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Urethro-Vesical Junction and Urethra in Relation to Stress Incontinence — a critical review

by

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The term stress incontinence was aptly introduced by Sir Eardley Holland in 1923 and in its present day concept implies an involuntary escape of urine with rise of intra-abdominal pressure. The patient is normally continent and maintains continence unconsciously. Micturition is normal neurophysiologically. Stress is mechanical, intermittent, often sudden; and common forms are coughing, laughing or sneezing. Stress is not constantly associated with incontinence, at least in the early cases; in these patients stress incontinence occurs when the bladder is relatively full. Incontinence is of short duration, often limited to the early phase of the sudden stress. Voluntary abruption of flow is always possible and with warning of stress, incontinence may be entirely prevented. Stress incontinence is usually progressive. Only if voluntary efforts at continence become strong and constant does stress incontinence appear self limiting. Stress incontinence is largely a subjective symptom. It is known to occur in 40-50 percent of normal nulliparous women (Kellar, 1960) and in most of them is observed so very occasionally and transiently that it is not volunteered as a symptom.

Anatomy

A discussion of factors contributing to continence of urine is best introduced by a description of the anatomy of the urethro-vesical junction and urethra. The adult female urethra is about 4 cm. long. From the bladder it follows a slightly curved course downwards and forwards, behind the lower half and lower border of the symphysis pubis. It ends in the vestibule, immediately in front of the opening of the vagina. In its course it passes through the pubo-cervical fascia which is an anterior extension of the endopelvic fascia. The pubococcygeus muscle runs lateral to the urethra just below the level of the pubocervical fascia.

Bailey (1954) described 2 connections between pubococcygeus muscle and urethrovesical junction.

1. From the fascia covering the medial fibres of the pubococcygeus muscle, a tendinous slip crosses over to the superolateral aspect of the bladder neck. This fascia also covers and blends with the outer coat of the proximal urethra.

2. Some muscular slips gain attachment to the inferolateral aspect of the bladder neck.

The existence of well defined connections between the pubococcygeus and bladder neck is not generally accepted. One may, in general terms, comment that where a voluntary mechanism welds with an involuntary one, voluntary muscles gain attachments, directly or indirectly, to involuntary muscles; *e.g.* junction of pharynx with oesophagus, junction of rectum and levator ani; likewise junction of female urethra and bladder neck with pubococcygeus muscle.

The urethra then passes through the triangular ligament consisting of 2 layers of fascia stretched between the pubic rami. Between these layers is the sphincter urethrae muscle which surrounds the urethra, and deep transversus perinei muscles. These muscles are not well developed in the female. From the upper surface of the triangular ligament lateral bands of

the pubic ramus. Below the triangular ligament the bulb of the vestibule and bulbo-spongiosus muscle form the lateral relations of the urethra.

The proximal urethra is relatively mobile and displaceable compared to the distal portion which, piercing the triangular ligament, is firmly adherent to the anterior vaginal wall.

The mucosa of the urethra is lined by transitional epithelium in its upper two-thirds and by stratified squamous epithelium in its lower one-third. It is thrown into longitudinal interlocking folds. A well developed venous plexus exists in the submucosa. The muscular layer of the urethra consists almost entirely of involuntary muscle which continues from the bladder. Many slips of voluntary muscle fibres investing the smooth muscle coats have been described and disproved; most of them were too puny to have any distinct physiological function.

The arrangement of the smooth muscular coats of the urethra is subject to considerable dispute. Varying patterns have been described, supporting the particular author's views on functional anatomy of the urethra. It has long been established that the urethra is empty during the resting state and that urine remains contained in the bladder. From this observation rose a concept that control of micturition required a bladder for a reservoir, a sphincter at the urethrovesical junction to contain urine in the bladder and urethra for a conduit. Von Ludlinghausen (1932) thus described a complicated form of decussation of the muscular coats as they continued into the urethra, resulting in interlocked anterior and posterior slings. Each sling pulled in the opposite direction, exerting a sphincteric action. If this was so, the detrusor would have to be in a continued state of contraction, relaxing during micturition to release the pull on the loops and so open the sphincter. This is in direct contradiction to what we know now of detrusor action (Muellner, 1959).

Assiduos dissection by Krantz (1951) and Woodburne (1960) has not revealed the presence of a sphincter at the urethro-vesical junction. Urethro-vesical pressure studies by Hodgekinson (1963), cineradiographic studies by Gardiner et al (1961) and other investigators support the idea that no functional sphincter as such exists at the urethro-vesical junction. Lapides (1958) regarded the urethra as a simple canal with its smooth muscle coats arranged as a thin inner longitudinal layer and a thick outer circular layer. Langreder (1956) suggested a spiral arrangement of the muscular coats: when closely packed they appear as circular fibres and when drawn out as oblique or lingitudinal fibres.

Sphincterometrographic findings of Youssef and Mahfouz (1956) convinced them of the presence of a sphincter at the urethro-vesical junction. Jeffcoate (1961) stated dogmatically that the involuntary muscle is so arranged at the urethro-vesical junction as to provide a functional sphincter, although an anatomical sphincter cannot be demonstrated.

Physiology

Much of the information on the maintenance of continence of urine in women has been gathered from observations made on patients with stress incontinence. A few of the postulated causes of stress incontinence may therefore be reviewed.

Displacement of Urethro—Vesical Junction

In normal continent women, the maximal downward thrust of the intra-abdominal pressure falls just behind the urethro-vesical junction (Hogekinson, 1963). Displacement backwards and downwards of the urethro-vesical junction to this region may result in stress incontinence. Low (1964) found that the urethro-vesical junction of normal nulliparous women was located within a rectangle 3.0 cm. by 1.5 cm. behind the lower half and inferior border of the symphysis pubis. Only in 14 percent of his patients with stress incontinence without prolapse was the urethro-vesical junction confined to this area. Inspite of Low's findings the spatial relationships of the urethro-vesical junction and proximal urethra are not directly related to either the incidence or severity of stress incontinence. The significant features predisposing to stress incontinence are the positioning of the urethro-vesical junction in the line of maximum hydrostatic thrust of intra-abdominal pressure and upset in the normal relationships of the urethro-vesical junction with the proximal urethra. There is no simple relationship between

fibrous tissue, called pubo-urethral ligaments, anchor the urethra anteriorly and laterally to utero-vaginal prolapse and stress incontinence (Jeffcoate, 1961).

The urethro-vesical junction and proximal urethra are supported, to a varying extent, by adjacent structures and, of course, indirectly by muscular and fibrous elements constituting the pelvic floor. Displacement may result from weakening of these supports and of the intrinsic muscular and fibro-elastic structures of the urethro-vesical junction and proximal urethra. Comparative anatomic studies show the peculiarly directed downward thrust of intra-abdominal pressure in the upright woman. Stress incontinence is more readily elicited with the patient in the upright position than when she is lying down.

Funnelling

Funnelling of the urethro-vesical junction has been described as a cause of stress incontinence. The bladder urine becomes a dilating hydraulic wedge and continually strains the apex formed by the urethro-vesical junction. The weakened urethro-vesical junction is subsequently unable to maintain continence during stress. Changes in a cystocele involving the urethro-vesical junction do not constitute funnelling. A larger and more rounded herniation occurs and more evenly supports increases in intravesical pressure. If during repair of a cystocele, funnelling of the urethro-vesical junction is produced or the urethro-vesical junction is so manipulated as to bring it to the environment of maximal intra-abdominal pressure, stress incontinence may result. Funnelling may result from reduced tone in the involuntary musculature of urethro-vesical junction and proximal urethra. Weakness of intrinsic "voluntary" muscle fibres of the proximal urethra may also play a significant part (Youssef, 1956).

Urethro—Vesical Angles

The urethra joins the bladder to form anterior and posterior urethro-vesical angles as evident on lateral urethro-cystographs. Thomsen (1932) believed that the loss of the anterior urethro-vesical angle caused stress incontinence. Jeffcoate and Roberts (1952) could not substantiate this and through their efforts the posterior urethro-vesical angle became famous. Lateral urethro-cystographic studies led them to conclude that:—

i) the inactive normal female urethra is straight;

ii) the base of the quiet bladder is flat or has a slight convexity downwards;

iii) the urethro-vesical junction is clearly defined and funnelling is normally absent;

vi) continence is ensured by sphincteric mechanism in the proximal urethra and a well formed posterior urethro-vesical angle is a sign of a competent sphincter. The posterior urethra-vesical angle is normally around 100 degrees. (During micturition it becomes more than 140 degrees.)

Jeffcoate and Roberts found exaggeration or loss of posterior urethro-vesical angle in 80 percent of their cases of stress incontinence. Restoration of the posterior urethro-vesical angle was associated with successful treatment.

Forward displacement of the bladder from retrovesical pressure (*e.g.* ventrisuspended uterus, anterior wall fibromyoma uteri) may cause virtual elimination of the bladder segment posterior to the coronal plane through the urethro-vesical junction. This alters the posterior urethro-vesical angle and may cause stress incontinence.

Ardran et al (1956) were unable to corelate alterations in the urethro-vesical angles with symptoms of stress incontinence. Other workers have not noticed the obliteration of the posterior urethro-vesical angle in patients with stress incontinence as consistently as the Jeffcoate school. Hodgekinson et al (1958) using a metallic-bead chain to outline the urethra showed that the proximal urethra was far from being a straight tube. The downward and backward rotation of the bladder base below the level of the urethro-vesical junction appears to produce a good posterior urethro-vesical angle. But this position also moves the urethro-vesical junction away from the maximal thrust of intra-abdominal pressure. It is not the angle

that requires correction for cure of stress incontinence. The factors that contribute to the posterior urethro-vesical angle need to be repaired.

Sphincterometrography

Youssef and Mahfouz (1956) by sphincterometrographic studies demonstrated that descent of the Miller-Abbot tube balloon (they used a Miller-Abbot tube with its balloon replaced by one made of very thin elastic rubber) below the bladder base during increases of intra-vesical pressure was a feature of stress incontinence. They concluded that this finding carried the same significance as the exaggeration of the posterior urethro-vesical angle on straining, namely: evidence of a weakened sphincteric mechanism at the urethro-vesical junction. They regarded the integrity of this sphincteric mechanism as by far the most important factor in urinary continence. Their findings have not been enthusiastically received. Probably too many factors in their experiments contributed to a not entirely physiological situation.

Rotational descent of urethra

The maintenance of the posterior urethro-vesical angle has been variously described as a concerted effort of:—

i) the intrinsic smooth musculature of the urethro-vesical junction and proximal urethra;

ii) the so-called voluntary muscle fibres of the urethra, and

iii) the extrinsic supports.

Bailey (1954) observed that the extrinsic supero-lateral tendinous supports of the urethro-vesical junction and proximal urethra helped maintain the 60-70 degrees upward incline of the urethra; the infero-lateral muscular supports helped maintain the configuration of the urethro-vesical junction posteriorly, including the posterior urethro-vesical angle. In cases of utero-vaginal prolapse, downward and backward rotational descent of the proximal urethra was a relatively common finding when stress incontinence was also present. He recommended that the rotational descent of the proximal urethra needed correction to cure stress incontinence. Rotational descent of the proximal urethra is to be expected in cases of cystourethrocele; the mobile proximal urethra, as opposed to the relatively fixed lower portion, moves with the herniating bladder. The urethro-vesical relationships need, however, not change and stress incontinence is not an inevitable result of rotational descent.

Intra—urethra pressure

A great deal of attention has of late been focussed on intra-urethral pressure, especially as a definite sphincteric mechanism has not been conclusively demonstrated at the urethro-vesical junction. It has long been observed that when micturition is voluntarily interrupted no sphincteric mechanism comes to play at the urethro-vesical junction. At the end of micturition, the proximal urethra contains urine which returns to the bladder, unimpeded by any sphincter at the urethro-vesical junction. Ardran et al (1956) noted that the proximal urethra may dilate with urine during straining even in symptomless women. These and other observations have extended the emphasis from urethro-vesical junction and proximal urethra to include the entire urethra, in concepts of urinary continence.

As the bladder fills, receptive relaxation of the bladder musculature precludes spikes of intra-vesical pressure. At rest with 200 ml. fluid in the bladder, the intra-vesical pressure is about 10 cm. water (Roberts, 1953) and is nearly equal to the intra-abdominal pressure. Lapides et al (1960) measured the resistance of the female urethra to retrograde flow. They found:—

Resistance at	
urethrovesical junction	28 cm. water pressure
upper-mid urethra	46 cm. ,, ,,
lower-mid urethra	51 cm. ,, ,,
lower urethra	21 cm. ,, ,,

In normal continent women this intra-urethral pressure more than adequately overcomes the intra-vesical pressure. When the intra-urethral pressure is reduced, rises of intra-vesical pres-

sure with stress may produce incontinence. The inherent resistance in the urethral wall contributing to intra-urethral pressure includes:—

i) tone of the urethral musculature;

ii) tissue resistance of fibro-elastic tissue in the urethral wall and of the interlocking longitudinal mucosal folds;

iii) flow and volume of blood in the submucous venous plexuses, this producing a washer like effect.

The extrinsic supports of the urethro-vesical junction and urethra probably help reduce the distensibility of smooth muscle of urethra; this helps in maintaining tone of the urethral musculature.

As shown by Lapides et al the intra-urethral pressure is highest at mid-urethral level; this is probably due to the more intimate connection with the triangular ligament and voluntary sphincter urethrae, offering greater resistance. The external sphincter mechanism is helpful in voluntarily raising intra-urethral pressure. Weakness of this sphincter does not however produce stress incontinence. Excision of the distal portion of the urethra, including the sphincter urethrae muscle, is compatible with perfect continence. In patients with severe utero-vaginal prolapse, even with changes in the urethro-vesical junction predisposing to stress incontinence, kinking of the urethra may provide the necessary intra-urethral pressure to maintain continence during stress.

Intra—Abdominal pressure

Enhorning (1960) with electronic urethrocystometric studies demonstrated that, with the urethra in its anatomical position, the pressures in the proximal urethra and bladder are similarly affected by changes in intra-abdominal pressure. He suggested that, in this respect, the bladder and proximal urethra should be considered intra-abdominal organs. Therefore, the intra-urethral pressure which is higher than the intra-vesical pressure at rest, would continue to be higher with rises of intra-abdominal pressure. Changes in urethro-vesical relationships and position or other mechanisms may result in differential transmission of intra-abdominal pressure on bladder and proximal urethra, thus predisposing to stress incontinence. This hypothesis appears quite feasible and certainly merits repeated evaluation.

Urethral length

Lapides et al (1960) measured urethral length with calibrated Foley's catheter method and noted that actual or functional shortening of the urethra below 30 mm. was relatively common in patients with stress incontinence. On the basis of the Law of LaPlace they rationalised that the length of the urethra was important in maintaining adequate intra-urethral pressure. The law states that the pressure extended upon the wall of a tube varied directly with the inherent tension in its wall and with its length and varied inversely with the radius of its lumen.

Shortening of the urethra cannot accurately be measured by means of a Foley's catheter; telescoping of a flaccid urethra over the distended balloon of the catheter is a distinct possibility. Using a metallic-bead chain technique Hodgekinson (1958) concluded that shortening of the urethra was indeed difficult to assess. Many investigatiors using other methods could not prove a relationship between shortening of the urethra and stress incontinence. It seems logical to state that the complicated factors involved in continence of urine cannot be readily explained by the mathematical simplicities of LaPlace Law. Even in the field of engineering where this law is used, it is not applicable in all cases.

Dilated urethral lumen

Stoeckl (1962) at urethroscopy of 43 patients with stress incontinence found combinations of lax internal urethral meatus, wide proximal urethra, sagging posterior urethral wall, in 35 patients. Urethroscopic findings are, however, not accurate enough to warrant serious thought. Low (1964) found that the urethral lumen at the urethro-vesical junction in symptomless women was 1.3 to 1.6 mm. In patients with stress incontinence it ranged from 3.5 to 4.2 mm. The diameter of the lumen at other levels was also higher in patients with stress incontinence. Enlarged urethral lumen signifies

decreased intra-urethral pressure and probably results from impaired functional efficiency of urethral musculature. Urethrocele, reflecting poor support of the urethro-vesical junction and urethra, appears to be commonly associated with stress incontinence. Beck (1964) found that urethrocele or cystourethrocele were associated with a 90% incidence of stress incontinence. Urethral diverticula have also been associated with stress incontinence.

Urethral Distortion

Fibrous rigidity, scarring, distortion and fixation of the urethral wall prevent proper closure of the urethra and could cause stress incontinence. These changes follow trauma, especially, repeated operative trauma. Emphasising on free pliability as opposed to fixation of the urethra for adequate functioning, Mulvany (1951) freed the urethro-vesical junction and proximal urethra from peri-urethral and extra-vesical tissues. Stabler (1952) conceding the importance of pliability of the urethra, commented, however, that Mulvany's procedure resulted in new adhesions supporting the urethro-vesical junction in a more favourable position thus curing stress incontinence.

Pregnancy

Francis (1960) demonstrated that stress incontinence of urine occurred more commonly during pregnancy than is anticipated. The loss of the posterior urethro-vesical angle was also significant. 30 out of 33 pregnant women who complained of stress incontinence showed loss of posterior urethro-vesical angle. In pregnancy the centripetal lines of transmission of intra-abdominal pressure alter in direction, especially as pregnancy advances. More significantly, the urethro-vesical relationships may alter. On the other hand, the intra-urethral pressure may rise due to the slight elongation of the urethra, increased fluid and tension in the urethral tissues,hypertrophy of the epithelial folds and increased blood flow along the submucous venous plexus. The increased hormonal levels may contribute to the laxity and reduction of tone in the urethro-vesical junction and proximal urethra or may operate in some unknown manner. Obviously very little is known of the exact anatomical and physiological changes in pregnancy which contribute to stress incontinence of urine.

Conclusions

From the foregoing studies certain conclusions may be made.

a) The urethro-vesical junction and proximal urethra are directly concerned in continence of urine. They are the key structures.

b) The fibrous and voluntary muscular structures in direct relationship to the urethro-vesical junction and proximal urethra serve as important supports. They are complementary in maintaining intra-urethral pressure and proper anatomical position.

c) The presence of a sphincteric mechanism at the urethro-vesical junction has not been conclusively established.

d) The voluntary sphincter mechanism contained in the triangular ligament can be called upon to play when due warning of stress is given. It serves as a second line of defence.

e) When intra-vesical press ure equals or exceeds intra-urethral pressure stress incontinence results.

f) Weakening of the urethral structures and their supports results in reduced capacity to maintain the required intra-urethral pressure.

g) If, by any form of treatment, the increased static pressure and distortion could be removed, and the structures adequately supported in the optimal anatomic position and shape, and sufficient mobility ensured, then the tone, muscular strength and elasticity of the urethro-vesical junction and proximal urethra could be restored. Adequate physiological activity could then be expected.

Though much remains to be learnt, our present state of knowledge does not justify the apologetic complacency that goes with only

80-85 percent operative cure of stress incontinence. Careful preoperative assessment of the anatomical and physiological changes, proper selection of type of operation should certainly contribute towards increasing the cure rate.

Summary

The clinical concept of stress incontinence has been outlined. Anatomy of the urethro-vesical junction and urethra has been described. The diverse theories on the arrangement of muscle fibres at the urethro-vesical junction and proximal urethra illustrate the incompleteness of our knowledge. The influence of the urethro-vesical relationships. and the position and configuration of the urethro-vesical junction on the production of stress incontinence has been critically reviewed. The significance of intra-urethral pressure and factors contributing to it have been justly emphasised.

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