Clinical Evaluation of Urinary Incontinence

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Departments of Obstetrics and Gynaecology and Anatomy, Saveetha Medical College, Saveetha University, Chennai, Tamil Nadu, India Pelvic organ prolapse is the downward descent of the pelvic floor organs and has a prevalence of 3%–6% and can even reach to 50% if defined by a vaginal examination. The anatomical concepts of pelvic diaphragm, lateral attachment of vagina to arcus tendineus fascia pelvis, and intrinsic and extrinsic sphincter control mechanisms are elaborated. The anatomic and physiological mechanisms of autonomic and voluntary control of continence are discussed. The clinical and urodynamic tests and their implications in guiding the management are explained. Finally, uroflowmetry, cystometry, urethral pressure profile, postvoid urine measurement, leak point pressure (LPP) test, video urodynamic tests, and electromyography studies of pelvic floor are discussed as an integral part of the assessment.

Keywords: Electromyography, physiopathology pelvic floor, stress, urethra, urinary bladder, urinary catheterization, urinary incontinence, urinary retention, urination disorders, urodynamic

INTRODUCTION

P elvic floor dysfunction can be studied in three compartments. The anterior compartment, the central compartment and the posterior compartment defect. Anterior compartment consists of the bladder and urethra. The central compartment consists of the uterus and the cervix and the rectum and perineal body form the posterior compartment. The lateral compartment defect is the detachment from the arcus tendineous fascia pelvis. The anterior compartment dysfunction is the most common.

Defining urinary incontinence is easy, women who leak urine must be incontinent. International continence society recently defined incontinence as "the complaint of any involuntary leakage of urine." Unfortunately, this definition does not take into account the wide variation in this symptom and the disruptions that cause it. For example, half of the young nulliparous women report occasional minor urinary leakage, most of them do not seek treatment. On the contrary, 5%–10% of adult women have severe leakage daily. These women alter their lifestyle significantly because of leakage, curtailing activities, social activity, and intimacy. In between these two categories, there lies another one-third of women

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who report leakage at least weekly, but without the same degree of life-altering severity.

Severity and quantity of urine lost and frequency of incontinence episodes that should be considered in history are as follows:

- 1. Duration of complaint and whether the problem has been worsening
- 2. Triggering factors or events (e.g., cough, sneezing, lifting, bending, feeling of urgency, feeling of running water, sexual activity, or orgasm)
- 3. Constant or intermittent urine loss and provocation by minimal increase in intraabdominal pressure. Such as movement, changes in position, and incontinence with an empty bladder
- 4. Associated frequency, urgency, dysuria, pain with a full bladder, and a history of urinary tract infections
- 5. Concomitant symptoms of fecal incontinence or pelvic organ prolapse
- 6. Coexisting complicating or exacerbating medical problems such as diabetes

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- 7. Obstetrical history, including difficult deliveries, episiotomy, grand multiparty, forceps use, obstetrical lacerations, and large babies. The labor history should be elaborated. The patient must be asked about normal labor, instrumental deliveries, or cesarean section. The birth weight of neonate and early resumption of daily activities in the puerperium should be asked. In prolonged second stage of labor when the fetal head hinges behind the symphysis pubis, there can be denervation of the smooth and striated urethral sphincter. The injury is more common in gynaecoid and least common in android pelvis
- 8. History of the previous pelvic surgeries, especially the incontinence procedures, hysterectomy, or pelvic floor reconstructive procedures
- 9. History of spinal and central nervous system surgeries
- 10. Lifestyle issues such as smoking, alcohol or caffeine abuse, and occupational and recreational factors causing severe or repetitive increase in intraabdominal pressure
- 11. Patients with coexisting pelvic organ prolapse may report dyspareunia, vaginal pain on ambulation, and a bulging sensation in vagina
- 12. Cancer of pelvic organs needs to be excluded from the study
- 13. Medication history has to elicit in detail. Medications that may cause urinary incontinence are:
 - a. Cholinergic and Anticholinergic drugs
 - b. Alpha blockers
 - c. Over counter allergy medications
 - d. Estrogen replacement irregular use
 - e. Betamimetics
 - f. Sedatives
 - g. Muscle relaxants
 - h. Diuretics
 - i. Angiotensin-converting enzyme inhibitors.

Incontinence can be improved and frequently cured with simple, nonsurgical interventions. In history, it is important to realize the reversible causes of incontinence.

Reversible causes of incontinence

- D-Delirium
- I-Infection
- A-Atrophic urethritis and/or vaginitis
- P-Pharmacological causes
- P-Psychological causes
- E-Excessive urine production
- R-Restricted mobility
- S-Stool impaction.

ANATOMICAL CONSIDERATIONS

In 1555, Andreas Vesalius referred to the pelvic floor muscles as "Musculus sedem attollens." Von Behr later

referred this as levator ani. Meyer (1861) first coined the term pelvic diaphragm that included coccygeous, iliococcygeus, and pubucoccygeous which all forms the levator ani. Puborectalis or "Sphincter Recti" was first described by German anatomist (Holl 1987) as the fibers of pubococcygeus which loop around the rectum, and this muscle is now included in the levator ani group. As the muscles of the pelvic diaphragm are intimately related to the urethra, vagina, rectum, and anal canal, the term pubovisceralis for the muscles of pelvic floor was first coined by Lawson and was later supported by Delancey and Ashton-Miller.^[1-3]

The muscles in pelvis can be classified into two groups. The lateral wall muscles and the pelvic floor muscles. The lateral wall muscles include the obturator internus and pyriformis and the pelvic floor muscles include the levator ani and coccygeus [Figure 1a and b]. The pelvic floor muscles form the pelvic diaphragm.^[4,5] The levator ani is a broad, thin sheet of muscle arising from the inner aspect of the pelvic walls unites with its fellow from the opposite side to form the floor of pelvic cavity. It supports the pelvic viscera, and some of its fibers gets attached to the wall of the visceral structures passing through it. The levator ani has an extensive origin starting from the posterior surface of the superior ramus of pubic bone, obturator fascia to the inner aspect of the ischial spine. The fibers pass downward and backward thus creating a shallow saucer-like structure on which the pelvic viscera rests [Figure 1c]. The posterior-most fibers from either side get attached to the sides of the terminal two pieces of coccyx. Fibers immediately anterior to this unit with the fellow fibers of opposite side to form a median anococcygeal raphe extending between the coccyx and the posterior margin of anus. The posterior fibers of levator ani which is called ischiococcygeus (arising from the ischial spine and posterior part of the tendinous arch of pelvic fascia) is very thin and even sometimes replaced with a fibrous tissue. The middle and the anterior fibers of levator ani are called the pubococcygeous, which arises from the inner aspect of pubis and anterior part of tendinous arch of pelvic fascia.[4,6]

The term pubovisceralis is extensively used in gynecological texts but it is not commonly mentioned in anatomical texts. The portions of pubovisceralis that are inserted into the urethra, vagina and perineal body and anal canal were given names as pubourethralis, pubovaginalis, and puboperinealis, respectively by Lawson^[4-6] The action of these muscles is to provide support to the visceral organs. The ileococcygeous muscle provides support to the coccyx with fibers of opposite



Figure 1: (a) Pelvic diaphragm viewed from below. (b) Muscles of pelvic wall: Lateral view. (c) Coronal section of pelvis. (d) Anatomical supports of bladder neck and urethra

side to form the ano coccygeal raphe or the levator plate in the median plane. This thin muscular plate supports the viscera of the pelvis especially when there is rise in intraabdominal pressure. Sagging of levator plate is an important defect leading to loss of support of these pelvic organs.^[7,8]

The perineal body, which lies posterior to the posterior vaginal wall and anterior to the wall of anal canal, is an important support of pelvic floor. The attachments and components of perineal body are still debated. In recent studies where three-dimensional (3D) endovaginal ultrasonography was used to assess the structure of the perineal body, it was shown that perineal body has mixed echogenicity and is situated between rectum, anal canal, and posterior wall of vagina.^[2,5] Perineal body is divided into two levels, i.e., a superficial level which is continuous with external anal sphincter, bulbospongiosus, and the superficial transverse perinei muscles and a deeper part, which is in continuity with the pubovisceral muscle of the pelvic floor.^[6,7]

Supports to the bladder neck and urethra are provided by certain intrinsic and extrinsic factors [Figure 1d]. The intrinsic factors mainly include the internal urethral sphincter (IUS), urethral submucosal venous plexus, urethral smooth muscles, sympathetic activity to maintain urethral tone by alpha-adrenergic receptors, and estrogen, which increases collagen connective tissue. The extrinsic factors include the contraction of pubourethralis part of levator ani, external urethral sphincter (EUS), pubourethral ligament, condensed endopelvic fascia, anterior vaginal wall, and fascia and exercise, which increases collagen, turn over.

During increase in intraabdominal pressure, the escape of urine is prevented by reflex contraction of urethral striated sphincter and periurethral musculature. There is kinking of urethra due to downward and backward movement of bladder base and thus bladder neck and proximal urethra are compressed against the anterior vaginal wall by the urethrovaginalis part of EUS. Stress urinary incontinence, that is an involuntary loss of urine in sufficient amount or frequency to constitute a social and or health problem, is a heterogeneous condition that ranges in severity from dribbling small amounts of urine to continuous passage of urine. It can result due to urethral hypermobility and downward displacement of bladder neck when there is a weakness of the supports.^[1,2,6]

The urethra passes from the bladder neck to open at the EUS for a distance of 4 cms. The IUS is a continuation of the detrusor muscle, and it is not under voluntary control. The IUS is made up of smooth muscle and is surrounded by layers of striated muscle. The sympathetic innervations of the bladder are from T10 to L2 segments of spinal cord, which also results in closing of the IUS. The parasympathetic activity from S2 to S4 segments of spinal cord causes the bladder to contract and allows the relaxation of IUS. The EUS in females is more elaborate and is made up of striated muscle fibers.

The female EUS consists of three parts (a) the true annular part, (b) part passing anterior to the urethra and attaching

to the ischial rami (compressor urethral muscle), and (c) part encircling both urethra and vagina (urethrovaginal sphincter). The urethral sphincter surrounds the urethra in the middle-third of its length. The ventral side of the urethra is thickest and dorsal side of the urethra is thinnest. The compressor urethrae muscle arises from the ischiopubic rami of each side by a small tendon. Fibers pass anteriorly to meet the counterparts in a flat band, which lies anterior to the urethra, and extend dorsally along the lateral wall of urethra and extend to the vagina along its posterior wall to meet the counterparts and thus encircle the vagina and urethra.

The spongy submucosa of the urethra has a rich vascular plexus that is responsible in part for providing pressure for sealing the urethra.^[7] The muscular layer of the urethra keeps the urethra closed. The posterior wall of urethra is embedded and supported by the endopelvic connective tissue that also envelops the anterior vagina. This support of proximal urethra and bladder neck has been compared to a sling or hammock. This connective tissue integrity has a direct relation with the hormonal and genetic factors.

The endopelvic connective tissue in this area attaches to the perineal membrane and laterally it stretches over the levator ani and condenses to form the arcus tendinous fascia pelvis, which stretches from pubic bone till the ischial spine. This arcus tendinous fascia pelvis lies at the junction of the fascia of the obturator internus and levator ani muscle. This tissue provides support to urethra and bladder neck and has been strengthened in repair surgeries. Defects in this tissue lead to cystocele and urethral hypermobility. The primary support to the bladder neck and urethra is from the levator ani muscle complex. The tone of these slow-twitch fibers of the levator ani muscle complex forms a major supportive mechanism. On the other side, with an increase in acute abdominal pressure, it is the fast-twitch fibers of levator ani muscles that contract and this elevates the pelvic floor and tightens the connective tissue and thus it provides support to the pelvic organs.^[2,8,9]

The pubourethral ligaments support the anterior wall of the urethra and thus suspend the urethra under pubic arch. These ligaments are extensions of arcus tendinous fascia pelvis and perineal membrane. With increase in intra-abdominal pressure, these ligaments stabilize the anterior wall of urethra and decrease its downward mobility. The internal sphincter in females is more of a functional concept. The external sphincter complex of female urethra is much more developed and can be strengthened with Keagel's exercises.

The urethral pressure normally exceeds the bladder pressure thus urine is retained in bladder, which helps in

filling. During urination, the detrusor muscle contracts and urethral sphincters relax thus letting urine to pass out. About 33% of all women experience urinary incontinence after giving birth and women who have delivered vaginally will have twice more chance of developing stress urinary incontinence.^[9]

The urinary tract functioning is dependent on the sympathetic and parasympathetic innervations to the urethra, urethral sphincter and bladder. During the filling and storage phase, there is mostly no change in bladder pressure, and this mainly occurs due to stimulation of beta-adrenergic receptors in the detrusor muscle by sympathetic stimulation, which leads to the relaxation of above muscle. The alpha-adrenergic predominate in the region of the bladder neck and urethra and stimulation of which by sympathetic activity leads to contraction of these structures promoting filling. The act of voiding or micturition is more due to parasympathetic stimulation. The muscarinic receptors of detrusor when stimulated by parasympathetic activity promote contraction of detrusor. Stimulation of alpha-adrenergic nerve activity on muscarinic receptors of bladder neck, urethra can be increased by estrogen whereas progesterone increase beta-adrenergic activity.^[8,9]

INITIAL EVALUATION

The initial evaluation of patients with incontinence requires a systematic approach to consider the probable causes.

History

A thorough medical history should be obtained from every incontinent patient. Quality of life measures such as King's health Questionnaire; Overactive Bladder Symptom score (OABSS) can be useful.^[10] Physicians treating the incontinent patient should empathically ask them how the incontinence specially affects their life and to what degree the incontinence bothers them. Taking history of diabetes mellitus is of special importance in urge urinary incontinence due to glycosuria, detrusor overactivity, recurrent urinary tract infections, and diabetic cystopathy.

Physical examination

The physical examination of the patient with incontinence should focus on both the general medical conditions that may affect the lower urinary tract as well as the problems related to urinary incontinence.^[11] Body mass index is of special importance because a body mass index of >30 is associated with the recurrence of symptoms following surgeries done for pelvic organ prolapse. Hence, lifestyle changes, Keagle exercises, and weight reduction are done along with incontinence surgery. Other conditions include cardiovascular insufficiency, pulmonary disease, occult neurologic processes, (e.g., Multiple sclerosis, stroke, Parkinson's disease, and anomalies of the spine and lower back), abdominal masses, and general activity of the patient.^[12,13]

Simple primary care tests

Simple primary care clinical tests are an integral part of initial evaluation.^[14] It is important to realize that formal sophisticated urodynamic tests are neither the only nor the most important, tests of bladder function. Simple clinical tests can be performed in the primary care and they provide useful information to guide the patient care.

The following questionnaires have been recommended by the international consultations on incontinence and its impact on quality of life in women.^[15-18]

- 1. Urogenital distress inventory (UDI)
- 2. Urge UDI
- 3. King's Health Questionnaire
- 4. Quality of life in person's with urinary incontinence (I-QOL)
- 5. Incontinence Impact Questionnaire-IIQ 7
- 6. Urge-IIQ
- 7. OABSS.

Voiding diary

A frequency volume bladder chart (bladder diary) is an invaluable aid in the evaluation of patient with urinary incontinence.^[19]

Systemic examination

Gait assessment should be done and mobility status should be noted. A detailed neurological examination should incorporate measures of mental status, perineal sensation, perineal reflexes, and patellar reflexes. Cardiovascular examination should be done to rule out lower extremities edema and feature of congestive heart failure.^[20,21]

Pelvic examination

A special note should be made regarding pelvic organ prolapse and atrophy. Pelvic examination should be done with full bladder. Degree of pelvic relaxation with cystocele or cystourethrocele should be noted. The examination tests are stress test, pad test, and Q-tip test. Levator ani muscle symmetry should be noted during the ability to squeeze test. Anal sphincter function, presence of fissures, and symmetry during squeezing should be noted.

Tests of urethral mobility

These clinical tests tell us about the urethral hypermobility and the impact urethral replacement surgery might have on relieving the incontinence.^[22-24]

1. Cough stress tests: Patients should be examined with full bladder, particularly if stress incontinence is being considered. Urine egress from urethra at the

time of cough documents stress incontinence.

- 2. Pad test: Pad test is performed by quantifying the volume of urine lost by weighing a perineal pad before and after specified activities. The patient is asked to drink 500 ml of water and rest for 5 min. She is then asked to walk for 30 min and perform as strenuous activity such a climbing stairs for 15 min. After 1 h, an increase in the pad weight by 1 g is considered significant
- 3. Paper towel test. A paper towel test provides a quick estimate of the degree of stress urine loss. The patient is asked to cough repetitively and forcefully with a paper towel held a short distance from the urethra
- 4. Cotton swab test/Q tip test. A cotton swab angle of more than 30° denotes urethral hypermobility. A sterile cotton swab tip is lubricated with 2% xylocaine jelly and is introduced through the urethra till the bladder neck. Then, the patient is asked to sit and cough. If the cotton swab angulates >30° during coughing, the urethra is considered hypermobile.

URODYNAMIC STUDIES

Urodynamic studies are a means of evaluating pressure flow relationship between bladder and urethra. These provide further evidence about lower urinary tract function. Obtaining clinically valuable information does not always require the use of expensive, complex technology; however, these may be indispensable if we want to reach a correct diagnosis of urge and mixed incontinence.^[25] After basic tests further tests are required in the circumstances given below:

- 1. The diagnosis is uncertain (major discrepancies between the history, voiding dairy, and symptom scale)
- 2. Planned surgery
- 3. Presence of hematuria
- 4. Elevated postvoid residual urine volume
- 5. Associated neurological conditions like multiple sclerosis
- 6. Associated pelvic organ prolapse
- 7. Previous surgery for incontinence correction
- 8. Presence of mixed symptomatology (genuine stress incontinence and detrusor instability).

Components of urodynamic studies

The following form an integral part of urodynamic assessment.

- 1. Uroflowmetry
- 2. Cystometry: a. Single channel cystometrography b. Multichannel cystometrography
- 3. Postvoid residual (PVR) urine volume
- 4. Urethral pressure profiles for urethral closure pressures

- 5. Leak Point Pressure (LPP) measurement
- 6. Neurophysiologic studies.

UROFLOWMETRY

Uroflowmetry is a measurement of rate of flow of urine. Volume of urine voided overtime is plotted a graph.^[26] Normal flow rate is 15–25 ml/s. Flow rates <10 ml/s indicate atonic bladder or bladder outlet obstruction. A specialized equipment automatically measures the amount of urine and flow rate. Uroflowmetry equipment consists of a device for measuring the volume of urine voided and a computer to record the data. During a uroflowmetry test, the person voids privately into a special funnel that has a container and a measuring scale. The equipment creates a graph that shows changes in flow rate with time. Figure 2a-d shows the graphical representation of abnormal uroflow patterns.

Cystometry

Cystometry is done to assess bladder and urethral function during the filling and voiding phase.^[27]

Single channel cystometry

In this, the bladder pressure is measured during the filling phase. Since the bladder is an intraabdominal organ, pressure recorded in bladder is a combination of several other pressures such as detrusor activity and pressure exerted on bladder by other intraabdominal organs. So, we cannot get the true bladder pressures and hence we use the multichannel cystometry.

Multichannel cystometry

The multichannel urodynamic give an accurate estimate of detrusor pressure (P_{det}) . The P_{det} is determined by measuring the total intravesical pressure (P_{ves}) and total intraabdominal pressure (P_{abd}) . To conclude $P_{det} = P_{ves} - P_{abd}$.

A catheter is used to empty the bladder completely. Then, a special catheter is placed in the bladder, which has a pressure-measuring device called a manometer. Another catheter is placed in the rectum to record the pressure there. The bladder is filled slowly with warm water or saline through a filling catheter. During this time, the patient is asked to inform when the need to urinate arises. When the urge to urinate occurs, the volume of water infused and the bladder pressures are recorded. Pressure measurements are also done during the voiding phase.

Cystometric phases of bladder function

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- 1. Initial small increase in $\mathrm{P}_{\mathrm{ves}}$ at the beginning of the filling
- 2. Stable pressure that lasts for the majority of the filling phase



Figure 2: Graphic representation of various uroflow patterns. (a) Superflow commonly seen with reduced urethral flow resistance. (b) Intermittent multiple peak pattern. (c) Intermittent interrupted pattern. (d) Abnormal flow rate characteristic of detrusor outlet obstruction

- 3. Terminal rise in pressure at the maximum bladder capacity representing limit of viscoelastic expansion
- 4. The last phase is the voiding phase with an inconsistently observed rise in P_{ves} as the patient voids.

In a person with normal detrusor function, the P_{det} remains at zero or rises slightly while saline is infused [Figure 3a]. When a person is asked to cough there is a sharp rise in intaabdominal pressure and an equal rise in P_{ves} . The P_{det} remains unaffected in a normal individual [Figure 3b] when asked to perform Valsalva maneuver. While voiding the P_{det} rises as the bladder contracts to empty. There is little or no rise in intaabdominal pressure unless the person strains [Figure 3c].



Figure 3: Recording of multichannel cystometry. (a) In a person with normal detrusor function, the detrusor pressure remains at zero or rises slightly while collecting urine, even at the maximum capacity. (b) In a person with normal detrusor function, cough and other maneuvers are seen as sharp rise and fall, with equal transmission to bladder and abdominal channels. There is no rise in detrusor pressure. (c) In a person with normal voiding pattern the detrusor pressure rises as the bladder contracts to empty. There is little or no rise in abdominal pressure during voiding (unless the person strains)

Normal findings in cystometry

- 1. First sensation of urination: 150-200 ml
- 2. Maximum capacity: 400-600 ml
- 3. P_{ves} on filling 0–15 cc of H_2O
- 4. Peak urinary flow rate of 15 ml/s
- 5. Absence of systolic detrusor contractions. The International incontinence society has identified a minimal contraction of 15 cm of H_2O over the baseline to be considered significant.

In genuine stress incontinence, cystometric evaluation is normal. Values are abnormal in detrusor instability and sensory urge incontinence.^[28] Demonstration of urgency coincident with increased P_{det} and urinary leakage in a neurologically intact patient defines the diagnosis of detrusor instability [Figure 4].

POSTVOID RESIDUAL URINE DETERMINATION

High-postvoid residual urine volume indicates outlet obstruction or impaired bladder contractility due to detrusor problems. PVR volumes can be measured directly by post void catheterization or ultrasonography. It is important to perform this test within 10 min of voiding to avoid any false positive results. It is agreed that a post void residual urine level of 50 ml to 200 ml is normal.^[29] Bladder scans appear to be reasonably sensitive and specific for elevated PVR measurements.

URETHRAL PRESSURE PROFILOMETRY

It is a technique of recording pressures along the length of urethra with the bladder at rest. The maximum urethral pressure in urethra is the maximum pressure in urethra minus the P_{ves} [Figure 5]. The functional length

of urethra is the distance along the urethra in which the urethral pressure exceeds the P_{ves}

The urethral pressure profile is determined by slowly pulling out a pressure sensitive catheter through the urethra from the bladder. Urethral pressure profile has many clinical implications. The resting urethral pressure is 20 cm H_2O . Closure pressure below 20 cm H_2O is associated with intrinsic sphincter deficiency. Maximum urethral closure pressures of <20 cm H_2O have been associated with higher failure rates when these patients are treated with Burch colposuspension.

LEAK POINT PRESSURE MEASUREMENTS

The bladder pressure at which there is an involuntary leakage of urine from the urethral meatus is termed as the LPP. There are two different LPPs – the detrusor LPP and the abdominal LPP.

The LPP measurements are done at bladder volumes of 200–300 ml. Patients are asked to cough forcefully with increasing force and finally asked to perform the Valsalva maneuver. The LPP is measured by marking on the pressure recording at which the urine leaks. This can be done by fluoroscopy, direct method, or the electrical conductance measurement. Valsalva LPP is defined, as the minimum total vesical pressure required causing incontinence in the absence of detrusor contractions.

The primary aim of measuring the LPP is to assess the risk to the urinary tract in a neuropathic bladder. The abdominal LPP is a direct measure of urethral contribution to incontinence. Higher values (>90–100 cm H_2O) suggest that the stress incontinence is associated with



Figure 4: Demonstration of urgency and/or urinary leakage coincident with increased detrusor pressure in multiple channel cystometry in a neurologically intact patient defines the diagnosis of detrusor instability



Figure 5: Maximal urethral closure pressure is measured by urethral pressure profile while a pressure sensor is gradually withdrawn from the urethra

hypermobility and cure rates with transobturator tapes will be good. Low values ($<60 \text{ cm H}_2\text{O}$) suggest short urethral functional length or intrinsic sphincter deficiency.

VIDEOURODYNAMICS

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Videourodynamics traditionally combines a routine urodynamic study with a Roentgenography or ultrasound imaging, although new imaging techniques MRI and nuclear cystometrography are coming into vogue. Video urodynamics is used for patients with complicated lower urinary tract dysfunction due to a neurological condition.^[29,30] It can also offer a more accurate diagnosis to other patients [Figure 6].^[31]

Most advantages of video urodynamics arise from its ability to simultaneously measure the pressures while looking at the anatomy. Incompetent bladder neck or inadequate urethral closure during the filling phase can be documented. It also helps to document the location of urethral obstruction during voiding. Descent of bladder base, bladder base hypermobility, and intrinsic sphincter deficiency can be accurately differentiated.

Normally, the bladder should fill without any abnormal appearances to its contour. The bladder neck should be closed throughout the filling phase and the bladder base should be located above the symphysis pubis. During coughing or abdominal straining, the bladder base should remain at this level.

During voiding, the bladder neck should open smoothly and widely (with no ballooning). The urethra should be closed during filling and form a smooth conduit during voiding.

NEUROPHYSIOLOGICAL STUDIES

Electromyography (EMG) of the urethral sphincter, anal sphincter and the pelvic floor is an established method for the diagnosis of pelvic floor dysfunction.^[31] Patch electrodes or surface electrodes are placed in the perineum to record the EMG activity. In women, vaginal surface EMG has a better correlation [Figure 7].^[32] However, the recorded EMG activity may not always show the activity of the sphincters.

THREE DIMENSIONAL ENDOSONOGRAPHY

Ultrasound with color Doppler is an important tool to assess the pelvic floor supports. The resting urethral



Figure 6: Videourodynamics of bladder and urethra can assess both the anatomy and physiology of urinary tract



Figure 7: Electromyography study can assess functional tone as well as the response to treatment (muscle strengthening exercises and neuromodulation)^[32]

pressure depends on three parameters. One-third is due to rhabdosphincter effects, one-third is due to smooth muscle effects, and one-third is due to its vascular plexus. Estrogen deficiency leads to atrophy of surrounding structures and periurethral vascularity decreases. Transvaginal sonography with 3 D images is used to assess the anatomy of bladder neck, urethra, and bladder wall thickness. Ultrasonography is specifically indicated in patients with hematuria, neuropathic bladder, congenital anomalies, calculi, and fistulas. There are also rectal, perineal, and intraurethral probes for 3D visualization.

CONCLUSION

The assessment of pelvic floor dysfunction can be done clinically aided with imaging. When the patient complains of stress incontinence, the pathophysiology is urethral displacement or urethral hypermobility, the diagnosis is genuine stress incontinence and the treatment is surgical correction to restore the anatomy. When the patient complains of urge incontinence, the pathophysiology is detrusor instability, the diagnosis is over active bladder and the treatment is selective antimuscurinic agents or bladder distention procedures. There are a few patients of sensory incontinence that be diagnosed with newer modalities such as videouroflowmetry and these respond to neuromodulator therapies. The newer imaging modalities help to diagnose mixed incontinence with accuracy.

Each case should be individualized; keeping in mind that mixed incontinence is frequently present. A detailed assessment can help us to decide the therapy that will be of maximal benefit to the patient.

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Conflicts of interest

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References

- 1. Delancey JO, Ashton-Miller JA. Pathophysiology of adult urinary incontinence. Gastroenterology 2004;126:S23-32.
- Ren S, Xie B, Wang J, Rong Q. Three-dimensional modeling of the pelvic floor support systems of subjects with or with out pelvic organ prolapse. Biomed Res Int 2015;2015:845985.
- De Groat WC. Neuroanatomy and neurophysiology: Innervation of the lower urinary tract. In: Raz S, Rodriguez L, editors. Female Urology. 3rd ed., Vol. 3. Philadelphia, PA: W.B. Saunders Company; 2008. p. 26-46.
- Raizada V, Mittal RK. Pelvic floor anatomy and applied physiology. Gastroenterol Clin North Am 2008;37:493-509, vii.
- Loubeyre P, Copercini M, Petignat P, Dubuisson JB. Levator ani muscle complex: Anatomic findings in nulliparous patients at thin-section MR imaging with double opacification. Radiology 2012;262:538-43.
- Mouchel T, Mouchel F. Basic Anatomic features in Pelviperineology. Pelviperineology 2008;27:156-9. Available

from: http://www.pelviperineology.org. [Last accessed on 2018 May 30].

- Larson KA, Yousuf A, Lewicky-Gaupp C, Fenner DE, DeLancey JO. Perineal body anatomy in living women: 3-dimensional analysis using thin-slice magnetic resonance imaging. Am J Obstet Gynecol 2010;203:494.e15-21.
- Richard S Snell Clinical Anatomy 9th edition. Wolters Kluver, Lippincott Williams & Wilkins. Philadelphia, PA 19103; 2012.
- Gill BC, Damaser MS, Vasavada SP, Goldman HB. Stress Incontinence in the Era of Regenerative Medicine: Reviewing the Importance of the Pudendal Nerve. The Journal of Urology, 2013;190:22-8. Available from: http://doi.org/10.1016/j. juro.2013.01.082. [Last accessed on 2018 May 30].
- Gormley EA, Lightner DJ, Burgio KL, Chai TC, Clemens JQ, Culkin DJ, *et al.* Diagnosis and treatment of overactive bladder (non-neurogenic) in adults: AUA/SUFU guideline. J Urol 2012;188:2455-63.
- Lucas MG, Bosch RJ, Burkhard FC, Cruz F, Madden TB, Nambiar AK, *et al.* EAU guidelines on surgical treatment of urinary incontinence.; European Association of Urology.Eur Urol. 2012;62:1118-29. doi:10.1016/j.eururo.2012.09.023. Epub 2012 Sep 17. PMID: 23040204.
- Tai HC, Chung SD, Ho CH, Tai TY, Yang WS, Tseng CH, et al. Metabolic syndrome components worsen lower urinary tract symptoms in women with type 2 diabetes. J Clin Endocrinol Metab 2010;95:1143-50.
- 13. Kupelian V, McVary KT, Kaplan SA, Hall SA, Link CL, Aiyer LP, *et al.* Association of lower urinary tract symptoms and the metabolic syndrome: Results from the Boston Area Community Health Survey. J Urol 2009;182:616-24.
- Goldman HB, Wyndaele JJ, Kaplan SA, Wang JT, Ntanios F. Defining response and non-response to treatment in patients with overactive bladder: A systematic review. Curr Med Res Opin 2014;30:509-26.
- Homma Y, Yoshida M, Seki N, Yokoyama O, Kakizaki H, Gotoh M, *et al.* Symptom assessment tool for overactive bladder syndrome – Overactive bladder symptom score. Urology 2006;68:318-23.
- Homma Y, Yoshida M, Yamanishi T, Gotoh M. Core lower urinary tract symptom score (CLSS) questionnaire: A reliable tool in the overall assessment of lower urinary tract symptoms. Int J Urol 2008;15:816-20.
- 17. Avery K, Donovan J, Peters TJ, Shaw C, Gotoh M, Abrams P, *et al.* ICIQ: A brief and robust measure for evaluating the symptoms and impact of urinary incontinence. Neurourol Urodyn 2004;23:322-30.
- Abrams P, Avery K, Gardener N, Donovan J, ICIQ Advisory Board. The international consultation on incontinence modular questionnaire: www.iciq.net. J Urol 2006;175:1063-6.
- 19. Homma Y, Kakizaki H, Yamaguchi O, Yamanishi T,

Nishizawa O, Yokoyama O, *et al.* Assessment of overactive bladder symptoms: Comparison of 3-day bladder diary and the overactive bladder symptoms score. Urology 2011;77:60-4.

- 20. Apostolidis A, Kirana PS, Chiu G, Link C, Tsiouprou M, Hatzichristou D, *et al.* Gender and age differences in the perception of bother and health care seeking for lower urinary tract symptoms: Results from the hospitalised and outpatients' profile and expectations study. Eur Urol 2009;56:937-47.
- Irwin DE, Milsom I, Hunskaar S, Reilly K, Kopp Z, Herschorn S, et al. Population-based survey of urinary incontinence, overactive bladder, and other lower urinary tract symptoms in five countries: Results of the EPIC study. Eur Urol 2006;50:1306-14.
- Swartz M, Vasavada S. Diagnosing, treating refractory OAB. Renal and Urology News; 2009. Available from: http://www. Renalandurologynews.com/diagnosing-treating-refractory-oab/ Article/129979. [Last accessed on 2018 May 30].
- 23. Benner JS, Nichol MB, Rovner ES, Jumadilova Z, Alvir J, Hussein M, *et al.* Patient-reported reasons for discontinuing overactive bladder medication. BJU Int 2010;105:1276-82.
- Maserejian NN, Wager CG, Giovannucci EL, Curto TM, McVary KT, McKinlay JB, *et al.* Intake of caffeinated, carbonated, or citrus beverage types and development of lower urinary tract symptoms in men and women. Am J Epidemiol 2013;177:1399-410.
- 25. Abdul-Rahman A, Al-Hayek S, Belal M. Urodynamic studies in the evaluation of the older man with lower urinary tract symptoms: When, which ones, and what to do with the results. Ther Adv Urol 2010;2:187-94.
- Sutherst JR, Brown MC. Comparison of single and multichannel cystometry in diagnosing bladder instability. Br Med J (Clin Res Ed) 1984;288:1720-2.
- Heidler S, Deveza C, Temml C, Ponholzer A, Marszalek M, Berger I, *et al.* The natural history of lower urinary tract symptoms in females: Analysis of a health screening project. Eur Urol 2007;52:1744-50.
- Rosier PF. The evidence for urodynamic investigation of patients with symptoms of urinary incontinence. F1000Prime Rep 2013;5:8.
- Tseng LH, Liang CC, Chang YL, Lee SJ, Lloyd LK, Chen CK, et al. Postvoid residual urine in women with stress incontinence. Neurourol Urodyn 2008;27:48-51.
- Marks BK, Goldman HB. Videourodynamics: Indications and technique. Urol Clin North Am 2014;41:383-91, vii-viii.
- Video Urodynamic vs. Traditional Urodynamic: A Comparison. Posted by on Clark Love. 2017. Available from: http://www. Vlogsiteinfo.bhnco.com. [Last accessed on 2018 May 30].
- LaPallo BK, Wolpaw JR, Chen XY, Carp JS. Long-term recording of external urethral sphincter EMG activity in unanesthetized, unrestrained rats. Am J Physiol Renal Physiol 2014;307:F485-97.

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