# INTRODUCTION

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The International Continence Society defined urinary incontinence as a condition in which involuntary loss of urine is a social or hygienic problem and is demonstrable.[1] Stress urinary incontinence (SUI) occurs when the intravesical pressure exceeds the urethral resistance as a result of increased intraabdominal pressure - on effort or exertion, or on sneezing or coughing, even on change of position - in the absence of a detrusor contraction. Several classifications of stress urinary incontinence have been proposed to differentiate subtypes of incontinence related to the mechanism of leakage. SUI is a relatively common problem in women that is thought to be induced from two different reason, urethral hypermobility and intrinsic sphincter deficiency (ISD).[2]

The term ISD was originally coined by McGuire et al.[3] in the urology literature from the 1980s and was used to describe a severe form of stress urinary incontinence, equating it to an earlier term "type III urinary incontinence" reported by Blavais and Olsson.[4] In the urogynecology literature, ISD was equated with a concept introduced by Sand et al. in 1987 in an article describing the "low pressure urethra". These terms were urodynamically defined as a Valsalva leak point pressure (VLPP) of less than 60 cm $H_2O$ and a maximal urethral closure pressure (MUCP) of less than 20 cm $H_2O$ respectively. ISD has been described as a condition in which the urethral sphincter is unable to coapt and generate enough resistance to retain urine in the bladder.

ISD is a poor prognostic indicator.[5] In women, ISD is commonly associated with multiple incontinence surgical procedures, as well as with hypoestrogenism, aging, or both. In this condition, the urethral sphincter is unable to generate enough resistance to retain urine in the bladder, especially during stress maneuvers. Patients with ISD often leak continuously or with minimal exertion. In some patients, stress incontinence results from coexisting ISD and hypermobility of the urethra and bladder neck.[5]

For a time ISD was defined as stress incontinence with a well supported bladder neck (lack of hypermobility) regardless of the VLPP or MUCP. As noted above, in the 1996 AHCPR document, a mention of lack of hypermobility being a component of ISD was noted. In the International Classification of Disease, 9th edition (ICD-9), ISD was defined as a disorder of the urethra "…due to intrinsic sphincter damage in which the urethra is usually well supported but there is posterior rotation and opening of the bladder neck and posterior urethra during straining".[6]

Some studies have used the urethral pressure profile (UPP) to determine sphincter tone and, thus, identify patients with ISD. In a retrospective analysis, Sand et al.[7] Reported a 54% failure rate after surgery in patients with a low maximum urethral closure pressure (MUCP) compared with the 18% in those with a normal MUCP. The UPP measures intrinsic and extrinsic factors which maintain the resting closure pressure in the urethra, indirectly assesses the urethral resistance at rest and associates the sphincter deficiency with a low urethral pressure, regardless of its position or mobility.[8]

Other reports have evaluated sphincter function on the basis of the Valsava leak point pressure (VLPP, the lowest abdominal pressure to cause urinary leakage as recorded during a slowly performed Valsalva maneuver) and estimated its resistance to increases in abdominal pressure. McGuire et al.[9] Demonstrated that 75% of patients with Type III incontinence had a low VLPP incontinence at videourodynamic study. Opinions differ greatly: supporters of one method are reluctant to believe the other is reliable and subjective judgments tend to increase the disagreement in the approaches to diagnosis.[8]

Traditionally, ISD had only been considered in patients with a history of a significant leakage of urine with minimal provocation and a fixed urethra. However, the presence or absence of urethral mobility is not a diagnostic criterion for ISD. In fact, urethral hypermobility is present in 47–60% of patients with a urodynamic diagnosis of ISD, thereby supporting the need for urodynamic testing before selecting a surgical procedure for urinary incontinence.[10]

## Surgical anatomy

The Dynamic Anatomy of Normal Function Fig. 1 is a schematic view of the bladder, bowel and uterus with the woman in a sitting position. The organs are storage containers. The bladder stores urine, the uterus the fetus, and the rectum faeces. Each organ is connected to the outside by a tube, the urethra, which is about 4 cm long, vagina, which is 10-12 cm long, and the anus, about 4 cm long respectively. Muscles compress these tubes to close them, and stretch them open for emptying.[11]

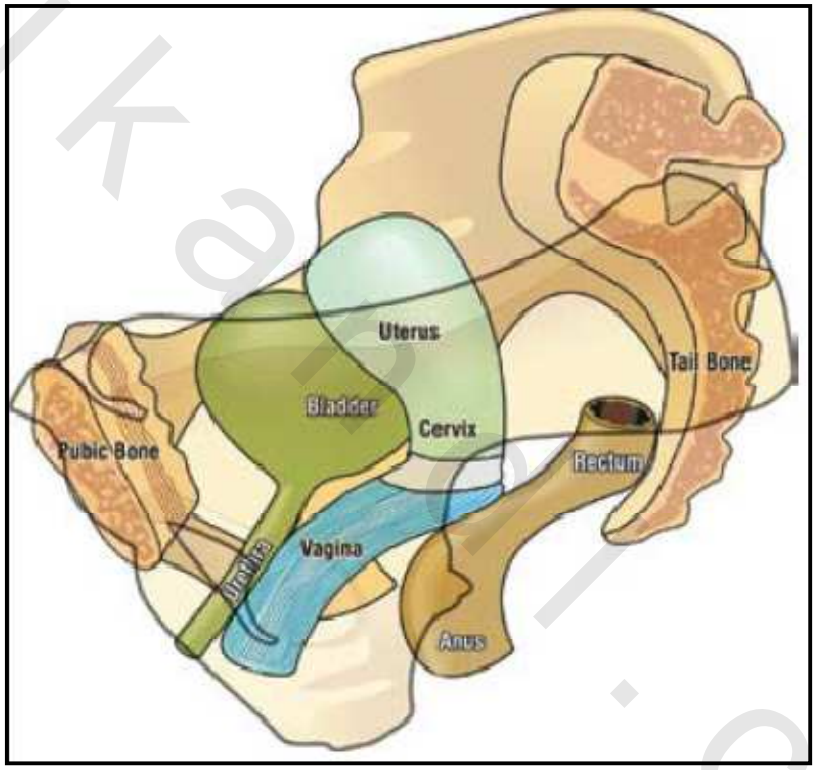


**Figure (1):** The organs and their outlet tubes.[11]

The Importance of Suspensory Ligaments and Pelvic Muscles, Problems of bladder, bowel, prolapse, and some types of pelvic pain, mainly originate from the vaginal ligaments, not from the organs themselves' – Integral Theory 1996.[12]

The bladder sits on top of the vagina, and is partly attached to it. Muscles pull against the ligaments to close or open the urethra. Therefore loose ligaments may weaken the muscle contraction to cause problems with closure (incontinence) or opening (evacuation of urine). The vagina, bladder, and bowel with no ligaments to suspend them, are but a blob of tissue, with no form, no structure, no strength, and no function.

In fact, the vagina is suspended exactly like a suspension bridge, with the ligaments above (Fig. 2), and the muscles (arrows) below (Fig. 3). The muscle forces (arrows) contract against the suspensory ligaments to give the bridge form and strength.[11]

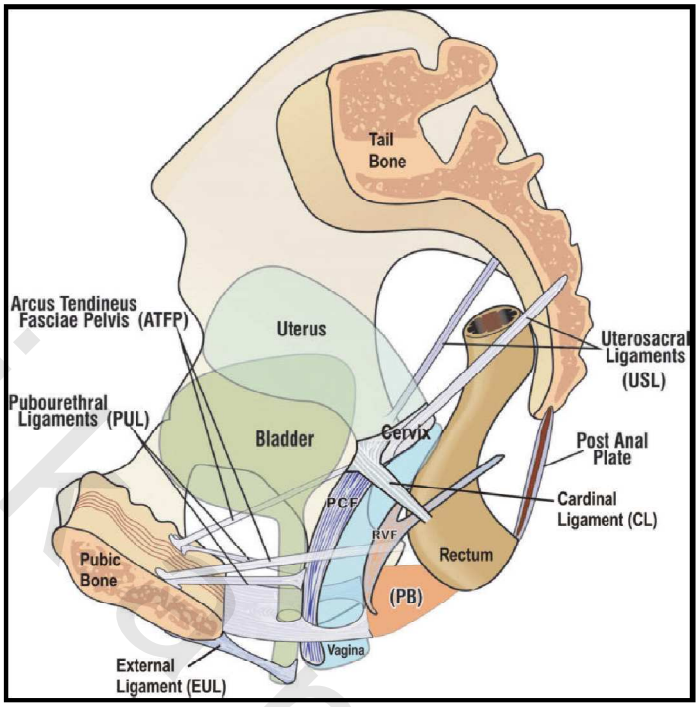


**Figure (2):** The ligaments of the pelvic floor perspective: standing position.[11]

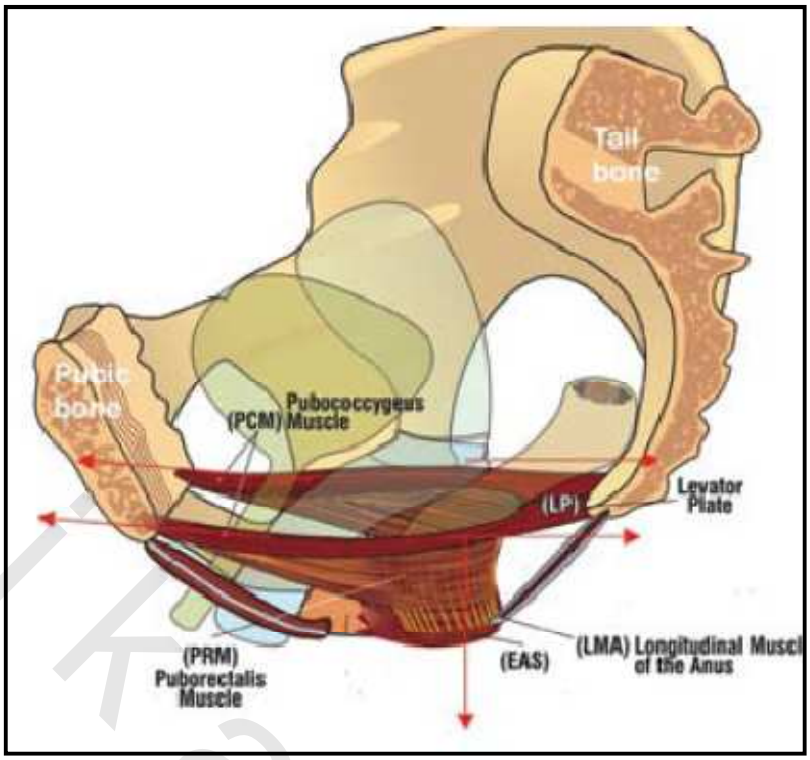


**Figure (3):** The muscles support the organs, vagina, bladder, and bowel from below, and also, open and close them by three external directional muscle forces (arrows).[11]

Five main ligaments suspend the organs from above, external urethral ligament (EUL), pubourethral ligament (PUL), Arcus Tendineus Fasciae Pelvis (ATFP), cardinal ligament (CL) and uterosacral ligament (USL). The perineal body (PB) supports the vagina and rectum from below.[13]

All the ligaments are attached to the vagina and/or uterus. The vagina supports the bladder situated above it, and the rectum situated below it, so anything which damages the vaginal structure, can also affect the bladder and rectum. Separating the lower end of the vagina from the rectum is a solid mass of tissue, the perineal body (PB) complex which is about 4 cm long. If this is damaged, the rectum may bulge forwards into the vagina as a rectocoele.

The role of the uterus in maintaining the structure and function of the pelvic floor is greatly underestimated. The uterus is an anchoring point for the ligaments; it needs to be preserved where possible. Some doctors routinely recommend removal of the uterus during surgery for prolapse. It is preferable to retain the uterus wherever possible, as many important ligaments are attached to it. During the menopause, the ovaries cease production of estrogen. Since estrogen is essential for maintaining the strength of the ligaments, the detrimental effects of hysterectomy on prolapse and incontinence become especially evident after the menopause. Hysterectomy reduces the blood supply to the cardinal and uterosacral ligaments, weakening them further. All these factors predispose to prolapse, and development of posterior zone symptoms.

The pelvic muscles (dark red, Fig. 3) have a dual function, organ support, and opening and closure of urethra and anorectum. They extend from the coccyx to the pubic bone, and contract to support the vagina, bladder, and bowel from below. The red arrows (Fig. 3) indicate the directions where the muscles contract, backwards to open these organs, forwards to close them. (11)

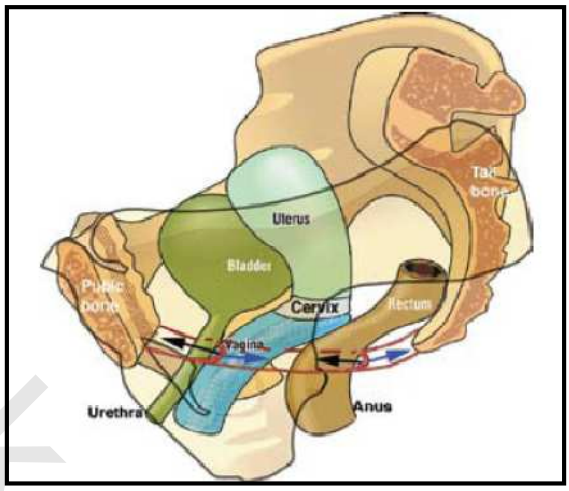


**Figure (4):** External striated muscle opening and closure mechanism. The red lines represent the pelvic muscles. Fibromuscular extensions from these muscle fibers loop around the urethra and anorectum to activate closure and opening.(11)

In the pelvic floor there are 4 main muscles. These are situated below the suspensory ligaments (Fig.5).(13)

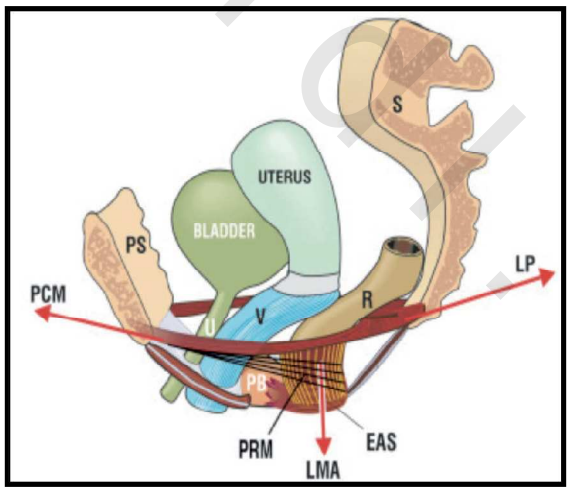


**Figure (5):** Opening and closure muscles of the pelvic floor Perspective: sitting position. Four main muscle forces work in a co-ordinated way to close and open the outlet tubes, urethra 'U', vagina 'V' and rectum 'R'. M. pubococcygeus (PCM), M. levator plate (LP), M. conjoint longitudinal muscle of the anus (LMA) contract against pelvic ligaments. Their contractile strength may diminish when their insertion ligaments are lax. M. puborectalis (PRM) contracts directly against the pubic bone and is not affected by lax ligamentous insertion points.(13)

Put simplistically, when the muscles pull backwards (blue arrows, Fig. 4), the urethra and anus are pulled open, vastly decreasing intracavity resistance to the 4$^{th}$ power, so that the woman can quickly and easily evacuate her urine and feces; when the muscles pull forwards (black arrows, Fig. 4), the urethra and anus are closed by a vast increase in resistance to the 4th power. Normally all the organs, even the vagina, are kept in the closed position by slow-twitch muscle contraction.

The closure mechanism (Fig.6) is a little more complex than that depicted in Fig. 4, involving a distal and proximal mechanism for closure of the urethral and anal tubes.[14]

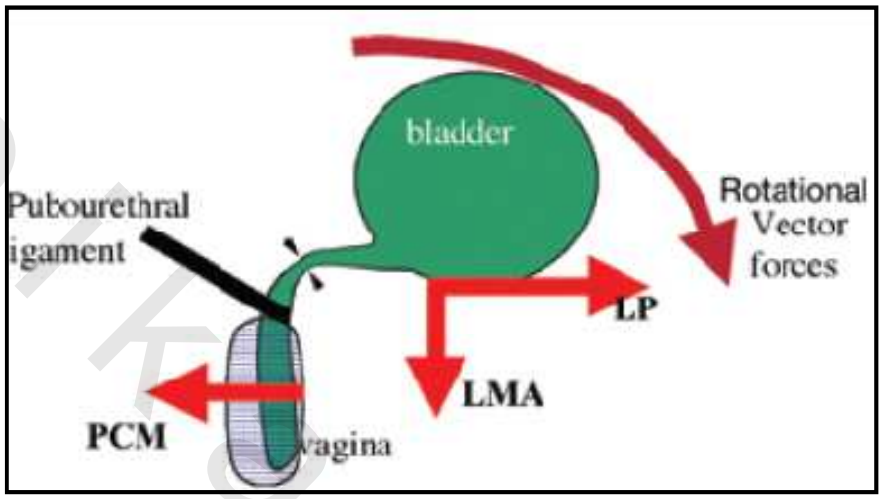


**Figure (6):** The mechanism for urethrovesical closure. Backward/downward muscle vector forces (arrows) stretch the proximal urethra around the pubourethral ligament, narrowing and kinking it; a forward vector stretches the suburethral hammock forwards against the pubourethral ligament for distal urethral closure. LP = levator plate; LMA = longitudinal muscle of the anus; PCM = anterior portion of pubococcygeus muscle.[14]

## Role of Urethral Resistance in Continence Control

External muscle forces (levator plate/longitudinal muscle of the anus (LP/LMA), Fig. 7 stretch and narrow the evacuation tube (urethra, Fig. 7) against a firm pubourethral ligament (PUL), then close it by angulating it downwards against the PUL. Narrowing a tube increases the resistance to flow inversely by the fourth power of the radius (Poisseuille's Law). A lax PUL prevents closure by the muscle forces, exponentially reducing resistance to flow of urine within the urethral tube – incontinence.[13]

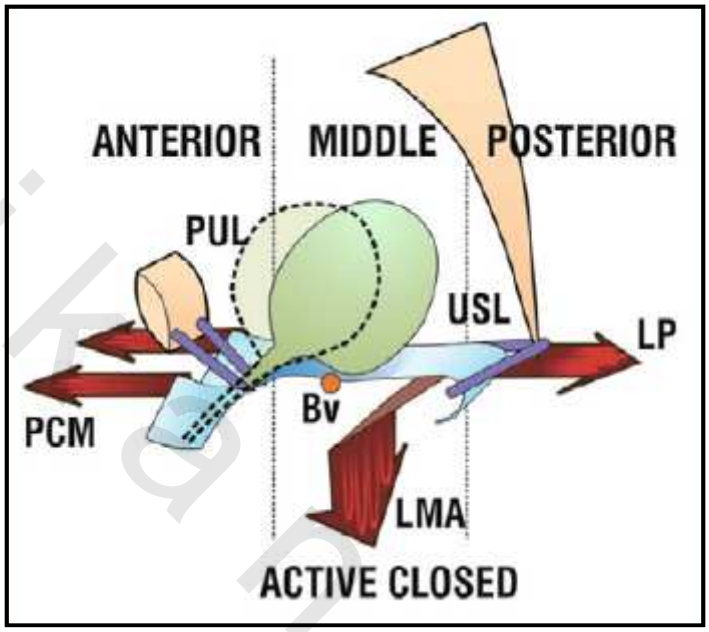


**Figure (7):** Urethral tube closure. The PCM (anterior portion of pubococcygeus muscle) contracts against the pubourethral ligament (PUL) to narrow the distal urethra. LP/LMA (levator plate/longitudinal muscle of the anus) vectors stretch the vagina backwards to narrow the proximal urethra, then rotate it around the PUL to "kink" and close the proximal urethra. Broken lines = resting phase; Bv, fibromuscular attachment of the bladder base to the vagina.[13]

How damaged ligaments may cause incontinence or emptying disorders. With reference to the suspension bridge analogy, the pelvic muscles pull against the ligaments.

So if the suspensory ligaments are loose, the muscle strength weakens, and may not be able to keep the bladder or bowel emptying tubes closed. As a consequence of this, a patient may feel a leakage of urine, wind, or faces, "incontinence". Another related condition is failure to close the vaginal tube, so water may enter the vagina during swimming, or complain of vaginal flatus. If the damaged ligaments do not allow the muscles to open these same emptying tubes, a patient may have to strain to empty her bladder or bowel, "evacuation disorder" or "emptying disorder".[15]

## Pathophysiology of continence mechanism

The stress continence control system can be divided anatomically into 2 parts: (i) urethral support system and (ii) sphincteric closure system.[16] (Fig 8)

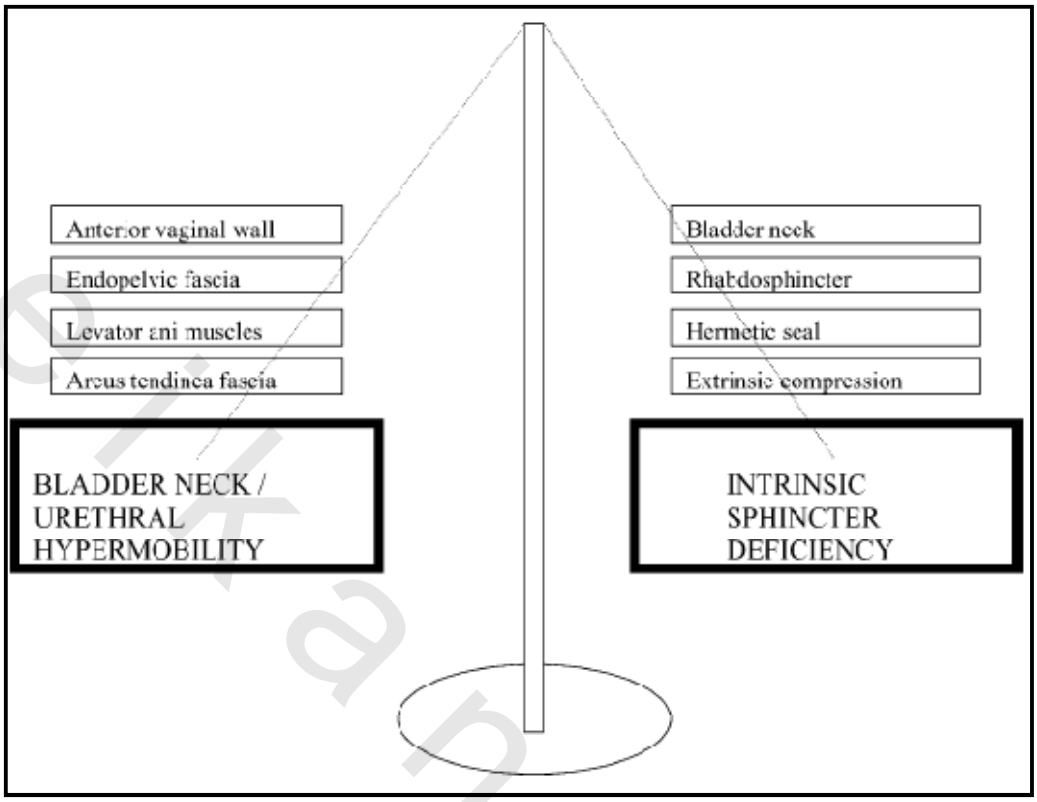


**Figure (8):** The stress continence control system.[16]

## Urethral support system

The urethral support system consists of the extrinsic structures that provide a supportive layer for the bladder neck and urethra. The major components are the vaginal wall, endopelvic fascia, arcus tendinea fascia pelvis (ATFP), levator ani muscle and the pubourethral ligament. The pressure transmission theory proposed by Enhorning [17] states that in continent women, the urethra resides in a normal intra-abdominal pressure zone. Inadequate supporting structures allow hypermobility of the bladder neck and urethra. If these lie below the pelvic floor, any increase in intra-abdominal pressure will be applied mostly to the bladder with a resultant rise in intravesical pressure only. Without the counterbalance of an equally increased intraurethral pressure Stress Urinary Incontinence (SUI) will result (Fig. 9). The pressure transmission theory remains popular and is supported by the success of bladder neck repositioning techniques. However, some authorities have cast doubt on the theory, not least Enhorning himself. The fact that retropubic operations are effective does not necessarily imply a pathophysiological mechanism. Many patients are continent despite large cystourethroceles where the bladder neck is permanently below the pelvic floor. Lin [18] showed no significant correlation between bladder neck hypermobility and SUI. Trans-vaginal Tape (TVT) procedures achieve high cure rates without elevating bladder neck or changing urethral hypermobility.

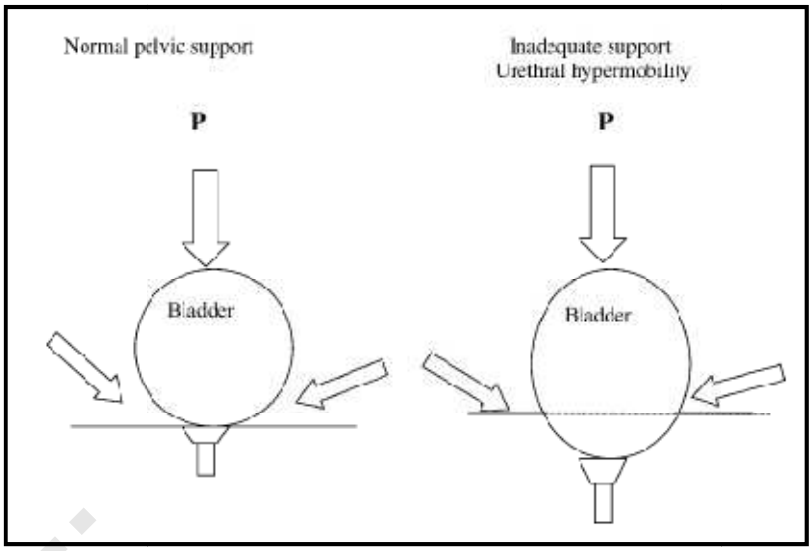


**Figure (9):** Pressure transmission theory and hypermobility.[16]

The Hammock Hypothesis proposed by DeLancey [19] describes the bladder neck and urethra as lying on a hammock-like structure composed of endopelvic fascia and anterior vaginal wall. The endopelvic fascia is draped over the arcus tendinea fascia pelvis. Also attached to this are the levator ani muscles. During a cough the hammock is tightened by contraction of the levators, which elevates the bladder neck and urethra and serves to resist the effects of increased intra-abdominal pressure (Fig. 10). Ashton-Miller et al. [20] has elaborated on this and described an interactive role of the hammock structures in maintaining pelvic support and continence. Together they provide pelvic floor ‘‘stiffness’’, i.e. the combined resistance to deformation which an increase in acting forces (intra-abdominal pressure) would otherwise cause. Stiffness will determine the degree of downward displacement of the urethra per unit increase in intra-abdominal pressure. Urethral orientation contributes to stress continence control. From its beginning at the internal urethral meatus, the urethra declines anteroinferiorly lying behind the pubic symphysis before passing between the pelvic floor muscles to reach the perineum. This almost horizontal orientation of the urethra means that dorsocaudal force leads to compression of the anterior wall of the urethra on the posterior wall, helping to close the lumen and prevent leakage. If this compressing force acts in the presence of inadequate urethral supports the effect is displacement of the urethra rather than opposition of its walls.[16]

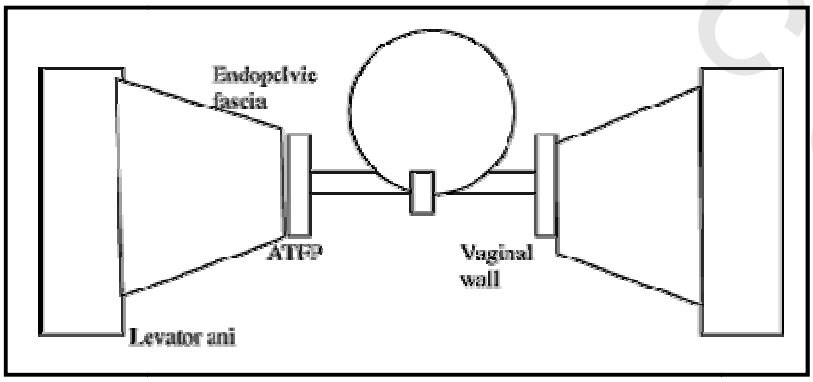


**Figure (10):** Hammock theory.[16]

## Sphincteric closure mechanism

The second anatomical part of the stress continence control system is the urethral sphincter closure mechanism. Debate surrounds the relative importance of the structural components but anatomists concentrate on the urethral striated muscle (rhabdosphincter), urethral smooth muscle, and vascular plexus. The contribution of each to the resting urethral closure pressure is unclear. What is apparent is that a discrete anatomical sphincter does not exist. Rather the combined inputs of the bladder neck, urethral musculature, the hermetic seal and the extrinsic compressive forces form a composite sphincter, play an integrated role in keeping the lumen of the urethra closed (Fig. 8). Bladder neck closure is thought to be passive due to its elastic component although the U-shaped loop of detrusor muscle surrounding the proximal urethra may constrict the lumen. Detrusor contraction causes funnelling and opening of the bladder neck. Chapple et al.[21] found that in 20% of nulliparous continent women the bladder neck was open at rest. These individuals probably maintain continence at the more distal sphincter. Gosling et al.[22] Suggest that the rhabdosphincter is the main contributor to urethral closure. Damage to the pudendal nerve during pregnancy and delivery may reduce the ability of this muscle to enhance urethral pressure during sudden increases in intra-abdominal pressure. Other nerves have been found running underneath the trigone to supply this muscle. These could be at risk of damage during surgery. Urethral smooth muscle consists of a thick inner longitudinal layer and an outer circular layer with attachments in the surrounding striated muscle layer. The exact role of this muscle is unclear but the circular layer may contribute to lumen closure when the layers contract. The urethral submucosa has a rich network of veins, elastic fibres and collagen, which fills the mucosal folds and allows formation of a seal. The importance of this seal is unclear but it is known to weaken with age and hypo-oestrogenism.[16]

## Integration of the stress continence control mechanism

It is clear that stress incontinence may be caused primarily by intrinsic sphincter deficiency and is not always due to inadequate urethral support. According to De Lancey,[23] 'the majority of women with stress incontinence have a combination of urethral dysfunction and support loss, and scientific study of these issues await insight into the quantification of each of these parameters as independent variables'. Stress continence is maintained by the integrated action of complex sub-systems which are ultimately under CNS control.

The principles underlying normal sphincteric function are integrated interactions among a number of factors:

1. Watertight apposition of the urethral lumen. Four urethral wall factors promote continence: (1) wall tension or external compression, (2) inner wall softness, (3) a filler material beneath the mucosa that helps to deform the mucosal folds into apposition, and (4) a lining of mucus provides the stickiness that enables cooptation of these mucosal folds. Histologic cross sections of the urethra show that the urethra is not simply a closed tube; rather, there are numerous mucosal folds, between which are potential spaces for urine leakage. In women, estrogen and a cushion effect of the submucosal vasculature have been suggested as ancillary factors.
2. Compression of the wall around the lumen. External compression of the urethral lumen is achieved by (1) smooth and striated muscle tone, (2) phasic contractions of the smooth and striated musculature, (3) elastic and viscoelastic properties of the extracellular matrix, (4) mechanical factors related to transmission of abdominal pressure, and (5) structural (anatomic) support of the posterior urethral wall.

3. Structural support to keep the proximal urethra from moving during increases in pressure.
4. A means of compensating for abdominal pressure changes (pressure transmission)
5. Neural control.[23]

## Combined hypermobility and intrinsic sphincter deficiency

Currently, there appears to be a shift away from this simple categorization of stress incontinence as being due either to hypermobility or ISD. This has arisen in part because of the development of the concept of Valsalva Leak Point Pressure (VLPP) and more recent analyses of long term results of stress incontinence surgery.

VLPP emerged as an alternative method to study urethral closure during stress for studies of urethral bulking with collagen. Investigators recognized that improvements in continence following urethral bulking did not correlate with urethral closure pressures, but did correlate with the amount of pressure required to produce leakage in the absence of intrinsic detrusor contraction. Although VLPP still lacks specific anatomic or theoretical grounding and many uncertainties related to standardization of recording methods and associated prolapse remain, low VLPP (without specified or established values) has been widely embraced as an indicator of ISD.

Just as the concept of VLPP blurred the previous distinction between simple ISD and simple hypermobility, long term outcome studies of correction of hypermobility have suggested that there may be more urethral weakness among patients with hypermobility than had been previously considered. Long term outcome studies of stress incontinence surgery have shown that there is a much greater failure rate of many of the commonly performed stress incontinence operations than had been generally appreciated, and that slings providing direct suburethral support seemed to give the greatest long term protection against recurrence of incontinence. Since slings had traditionally been the procedure of choice for recurrent incontinence or "Type III" (now ISD) incontinence, the possibility that ISD was more common than previously thought was more widely considered. Recently, Horbach and Ostergaard[24] have found that age is a significant, independent predictor of ISD in the setting of urodynamic stress incontinence, suggesting that age-related reduction in muscle mass, slowed reflexes or repeated episodes of prolapse may all contribute to the condition.[25]

## Causes of ISD in women

Several conditions are associated with increased risk for ISD in women:[26]

1. Previous urethral or periurethral surgery (e.g., antiincontinence surgery, urethral diverticulectomy) may result in postoperative ISD from periurethral fibrosis, scarring, or denervation. Prevalence of ISD after two or more failed anti-incontinence operations was found to be as high as 75%.[3]
2. Neurologic insult may cause ISD. Sacral neurologic lesions have a variable effect on micturition, depending on the extent to which the neurologic injury affects the parasympathetic, sympathetic, and somatic systems. In complete parasympathetic lesions, the bladder is areflexic and the patient is in urinary retention. When, in addition to a parasympathetic lesion, there is a sympathetic lesion, the proximal urethra loses its sphincteric function. Clinically, this results in incomplete bladder emptying, caused by the acontractile detrusor, and sphincteric incontinence, caused by the non-functioning proximal urethra. Somatic neurologic lesions affect pudendal afferent and efferent nerves. In addition to loss of perineal and perianal sensation, these lesions abolish the

bulbocavernosus reflex and impair the ability to contract the urethral and anal sphincters voluntarily. Sacral neurologic lesions are caused by herniated disks, diabetic neuropathy, multiple sclerosis, and spinal cord tumours. They are also commonly encountered after extensive pelvic surgery such as abdominoperineal resection of the rectum and radical hysterectomy.[3]

3. Pelvic radiation therapy has been associated with damage to the mucosal seal coaptation of the urethra and local neurologic damage.[26]

## Potential risk factors for developing UI

UI is a common symptom that may affect women at all ages, and there is a wide range of severity and nature of symptoms. UI is not a life-threatening disease, but the symptoms may seriously influence the physical, psychological, and social well-being of the affected individuals.

1. Pregnancy, labor, and vaginal delivery versus caesarean section are significant risk factors for later UI, but the strength of this association diminishes substantially with age.
2. Although several specific parturition factors such as instrumental delivery and birth weight are risk factors for UI in the postpartum period, their association with UI in later life is weak or nonexistent, suggesting that changes in birthing practices in developed countries are unlikely to affect UI in older age.
3. Additional evidence has now established body mass as important, modifiable risk factors for UI.
4. Physical function also appears to be an independent risk factor for UI in older women. Whether improvement in physical function leads to a reduction in UI remains to be established.
5. Evidence from two blinded, randomized controlled trials indicate that oral estrogen, with or without progestogen, is a significant risk factor for UI in women age 55 and older.
6. Diabetes is a risk factor for UI in most studies. Although diabetic neuropathy and/or vasculopathy are possible mechanisms by which diabetes could lead to UI, no mechanism has been established, nor is it clear whether prevention or treatment of diabetes, separate from weight reduction, will reduce the risk of UI.
7. Menopause, as generally defined, does not appear to be an independent risk factor for stress UI.
8. Hysterectomy remains a possible risk factor for later UI, but the evidence is inconsistent.
9. Moderate to severe dementia in older women is a moderate to strong independent risk factor for UI. Whether interventions to maintain or improve cognitive functioning also reduce UI has not been evaluated.
10. Mild loss of cognitive function in community-dwelling women, separated from physical function and other factors, increases the risk of UI slightly, if at all, but may increase the impact of UI.
11. Data from twin studies suggests that there is a substantial genetic component to UI.
12. Other potential risk factors including smoking, diet, depression, constipation, UTIs, and exercise, although associated with UI, have not been established as etiologic risk factors and are, in fact, difficult to study with observational data because of the potential for unmeasured confounding and questions of direction of the association.

Based on the literature available, it is not possible to unequivocally state the primary cause of stress urinary incontinence.[27]

# Clinical presentation and diagnostic evaluation

## 1. History

The general history should include questions relevant to precipitating and aggravating factors of urinary loss, time of onset and duration of symptoms, and degree of bother. Intrinsic sphincteric deficiency should be suspected based on the history and severity of the urinary incontinence. Patients with stress urinary incontinence related to intrinsic sphincteric deficiency usually present with severe incontinence, often occurring with minimal changes in position.[28]

## 2. Symptoms

Numerous questionnaires have been developed for assessment of health related quality of life (QOL). The fourth consultation of the International Continence Society (ICS) recommends that it will be preferable to use a questionnaire from the International Committee for Incontinence Questionnaire (ICIQ) modules. The ICIQ's international nature requires that linguistically validated translations are available. More than 50 language versions of various modules have been validated to date, conducted according to established protocol.

Another method to asses' severity of symptoms is the frequency-volume chart or micturition diary which records a patient's voiding pattern during normal daily activities. The ICS has described three different forms of diary, namely the micturition time chart which records the timing of voids in twenty four hours; the frequency volume chart (FVC) which also includes the volumes voided, and the bladder diary which in addition includes incontinence episodes, pad usage, fluid intake, degree of urgency and degree of incontinence. The optimum duration of recording depends on the clinical context and the purpose of the measurement. A properly performed 1-day FVC, which includes the first morning void the following day, is a reasonable tool to gain insight into voiding habits during normal daily routine. However, a 3-day FVC or diary is recommended for accurate assessment of lower urinary tract symptoms and for confirming a consistent clinical pattern in day to day practice.[29]

## 3. Physical examination

### a) General examination

Height and weight should be recorded so that body mass index can be calculated (Kg/M2); this has recently been shown to be a significant risk factor for incontinence.[30]

### b) Abdominal examination

Scars from previous surgery should be noted. Increased abdominal striae may be found in association with other markers of abnormal collagen metabolism, and are more likely in patients with prolapse and stress incontinence.

### c) Perineal/genital inspection

Inspection of the vulva and perineum allows a description of the skin and, for example, the presence of any abnormal anatomical features, of atrophy or excoriation, and erythema due to incontinence and the wearing of pads. The patient should be asked to cough and strain to demonstrate stress urinary incontinence and to observe urethral length, position, and mobility, and reflex contraction of the external anal sphincter.

**d)Vaginal examination**

It seems intuitive the examination should include an assessment of the bony architecture, pelvic floor muscle tone and muscle mass, connective tissue support, the epithelial lining of the vagina, the size, location, and mobility of the uterus, the adnexal structures, and innervation of the pelvic floor structures. The anterior, superior, and posterior segments of the vagina should be examined for pelvic organ prolapse.

**e)Special tests**

1. Cough Stress test: this is a provocative stress test (direct visualization) that can be performed by having the individual relax and then cough vigorously while the examiner observes for urine loss from the urethra. Optimally these tests should be done when the patient's bladder is half full, but they should not be performed when the patient has a precipitant urge to void. The test is usually performed initially in the lithotomy position, although if no leakage is observed, it should be repeated in the standing position, since the yield is increased when the test is repeated in the upright position. The spurt of urine should coincide with duration of cough. If leak continues after the cough, a cough induced bladder overactivity is probable.
2. Q tip (cotton swab) test: is a simple out-patient procedure to quantify bladder neck mobility. A sterile, lubricated cotton or Dacron swab (Q-tip) is inserted into the urethra until it lies just within the urethra-vesical junction. Using a protractor, the angle circumscribed by the distal end of the swab is measured relative to the horizontal plane while the woman is performing a maximum Valsalva effort. Urethrovesical junction hypermobility is defined by a maximum strain axis exceeding +30 degrees from the horizontal (Fig. 11).[31] A study investigating factors associated with severity of stress incontinence in women found that reduced urethral mobility using the Q-tip test was associated with greater severity of urinary incontinence.[32] Although intrinsic sphincteric deficiency has been associated with a fixed, well supported urethra, some patients with urethral hypermobility may also have significant compromise of urethral function.[28]

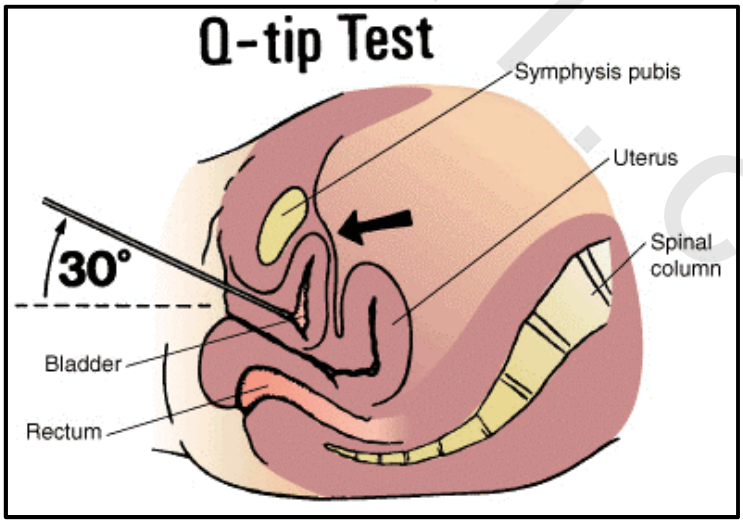


**Figure (11):** Q-tip test.[31]

**f) Investigations**

1. Urine analysis: is a fundamental test that should be performed in all urological patients. Although in many instances a simple dipstick urinalysis provides the necessary information, a complete urinalysis includes both chemical and microscopic analysis.
2. Ultrasound abdomen and pelvis with post void residual:
3. Urodynamics: Filling cystometry is done to confirm incontinence and its cause, define any detrusor activity during filling and assessment of degree of sphincter weakness. Urodynamic Stress Incontinence (USI) is the involuntary leakage of urine during increase abdominal pressure in the absence of detrusor contraction during filling cystometry. VLPP is preformed during cystometry, usually with bladder volume around 150-200 ml and repeated every 100 ml till the leak point is reached. Proponents of VLPP believe it is a measure of the severity of incontinence and it can differentiate ISD from urethral hypermobility. Commonly, 60cm$H_2O$ is the cut off below which ISD is diagnosed.

## Management

Anterior vaginal wall slings (AVWS) have been used for decades in the treatment of SUI. Initially pubovaginal slings using autologous fascia, either rectus fascia (Fig. 12)[33] or fascia lata, were the gold standards for treatment of SUI till the nineties.[34]

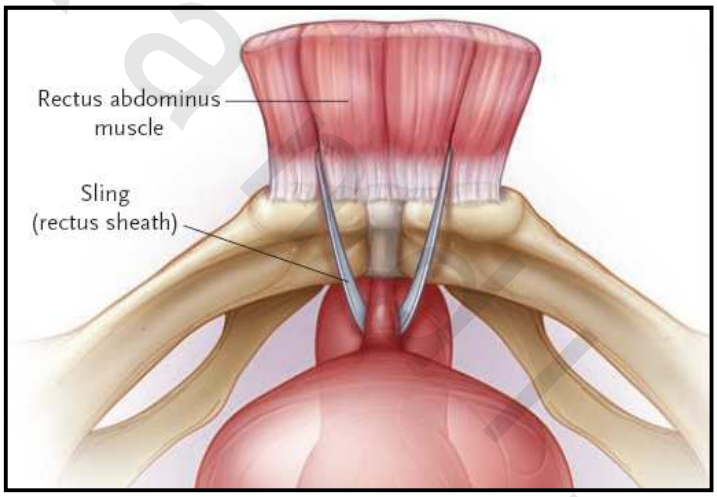


**Figure (12):** Pubovaginal sling using rectus fascia.[33]

In an effort to reduce the morbidity and discomfort associated with fascial harvesting, synthetic tapes placed by a retropubic or transobturator route were developed and have been applied successfully for over a decade. Tension free vaginal tape procedure may have an acceptable rate of complications such as infection, organ injuries and erosions, but they are costly.[35,36]

Recently, Trans Obturator Tape (TOT) has been used for the treatment of SUI patients along with Tension-Free Vaginal Tape (TVT, Fig. 13), but there have been only a few reports on its outcomes for SUI with ISD. O'Connor et al evaluated a group of patients with VLPP of less than 60 cm H2O who had TOT. They reported that the success rate of the group was only 25%, because the mesh tape, which was more horizontally placed, lacked support because it wrapped a smaller part of the urethra in comparison with that by TVT.[37]

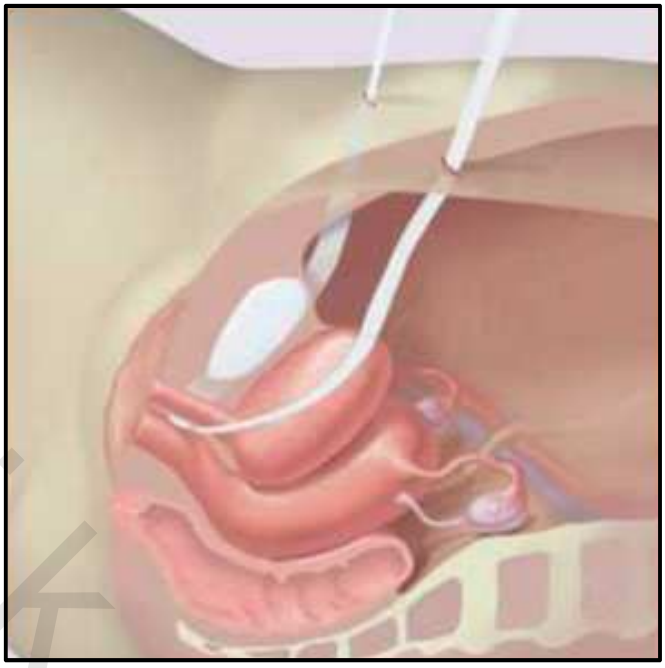

**Figure (13):** Tension free vaginal tape (TVT).[37]

The AVWS was originally described by Raz et al[38] in 1989. This technique uses in situ vaginal wall over the bladder neck and proximal urethra as the sling material (Fig. 14).[39] It avoids morbidity of autologous fascial harvesting, does not have an increased rate of infection or erosion and is available at no cost. The advantages are its simplicity, need for only a small incision, short operative time and hospital stay and reliance on healthy well vascularised in situ tissue.[38]

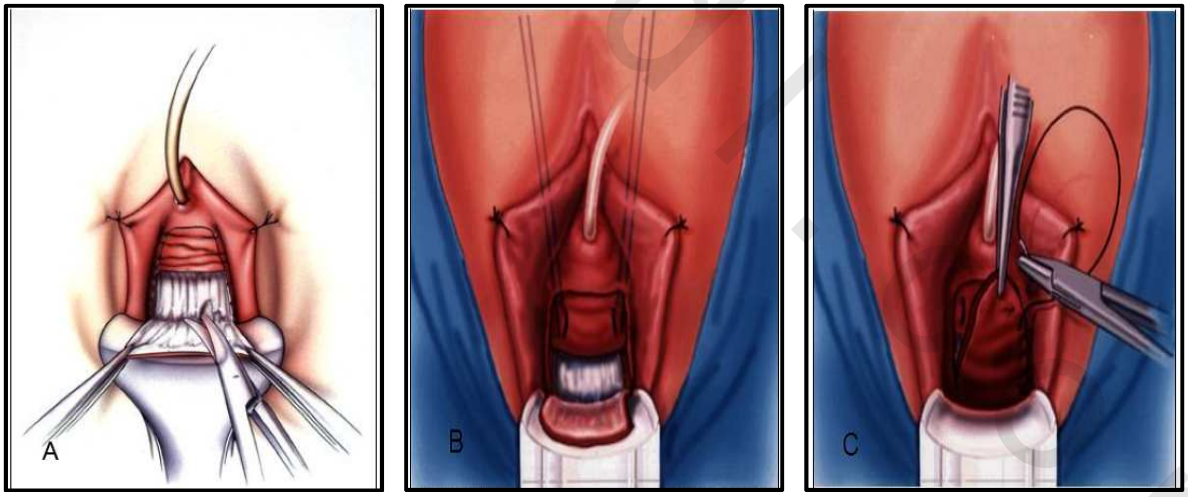


**Figure (14):** Insitu vaginal wall sling (ISS) technique. A: "block A" incision is made in anterior vaginal wall. B: Horizontal mattress suture is preformed, beginning behind sling and backing out proximally. C: Closure of sling entails closure of anterior vaginal wall over top of sling.[39]

A modified technique for the treatment of Stress Urinary Incontinence (SUI) in females is the Tension-Free Vaginal Flap (TVF) technique was described by Fayed et al.[40] It is based on both concepts of TVT and vaginal wall sling procedures, in order to achieve similar success rate with low costs. A vaginal wall flap based on the mid urethra was created. The flap is 4 cm wide and 2 cm long, with the distal incision slightly longer than the proximal one. Minimal dissection of the flap maintains its adequate blood supply. Four corners sutures 1/0 were taken into the flap (Fig. 15) and passed into the anterior abdominal wall using Stamey needle. Sutures of each side were tied to each other with undue tension, the knots were away from the rectus sheath by 1 cm.[40]

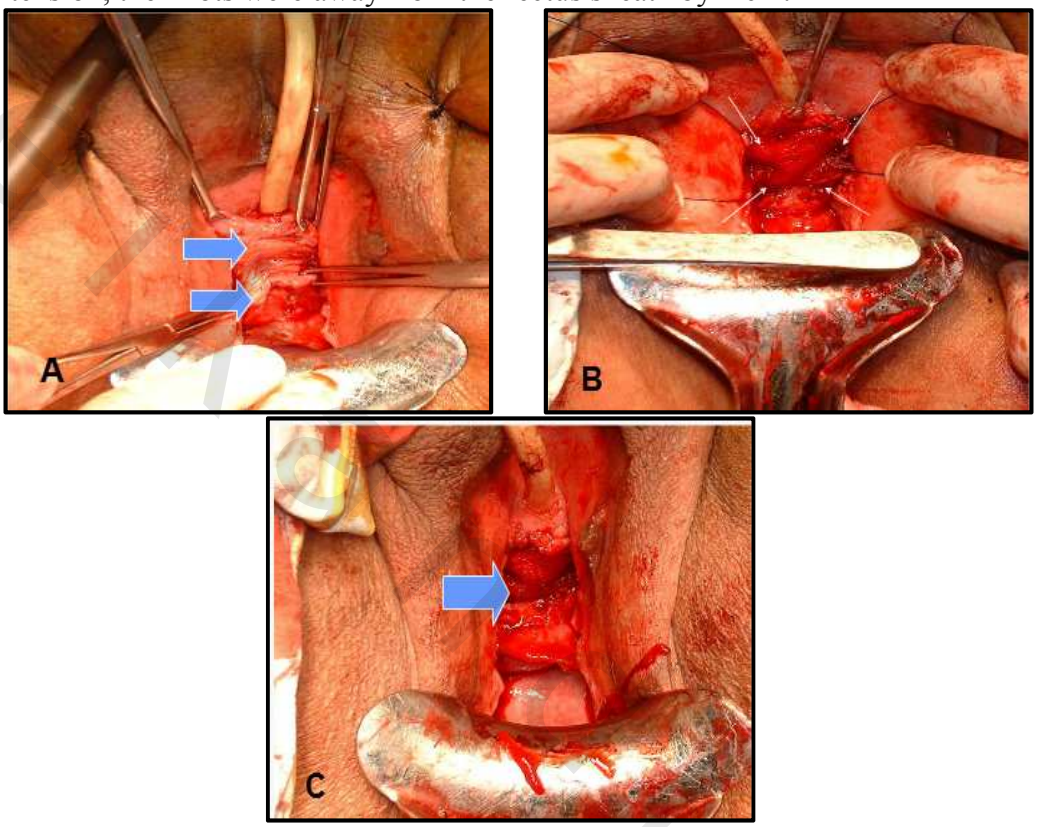


**Figure (15):** Tension-Free Vaginal Flap (TVF). A: Orientation of the flap. B: Sutures at the four angles of the flap. C: Preparation for closure of the anterior vaginal wall.[40]

The main drawback of using the vaginal wall as a sling is its tendency to stretch and weaken overtime. A modification of the TVF is the Rolled Fortified Vaginal Flap (RFVF)[41] which has been used in our study. The aim of this modification is to reinforce the vaginal wall flap and prevent its laxity over time to offer them an effective and durable alternative to the successful modern tapes.[41] In a previous study, RFVF showed encouraging results in both groups of patients with SUI due to ISD and urethral hypermobility.[41] This encouraged us to further study the RFVF in a larger group of patients with ISD.