Distribution and significance of 'emptying dysfunction symptoms' to degree of prolapse

Emptying dysfunction symptoms	Frequency	Degree of prolapase		Procedentia	'p' value
		2nd	3rd		
Hesitancy	18	2	13	3	0.368
Straining to void	42	3	33	6	0.006 (s)
Incomplete emptying	46	4	37	5	0.022 (s)
Poor stream	23	1	20	2	0.125
Intermittent stream	10	2	8	0	0.544
Post micturation	13	1	10	2	0.471
Has to reduce to void	47	2	39	6	<0.0001 (s)



Incomplete emptying was complained by 46 patients. Out of them, 4 patients had 2nd degree, 37 patients had 3rd degree prolapse and 5 patients had procedentia. It was also significantly related with the degree of prolapse. P value was 0.022.

Poor stream was present in 23 patients. Intermittent stream was present in 10 patients and 13 patients had complaints about post micturition dribbling without significant relation with the degree of prolapse.

47 patients complained about 'has to reduce prolapse to void' and it was significantly associated with the degree of prolapse. P value was <0.0001.

Out of 33 patients with PVR >50 ml, only 1 patient had POPQ stage 2 prolapse, 14 patients had POPQ stage 3 prolapse and 18 patients had POPQ stage 4 prolapse. P value was 0.024. (Table.6)

Table 6: POP-Q grades of prolapse in relation to PVR

POP Q Grade	PVR	
	≤ 50 ml	**> 50 ml**
1	01	00
2	08	01
3	14	14
4	09	18
Total	**32**	**33**

The number of patients with PVR >50 ml had increased with increase in the grade of Baden Walker prolapse, Out of six patients of procedentia, five patients had raised PVR. P value is 0.005.

In this study, increasing degree of prolapse by both the classifications has shown a statistically significant relation with Raised PVR.

Table 7: Association of age and raised PVR

Age groups	PVR		Total
	≤ 50 ml	**> 50 ml**	
A (25 – 34)	6	0	6
B (35 – 44)	8	5	13
C (45 – 54)	8	8	16
D (55 – 64)	9	10	19
E (65 – 74)	1	10	11
Total	32	33	65

Age has shown significant relation with the raised PVR > 50 ml. P value is 0.007 (s) (Table 7).

Increasing parity was not associated with an increased PVR, nor with the degree of prolapse. P value was 0.757.

The storage dysfunction symptoms were not associated with the raised PVR. But the emptying dysfunction symptoms like straining to void and has to reduce the prolapse to void, have shown significant association with raised PVR. The P values were 0.047 and 0.004 respectively. (Table 8)

Table 8: Association of urinary symptoms with raised PVR

Symptoms	Total	PVR		p value
		≤ 50 ml	> 50 ml	
Stress incontinence	17	9	8	0.722
Urge incontinence	28	13	15	0.694
Increased frequency	44	24	20	0.215
Nocturia	42	20	22	0.725
Hesistancy	18	9	9	0.939
Straining to voiding	42	18	24	0.047 (s)
Incomplete emptying	46	20.	26	0.165
Poor stream	23	11	12	0.867
Intermittent stream	10	3	7	0.186
Post micturation	13	5	8	0.385
Has to reduce prolapse	47	18	29	0.004 (s)

Seven patients had abnormal urine microscopy and on routine examination, Out of them, six patients had PVR > 50 ml. But association between UTI and elevated PVR was not statistically significant. P value was 0.050. Among four patients with deranged renal function, three patients had PVR > 50 ml. P value was 0.37 showing no significance.

33 patients had raised PVR before operation. After vaginal hysterectomy and anterior colporrhaphy, 32 patients had PVR within normal range. P value was 0.001 which is statistically significant. It shows that the procedure was effective in reducing the elevated PVR. Two patients had post operative urinary retention and only one patient had stress urinary continence.

DISCUSSION

In the present study, the voiding dysfunction was analyzed in relation with the genitourinary prolapse by measurement of post void residual urine volume. The effectiveness of vaginal hysterectomy with anterior colporrhaphy in reducing raised PVR is also discussed.

Other studies have used various reference values for defining elevated PVR ranging from 30 ml to 100 ml.[4,19] In this study upper limit of normal PVR was 50 ml.

Catheterization is used for determination of PVR as it is a gold standard and gives most accurate readings.[19,24]

The prevalence of elevated PVR more than 100 ml was 10%. The elevated PVR was associated with increasing age and greater degrees of prolapse. (Table 4 and 5) This finding is consistent with the study done by Lukacz and Fitzgerald et al.[19,23]

Sr. No	Author	Year	Sample size	Prevalence of raised PVR
1.	Lukacz[19]	2004	1399	11%
2.	Haylen[4]	2008	1140	6%
3.	Fitzgerald[19]	2001	393	9%
4.	Present study	2009	65	11%

The diagnosis of the prolapse has a significant positive relationship with high PVR. The proposed mechanism of genital prolapse is the distortional or kinking effect on the urethra to create bladder outflow obstruction.[4]

In the present study, mean age of the patients admitted with prolapse was 50 years. In the studies done by Bradley and Haylen, the mean age of prolapse patients was 68 and 58 years respectively.[4,25] Compared to these studies, the present study had a young study population (Table 1 and 7).

This study shows that increasing parity is not associated with urinary retention and elevated PVR. Similar results were found in study done by Lukacz and Lowenstein.[19, 22]

In this study, stress urinary incontinence and overactive bladder symptoms like urge incontinence, frequency, nocturia were not associated with the increasing grades of prolapse (Table 3).

Various obstructive urinary symptoms like straining to void (p=0.006), incomplete emptying (p=0.022) and has to reduce prolapse to void (p=0.0001) were associated with the increasing grades of prolapse or vaginal descent (Graph 2 and 4; Table 3 and 5).

G Alessandro Digesu et al also found a poor correlation between prolapse and storage urinary symptoms. But symptoms like 'feeling of incomplete bladder emptying' and the 'need of straining during micturation' were associated with prolapse.[26] In contrast to these findings other study has shown that occult stress

incontinence, detrusor instability and urethral hypermobility were associated with prolapse.[2] These conditions are storage disorders.

In this study, PVR was used for the determination of voiding dysfunction. Elevated PVR cannot be predicted based on symptoms alone; however, prolapse beyond the hymen may help identify women with incomplete bladder emptying.[19]

The present study supports the lack of association between raised PVR and storage disorder symptoms; however the emptying dysfunction symptoms like 'straining to void' and 'has to reduce prolapse to void' have shown significant association with raised PVR ($p=0.047$ and 0.004 respectively; Table 6).

In study done by Fitzgerald et al, symptoms of voiding difficulty were found significantly related with elevated PVR.[23]

The poor predictive value of obstructive voiding symptoms in diagnosis of raised PVR was reported by Al -Shahrani M and Lovatsis in 2005. They have shown that the poor relationship between raised PVR and the symptoms of incomplete emptying, poor flow and straining to void.[27]

The present study shows significant reduction in elevated PVR by the anterior colporrhaphy and vaginal hysterectomy ($p< 0.0001$). This suggests that the anterior colporrhaphy with vaginal hysterectomy is an effective procedure for the reduction of elevated PVR.

In the study done by Stanton et al also shown that symptoms of urge incontinence, stress incontinence and prolapse were significantly reduced after

anterior colporrhaphy and vaginal hysterectomy. The surgery has improved the urodynamic findings by correcting a large cystocele and cystourethrocele which impede the urinary flow.[28]

It is commonly considered that retention of urine will be associated with an increase in risk of urinary tract infection.[6,29] In this study, 11% of patients were diagnosed to have an abnormal urine microscopic examination. But it was not significantly related to elevated PVR or increase in degree of prolapse ($p=0.050$).

The levels of blood urea and creatinine were evaluated for assessing the renal function. In cases of major degree of prolapse, because of the kinks in the ureter, back pressure would cause hydroureter and hydronephrosis.[18,30] In the present study, only 4% of patients had elevated levels of blood urea or serum creatinine.

Strengths of this study include its prospective design and use of direct catherization to determine PVR which is the most reliable method.

Although the study assessed the symptoms associated with progression of vaginal descent, it has not measured the level of discomfort and level of patient satisfaction after the surgery.

CONCLUSION

Post void residual urine output was measured for the detection of bladder dysfunction in prolapse patients. Raised PVR has shown significant association with increasing degree of prolapse. Obstructive urinary symptoms like straining to void, incomplete emptying and has to reduce prolapse to void were associated with raised PVR.

Storage symptoms like stress urinary incontinence, urge incontinence, frequency and nocturia were commonly present in study population. But there was no association of these storage symptoms with increasing degree of prolapse and raised PVR.

Emptying dysfunction symptoms like ‘straining to void’ and ‘has to reduce prolapse to void’ were associated with raised PVR and increasing degree of prolapse.

Vaginal hysterectomy with anterior colporrhaphy was found to be an effective procedure for the reduction of raised PVR in prolapse patients. PVR evaluation and detailed history about urinary tract symptoms is necessary for the preoperative and postoperative care of patient.

SUMMARY

In anterior vaginal wall prolapse, bladder dysfunction is expected due to anatomical proximity of the anterior vaginal wall with the bladder neck and urethra.

In this study, sixty five patients of different degrees of prolapse were examined for bladder dysfunction. Post void residual urine volume was measured before the vaginal hysterectomy with anterior colporrhaphy and four days thereafter. And it was used to find the effectiveness of the procedure and the relation of raised PVR with different bladder symptoms.

Catheterization was used to measure post void residual urine to achieve substantially accurate results. History of bladder symptoms was taken along with physical examination.

The raised PVR was associated with increasing degree of prolapse and the emptying dysfunction symptoms. Increased age was also significantly associated with raised PVR. The vaginal hysterectomy with anterior colporrhaphy was found to be effective procedure in reducing elevated PVR.

BIBLIOGRAPHY

1. Rock JA, Howard JW. TeLinde's Operative Gynecology. 9th ed. Philadelphia: Lippincott Williams and Wilkins; 2003.

2. Romanzi LJ, Chaikin DC, Blaivas JG. The effect of genital prolapse on voiding. J Urol 1999; 161: 581-6.

3. Stanton SL, Cavit O, Paul H. Voiding difficulties in the female: Prevalence, Clinical and urodynamic review. Obstet Gynecol 1983; 61: 144-7.

4. Haylen BT, Lee J, Logan V, Husselbee S, Zhou J, Law M. Immediate postvoid residual volumes in women with symptoms of pelvic floor dysfunction. Obstet Gynecol 2008; 111: 1305-12.

5. Haylen BT. Resudual urine volumes in a normal female population: Application of transvaginal ultrasound. Br J Urol 1989; 64: 347-49.

6. Gehrich A, Stany MP, Fisher JR et al. Establishing a mean post void residual volume in asymptomatic perimenopausal and postmenopausal women. Obstet Gynecol 2007; 110: 827-32.

7. Costantini E, Mearini E, Pajoncini C, Biscotto S, Bini V, Porena M. Uroflowmetry in female voiding disturbances. Neuro Urol Urodyn 2003; 22: 569-73.

8. Dwyer PL, Desmedt E. Impaired bladder emptying in women. Aust NZJ Obstet Gynaecol 1994; 34: 73-8.

9. Megan SO, Sullivan DM, LaSala CA, Tulikangas PK. et al. Anterior vaginal wall prolapse and voiding dysfunction in urogynecology patients. Int Urogynecol J Pelvic Floor Dysfunct 2007; 18(7): 721-5.

10. Delancey JOL. Anatomy In: Stanton SL, Monga AK (Eds). Spain: Clinical urogynaecology. 2nd ed. Churchill Livingstone; 3-19.

11. Gosling JA. Structure of the lower urinary tract and pelvic floor. Clin Obstet Gynecol 1985; 12: 285-94.

12. Staskin DR, Zimmern PE, Hadley HR, Raz S. Pathophysiology of stress incontinence. Clin Obstet Gynaecol 1985; 12(2): 357-67.

13. Sullivan MP, Yalla SV. Physiology of female micturition. Urol Clin N Am 2002; 29: 499-514.

14. Julian P, Shah R, Dasgupta P. Voiding difficulties and retention. In: Stanton SL, Monga AK. Clinical urogynaecology. 2nd ed. Spain: Churchill Livingstone; 259-72.

15. Nichols DH, Randall CL. Vaginal Surgery. 3rd ed. Baltimore: Williams and Wilkins; 1989.

16. Blaivas JG. Pathology of lower urinary tract dysfunction. Clin Obstet Gynecol 1985; 12: 295-309.

17. Bump RC, Mattiasson A, Bo K, Brubaker LP, DeLancey JO, Klarskov P, et al. The standardization of terminology of female pelvic organ prolapse and pelvic organ dysfunction. Am. J Obstet Gynecol 1996; 175:10-17.

18. Stanton SL. Clinical Gynecologic Urology. St. Louis: The C.V. Mosby Company; 1984.

19. Lukacz ES, DuHamel E, Menefee SA, Luber KM. Elevated postvoid residual in women with pelvic floor disorders: Prevalence and associated risk factors. Int Urologynecol J 2007; 18: 397-400.

20. Smith PP, Appell RA. Pelvic organ prolapse and the lower urinary tract: The relationship of vaginal prolapse to stress urinary incontinence. Curr Urol Reports 2005; 6: 340-7.

21. Carlson KV, Rome S, Nitti VW. Dysfunctional voiding in women. J Urol 2001; 165: 43-8.

22. Lowenstein L, Anderson C, Kenton K, Dooley Y, Brubaker L. Obstructive voiding symptoms are not predictive of elevated post residual urine volumes. Int Urogynecol J 2008; 19: 801-4.

23. Fitzgerald MP, Jaffer J, Brubaker L. Risk factors for an elevated postvoid residual urine volume in women with symptoms of urinary urgency, frequency and urge incontinence. Int Urogynecol J Pelvic Floor Dysfunct 2001; 12(4): 237-9.

24. Haylen BT, Frazer MI, Sutherst JR, Ashby D. The accuracy of measurement of residual urine in women by urethral catheterization. Br J Urol 1989; 63: 152-4.

25. Bradley CS, Zimmerman MB, Wang Q, Nygaard IE. Vaginal descent and pelvic floor symptoms in postmenopausal women: a longitudinal study. Obstet Gynecol 2008; 111: 1148-53.

26. Digesu GA, Chaliha C, Salvatore S, Hutchings A, Khullar V. The relationship of vaginal prolapse severity to symptoms and quality of life. BJOG 2005; 112 (7): 971-6.

27. Al Shahrani M, Lovalsis D. Do subjective symptoms of obstructive voiding correlate with post void residual urine volume in women? Int Urogynecol J Pelvic Floor Dysfunct 2005; 16: 12-4.

28. Stanton SL, Hilton P, Norton C, Cardozo L. Clinical and Urodynamic effects of anterior colporrhaphy and vaginal hysterectomy for prolapse with and without incontinence. BJOG 1982; 89: 459-63.

29. Agarwal S, Verma M. Urinary tract changes in cases of genital prolapse. J Obstet Gynaecol Ind 1978; 28: 1066.

30. Churchill DN, Afridi S, Dow D, McManaman P. Uterine prolapse and renal dysfunction. J Urol 1980; 124: 899.

ANNEXURE I

CONSENT FORM

TITLE: Evaluation of post void residual volume in urogenital prolapse.

Urogenital prolapse is common in our old age population of women. It usually develops due to fault in support of uterus. It also involves urinary problems in some patients, which are neglected by them knowingly or unknowingly. The patients with difficulty in passing urine may have poor quality of life or may be totally asymptomatic.

In this study to analyse the magnitude of urinary problem and it's correction by the surgery undergone, you will be examined physically and the quantity of urine remaining in the bladder after you pass urine will be measured by ultrasonography and you will not be charged for the same.

You are invited to participate in this research study being conducted under the guidance of Dr B R Nilgar, Professor, Department of Obstetrics and Gynaecology, J. N. Medical College, Belgaum.

Your participation in the study is voluntary. As it is observational study, no risk is involved to participants. You are free to deny participation. This study may not benefit you, however may be beneficial to the other patients. Standard care will be provided even if you refuse to participate in this study.

Every effort will made to protect the confidentiality of the information you provide. You will be given an ID number and the same will be used for study purpose preventing your identification.

Results of the study may be published for scientific purposes but you will not be identified.

You will not be paid anything for participating in the study.

If you have any questions about the study, you may please call, visit or write to

Dr Nitu Kadam, Post Graduate (Obstetrics and Gynaecology), Telephone Number 9986746108 or Dr B. R. Nilgar, Professor, Department of Obstetrics and Gynaecology, J. N. Medical College, Belgaum – 590 010. Telephone Number 9448126613. If you have any questions regarding your rights as study participant, you may contact Dr. V. D. Patil, Principal and Chairman of Ethical Committee, Jawaharlal Nehru Medical College, Belgaum.

Statement of consent:

I volunteer and consent to participate in this study. I have read the consent document or it has been read to me in my vernacular language. I accept to participate in this study. All the information regarding this study has been provided to me and I have understood the same. I have been given an opportunity to ask questions and obtain appropriate answers.

Signature or left thumb print of the participant or legally authorized representative

Participant's Name : _____

Signature or left thumb print : _____

Address : _____

Telephone No. : _____

Witness's Name : _____

Signature or left thumb print : _____

Investigator's Name : _____

Signature : _____

Date: | | | | | | | | |

ANNEXURE II

PROFOMA

Patient no:

1. Name of patient:
2. Age
3. Inpatient no:
4. Phone No:
5. Address:
6. **History of prolapse:**

   Since

   Associated with backache

   WDPV

   Post coital bleeding

   Constipation

   Cough for long time

   History of drug affecting voiding function
7. Menstrual history:

   Menopause since, OR If menstruating LMP
8. Obstetric history:

   Para
   How many vaginal deliveries, or cesarean section
   First delivery at home or hospital
   If any difficulty in delivery, in terms of prolonged labor or instrumental delivery, h/o premature bearing down.

9. **History of lower urinary tract symptoms:**

   Storage disorder symptoms

   Stress incontinence

   Urge incontinence

   Mixed incontinence

   Increased frequency

   Nocturia

   Nocturnal enuresis

10. **Abnormal emptying symptoms:**

    Hesitancy

    Straining to void

    Incomplete emptying

    Poor flow

    Intermittent stream

    Post micturation dribbling

    Acute urinary retention

11. **Abnormal bladder contents:**

    Abnormal colour

    Abnormal smell

    Hematuria

    Pneumaturia

    Stones

    Foreign bodies

    Miscellaneous

12. **examination:**

    Cough stress test

    Grading for prolapse

- POP-Q
- Baden-walker halfway system
- Vaginal rugae
- Decubital ulcer

Urethrocele

Cystocele

13. **Investigations:**

- Post Void Residual urine volume before operation

- Post Void Residual urine volume after operation on fifth POD

- Post Void Residual urine volume after three months POD

(PVR measured by ultrasonography)

Fasting blood sugar:

Urine Routine:

Sugar

Albumine

Urine Microscopy:

Pus cells

Epithelial cells

Casts

Blood Urea:

Serum Creatinine

ANNEXURE III - MASTER CHART

KEY TO MASTER CHART

A	-	Absent
N	-	No
P	-	Present
POP-Q	-	Pelvic organ prolapse quantification
Post OP	-	Post operative
Pre OP	-	Preoperative
Sr. No.	-	Serial Number
UTI	-	Urinary tract infection
Y	-	Yes

Sl. No.	Age (Years)	ProlapseSince	Menopause	Stress Urinary Incotinance	Urge Incotinance	Increase Frequency	Nocturia	Hesitancy	Straining to Void	Incomplete Emptying	Poor Stream	Intermitant Stream	Post Micturation Dribbing	Need to reduce	Cough Stress Test	Vaginal Rugosity	Decubitous Ulcer	Cystocele	Degree Of Prolapse	POP Q	Parity	Home Delivery	Difficult Delivery	UTI	Renal Function	Pre OP	Post OP
1	55	20	6	Y	Y	Y	N	Y	Y	N	Y	N	N	Y	N	A	A	Y	4	4	4	Y	Y	N	A	42	8
2	59	1	6	N	N	Y	Y	N	Y	Y	Y	N	N	Y	N	P	P	Y	3	3	4	N	N	N	N	46	10
3	30	2	-	Y	N	Y	Y	N	N	N	N	N	N	N	Y	P	A	N	2	2	2	Y	N	N	N	50	20
4	45	2	-	N	N	N	N	N	N	N	N	N	N	Y	N	P	A	Y	3	4	3	Y	N	A	N	92	15
5	70	25	30	N	N	Y	Y	Y	Y	Y	N	N	Y	Y	N	A	P	Y	3	3	13	Y	N	N	A	106	18
6	50	4	2	N	N	Y	Y	N	Y	Y	Y	N	N	Y	N	P	P	Y	3	4	3	N	N	N	N	24	30
7	62	1	12	N	N	N	Y	N	N	N	N	N	N	N	N	A	A	Y	2	3	2	Y	N	N	N	33	12
8	65	3	20	N	Y	Y	Y	N	N	Y	Y	N	N	Y	N	A	A	Y	3	3	4	Y	N	A	N	56	15
9	54	4	5	N	N	N	N	Y	Y	Y	Y	N	N	N	N	A	P	Y	3	4	2	N	N	N	N	68	5
10	55	8	10	N	N	N	Y	N	Y	Y	Y	N	Y	Y	N	A	P	Y	3	3	5	Y	N	N	N	36	10
11	35	2	-	N	N	Y	Y	N	N	N	N	N	N	N	N	P	A	Y	3	3	2	Y	Y	N	N	12	20
12	40	1	-	Y	N	Y	N	N	N	Y	Y	N	N	Y	Y	P	A	Y	3	3	4	Y	N	N	N	44	0
13	40	18	-	N	Y	Y	Y	N	Y	Y	Y	N	Y	Y	N	P	P	Y	3	3	2	Y	Y	N	N	10	18
14	45	2	-	Y	N	Y	Y	N	Y	Y	Y	N	N	Y	N	P	P	Y	3	3	3	N	Y	N	N	62	25
15	60	1	16	Y	N	Y	Y	Y	Y	Y	N	N	Y	Y	Y	A	P	Y	4	3	4	Y	N	N	N	73	15
16	45	6	-	N	N	Y	Y	Y	Y	Y	N	N	N	Y	N	P	A	Y	3	3	3	Y	N	N	N	10	22
17	67	2	10	N	Y	Y	Y	Y	Y	Y	N	N	N	N	N	A	A	Y	3	4	8	Y	N	N	N	38	10
18	42	2	-	Y	Y	Y	Y	N	N	Y	N	N	N	N	Y	P	A	Y	3	3	1	N	Y	N	A	70	25
19	60	2	20	Y	N	N	Y	N	Y	Y	N	N	N	Y	Y	A	P	Y	3	4	5	Y	N	A	N	75	25
20	35	2	-	Y	Y	Y	Y	N	N	Y	N	N	N	N	Y	P	A	Y	3	4	3	Y	Y	N	N	40	20
21	66	1	20	N	Y	Y	Y	Y	Y	Y	N	N	N	Y	N	A	P	Y	4	4	6	Y	N	A	N	114	8
22	55	10	10	Y	N	Y	Y	Y	Y	Y	N	N	N	Y	N	A	A	Y	3	4	6	Y	N	N	N	162	5
23	55	30	20	N	Y	Y	Y	Y	N	Y	N	N	N	Y	N	A	A	Y	3	4	9	Y	Y	N	N	44	16
24	45	5	-	Y	N	Y	Y	N	N	Y	N	N	N	N	N	A	P	Y	3	4	1	N	N	N	N	68	12
25	72	3	12	N	N	Y	Y	N	Y	Y	N	N	N	Y	N	P	A	Y	3	4	4	Y	N	N	N	65	2
26	65	2	15	N	Y	Y	Y	N	N	Y	Y	Y	Y	Y	N	P	P	Y	3	4	5	N	Y	N	N	58	10
27	56	1	6	N	N	Y	Y	N	N	N	Y	N	N	Y	N	P	A	Y	3	3	3	N	N	N	N	60	30
28	30	5	-	N	N	N	N	N	Y	N	N	N	N	N	N	P	A	Y	2	2	4	Y	N	N	N	30	10
29	60	3	10	N	N	N	N	N	Y	Y	Y	N	N	Y	N	A	A	Y	3	4	3	Y	N	N	N	75	10
30	61	1	20	Y	N	N	N	N	N	N	N	N	N	N	N	P	P	Y	2	2	1	Y	N	N	N	40	30
31	50	1	10	N	N	Y	Y	N	Y	Y	N	N	N	Y	N	P	P	Y	3	3	4	N	N	N	N	24	15
32	30	1	-	N	Y	N	N	N	Y	N	N	N	N	Y	N	P	A	Y	3	3	3	Y	N	N	N	15	8
33	70	10	25	N	N	N	N	N	Y	Y	N	N	N	Y	N	A	A	Y	3	3	7	Y	N	N	N	150	40
34	65	2	10	N	Y	Y	N	N	Y	Y	N	N	N	Y	N	A	P	Y	3	3	2	N	N	N	N	70	15
35	55	3	8	N	Y	Y	N	N	Y	Y	Y	Y	N	Y	N	A	A	Y	3	4	3	Y	Y	N	N	90	25
36	55	1	14	N	Y	N	Y	N	Y	Y	N	N	N	Y	N	A	A	Y	3	4	5	Y	N	N	N	220	40
37	46	2	-	N	N	N	N	Y	Y	N	Y	Y	N	Y	N	A	A	Y	3	3	4	Y	N	N	N	90	25
38	32	2	-	N	N	Y	N	Y	Y	Y	N	Y	Y	N	N	A	P	Y	3	4	3	Y	Y	N	N	46	20
39	35	6	-	N	N	N	N	N	Y	Y	N	N	N	Y	N	A	P	Y	3	3	3	Y	N	N	N	60	30
40	45	2	6	N	Y	Y	N	N	N	N	N	N	N	N	N	P	A	Y	2	2	5	Y	N	N	N	20	20
41	47	2	1	N	N	N	N	N	N	N	N	N	N	Y	N	P	A	Y	3	3	5	Y	N	N	A	70	40
42	30	3	-	N	Y	Y	Y	N	Y	Y	N	Y	N	N	N	P	A	Y	3	3	3	N	N	N	N	50	20
43	40	2	2	N	Y	Y	Y	N	Y	Y	Y	Y	N	N	N	P	A	Y	2	1	3	Y	N	N	N	14	20

Sl. No.	Age (Years)	ProlapseSince	Menopause	Stress Urinary Incotinance	Urge Incotinance	Increase Frequency	Nocturia	Hesitancy	Straining to Void	Incomplete Emptying	Poor Stream	Intermitant Stream	Post Micturation Dribbing	Need to reduce	Cough Stress Test	Vaginal Rugosity	Decubitous Ulcer	Cystocele	Degree Of Prolapse	POP Q	Parity	Home Delivery	Difficult Delivery	UTI	Renal Function	Pre OP	Post OP
44	70	2	30	Y	Y	Y	Y	N	Y	Y	N	N	Y	Y	Y	A	A	Y	3	3	4	Y	N	N	N	108	26
45	42	1	1	Y	Y	Y	Y	Y	Y	Y	Y	N	N	Y	N	P	A	Y	3	3	4	N	N	N	N	36	22
46	40	6	-	N	Y	Y	Y	Y	Y	Y	Y	N	N	Y	N	P	A	Y	3	4	3	Y	Y	N	N	58	10
47	48	1	10	N	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	N	P	A	Y	3	3	1	N	N	N	N	33	2
48	61	25	20	N	Y	Y	Y	N	Y	Y	N	N	N	Y	N	A	P	Y	3	3	4	Y	N	A	N	58	20
49	45	3	3	N	N	Y	N	Y	N	N	N	N	N	Y	Y	P	A	Y	2	2	3	N	Y	N	N	12	34
50	40	2	-	Y	N	N	N	Y	N	N	N	N	N	N	N	P	A	Y	2	2	4	Y	N	N	N	15	23
51	60	20	20	Y	Y	Y	Y	N	Y	Y	Y	N	Y	Y	Y	P	A	Y	4	4	6	Y	Y	N	N	90	8
52	72	4	20	N	N	Y	Y	N	Y	Y	N	N	N	Y	N	A	P	Y	4	4	7	Y	N	N	N	93	12
53	35	6	-	N	N	N	N	Y	Y	Y	Y	Y	Y	Y	N	A	A	Y	3	4	6	Y	N	N	N	96	40
54	45	2	-	N	Y	Y	Y	N	Y	N	N	Y	N	N	N	A	P	Y	3	4	3	Y	N	N	N	100	64
55	58	1	10	N	N	Y	Y	N	Y	Y	N	N	N	Y	N	P	A	Y	3	3	4	Y	N	N	N	10	24
56	25	2	-	Y	N	Y	Y	N	Y	Y	Y	N	Y	Y	N	P	A	Y	3	4	3	N	N	N	N	20	35
57	40	18	-	N	N	N	Y	N	Y	Y	Y	Y	Y	Y	N	P	P	Y	3	4	2	Y	Y	N	N	80	35
58	50	3	10	N	Y	Y	Y	N	Y	Y	N	N	N	Y	N	A	A	Y	4	4	3	Y	N	N	N	70	18
59	55	1	5	N	N	Y	Y	N	N	Y	N	N	N	N	N	A	P	Y	2	2	3	Y	N	A	N	26	10
60	45	1	-	N	N	N	N	N	N	N	Y	N	N	Y	N	P	A	Y	3	4	3	Y	N	N	N	22	8
61	60	3	6	N	Y	Y	Y	N	N	N	N	N	N	Y	N	P	A	Y	3	3	3	Y	N	N	N	38	12
62	48	1	-	N	N	N	N	N	N	N	N	N	N	Y	N	P	A	Y	3	4	3	Y	N	N	N	18	20
63	55	1	11	N	Y	N	N	Y	N	N	N	N	N	Y	N	A	A	Y	3	3	6	N	N	A	N	67	20
64	40	6	-	Y	Y	Y	N	N	Y	Y	N	N	N	N	Y	P	A	Y	2	2	4	N	N	N	N	16	12
65	65	6	15	N	Y	N	Y	N	N	Y	N	Y	Y	Y	N	P	A	Y	2	2	7	Y	Y	N	N	58	24