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Functional Anatomy of the Female Pelvic Floor

James A. Ashton-Miller and John O.L. DeLancey

OUTLINE

INTRODUCTION

The anatomic structures that prevent incontinence during elevations in abdominal pressure are primarily sphincteric, augmented secondarily by musculofascial supportive systems. In the urethra, for example, the action of the vesical neck and urethral sphincteric mechanisms at rest constrict the urethral lumen and keep urethral closure pressure higher than bladder pressure. The striated urogenital sphincter, the smooth muscle sphincter in the vesical neck and the circular and longitudinal smooth muscle of the urethra all contribute to this closure pressure. In addition, the mucosal and vascular tissues that surround the lumen provide a hermetic seal via coaptation, aided by the connective tissues in the urethral wall. Decreases in the number of striated muscle sphincter fibres occur with age and parity, but changes in the other tissues are not well understood.

A supportive hammock under the urethra and vesical neck provides a firm backstop against which the urethra is compressed during increases in abdominal pressure to maintain urethral closure pressures above the rapidly increasing bladder pressure. This supporting layer consists of the anterior vaginal wall and the connective tissue that attaches it to the pelvic bones through the pubovaginal portion of the levator ani muscle and the uterosacral and cardinal ligaments comprising the tendinous arch of the pelvic fascia.

At rest the levator ani acts to maintain the urogenital hiatus closed in the face of hydrostatic pressure due to gravity and slight abdominal pressurization. During the dynamic activities of daily living the levator ani muscles are additionally recruited to maintain hiatal closure in the face of inertial loads related to having to decelerate caudal movements of the viscera as well as the additional load related to increases in abdominal pressure resulting from activation of the diaphragm and abdominal wall musculature.

Urinary incontinence is a common condition in women, with prevalence ranging from 8.5% to 38% depending on age, parity and definition (Thomas et al., 1980; Herzog et al., 1990). Most women with incontinence have stress urinary incontinence (SUI), not

infrequently with urge incontinence (Diokno et al., 1987). Both types of incontinence are primarily due to an inadequate urethral sphincter which develops too little urethral closure pressure to prevent urine leakage (DeLancey et al., 2008, 2010). Usually this is treated using conservative therapy or, if that fails, then surgery. Despite the common occurrence of SUI, there have been few advances in our understanding of its cause in the past 40 years. Most of the many surgical procedures for alleviating SUI involve the principle of improving bladder neck support (Colombo et al., 1994; Bergman & Elia, 1995). Treatment selection based on specific anatomic abnormalities has awaited identification, in each case, of the muscular, neural and/or connective tissues involved.

Understanding how the pelvic floor structure/function relationships provide bladder neck support can help guide treatment selection and effect. For example if, while giving vaginal birth, a woman sustains a partial tear of a portion of her pelvic muscles that influence her continence, then pelvic muscle exercises may be effective.

On the other hand, if portions of those muscles are irretrievably lost, for example due to complete and permanent denervation, then no amount of exercising will restore them; pelvic muscle exercises may well lead to agonist muscle hypertrophy, but whether or not this will restore continence will depend upon whether the agonist muscles can compensate for the lost muscle function.

This chapter reviews the functional anatomy of the pelvic floor structures and the effects of age on urethral support and the urethral sphincter, and attempts to clarify what is known about the different structures that influence stress continence. This mechanistic approach should help guide research into pathophysiology, treatment selection and prevention of SUI. In addition, we also review the structures that resist genital prolapse because vaginal delivery confers a 4- to 11-fold increase in risk of developing pelvic organ prolapse (Mant et al., 1997).

HOW IS URINARY CONTINENCE MAINTAINED?

Urethral closure pressure must be greater than bladder pressure, both at rest and during increases in abdominal pressure, to retain urine in the bladder and prevent leakage. The resting tone of the urethral muscles maintains a favorable pressure relative to the bladder when urethral pressure exceeds bladder pressure. The primary factor that determines continence is the maximum urethral closure pressure developed by the urethral sphincter (DeLancey et al., 2008, 2010).

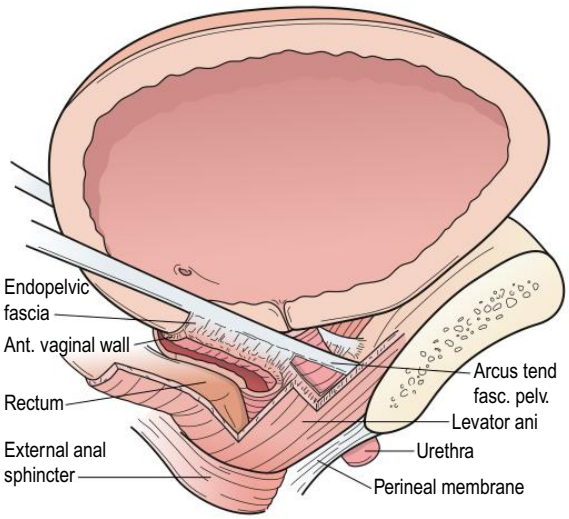

Fig. 3.1 Lateral View of the Components of the Urethral Support System. Note how the levator ani muscles support the rectum, vagina and urethrovesical neck. Also note how the endopelvic fascia beside the urethra attaches to the levator ani muscle; contraction of the levator muscle leads to elevation of the urethrovesical neck. Puborectalis muscle is removed for clarity. (Redrawn from DeLancey 1994, with permission of C V Mosby Company, St Louis. DeLancey)

During activities such as coughing, when bladder pressure increases several times higher than urethral pressure, a dynamic process increases urethral closure pressure to enhance urethral closure and maintain continence (Enhörning 1961). Both the magnitude of the resting closure pressure in the urethra and the increase in abdominal pressure generated during a cough determine the pressure at which leakage of urine occurs (Kim et al., 1997).

Although analysis of the degree of resting closure pressure and pressure transmission provides useful theoretical insights, it does not show how specific injuries to individual component structures affect the passive or active aspects of urethral closure. A detailed examination of the sphincteric closure and the urethral support subsystems (Fig. 3.1) is required to understand these relationships.

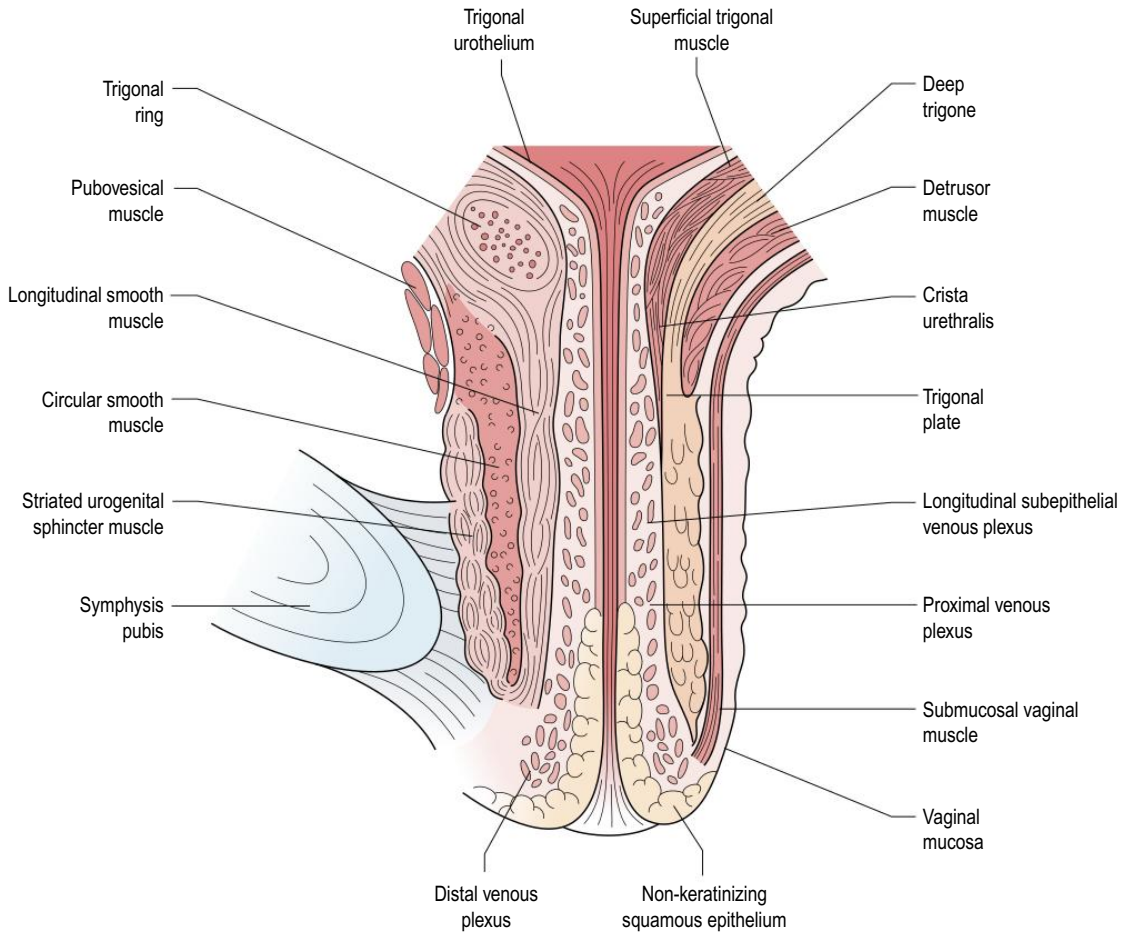

Fig. 3.2 Midsagittal section showing the anatomy of the urethra. (DeLancey)

The dominant element in the urethral sphincter is the striated urogenital sphincter muscle, which contains a striated muscle in a circular configuration in the middle of the urethra and strap-like muscles distally. In its sphincteric portion, the urogenital sphincter muscle surrounds two orthogonally-arranged smooth muscle layers and a vascular plexus that helps to maintain closure of the urethral lumen.

THE URINARY SPHINCTERIC CLOSURE SYSTEM

Sphincteric closure of the urethra is normally provided by the urethral striated muscles, the urethral smooth muscle and the vascular elements within the submucosa (Figs 3.2 and 3.3) (Strohbehn et al., 1996; Strohbehn & DeLancey, 1997). Each is believed to contribute equally to resting urethral closure pressure (Rud et al., 1980).

Anatomically, the urethra can be divided longitudinally into percentiles, with the internal urethral meatus representing point 0 and the external meatus representing the 100th percentile (Table 3.1). The urethra passes through the wall of the bladder at the level of the vesical neck where the detrusor muscle fibres extend below the internal urethra meatus to as far as the 15th percentile.

The striated urethral sphincter muscle begins at the termination of the detrusor fibres and extends to the 64th percentile. It is circular in configuration and completely surrounds the smooth muscle of the urethral wall.

Starting at the 54th percentile, the striated muscles of the urogenital diaphragm, the compressor urethrae and

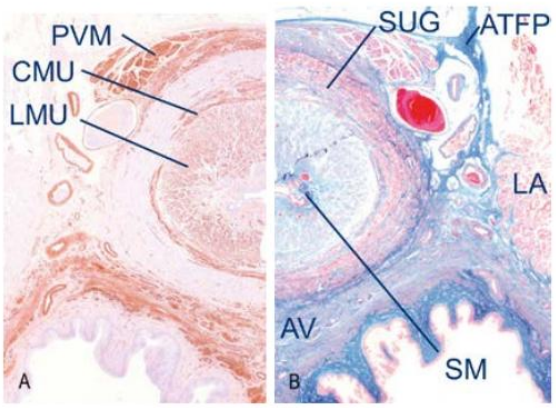

Fig. 3.3 Transverse Histologic Section of the Mid-Urethra of a 21-Year-Old Woman. (A) Structures are visualized using a sigma-actin smooth muscle stain, which shows the pubovesical muscle (PVM), the circumferential smooth muscle (CMU) layer, and the longitudinal smooth muscle (LMU) layer. (B) The contralateral side is stained with Masson's trichrome to show the arcus tendineus fascia pelvis (ATFP), the striated urogenital sphincter (SUG), the levator ani (LA), the anterior vaginal wall (AV), and the submucosa of the urethra (SM). (From Strohbehn et al., 1996, with permission of Lippincott Williams & Wilkins, Baltimore, MD.)

the urethrovaginal sphincter can be seen. They are continuous with the striated urethral sphincter and extend to the 76th percentile. Their fibre direction is no longer circular. The fibres of the compressor urethrae pass over the urethra to insert into the urogenital diaphragm near the pubic ramus.

The urethrovaginal sphincter surrounds both the urethra and the vagina (Fig. 3.4). The distal terminus of the urethra runs adjacent to, but does not connect with, the bulbocavernosus muscles (DeLancey 1986).

Functionally, the urethral muscles maintain continence in various ways. The U-shaped loop of the detrusor smooth muscle surrounds the proximal urethra, favoring its closure by constricting the lumen.

The striated urethral sphincter is composed mainly of type 1 (slow-twitch) fibres, which are well suited to maintaining constant tone as well as allowing voluntary increases in tone to provide additional continence protection (Gosling et al., 1981). Distally, the recruitment of the striated muscle of the urethrovaginal sphincter and the compressor urethrae compress the lumen.

The smooth muscle of the urethra may also play a role in determining stress continence. The lumen is surrounded by a prominent vascular plexus that is believed to contribute to continence by forming a watertight seal

TABLE 3.1 Urethral topography and urethral and paraurethral structures

Percentile of urethral length	Location: Region of the urethra	Structures
0–20	Intramural	Internal urethral meatus Detrusor loop
20–60	Mid-urethra	Striated urethral sphincter muscle Smooth muscle
60–80	Urogenital diaphragm	Compressor urethrae muscle Urethrovaginal sphincter Smooth muscle
80–100	Distal urethra	Bulbocavernosus muscle

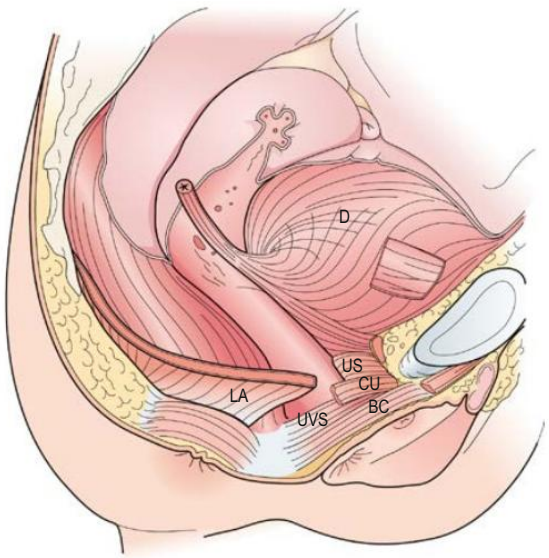

Fig. 3.4 Lateral View of Urethral and Pelvic Floor Muscular Anatomy. BC, bulbocavernosus; CU, compressor urethrae; D, detrusor; LA, levator ani; US, urethral sphincter; UVS, urethrovaginal sphincter. Puborectalis muscle is removed for clarity. (DeLancey)

via coaptation of the mucosal surfaces. Surrounding this plexus is the inner longitudinal smooth muscle layer. This in turn is surrounded by a circular layer, which itself lies inside the outer layer of striated muscle.

The smooth muscle layers are present throughout the upper four-fifths of the urethra. The circular

configuration of the smooth muscle and outer striated muscle layers suggests that the contraction of these layers has a role in constricting the lumen. The mechanical role of the inner longitudinal smooth muscle layer is presently unresolved. Contraction of this longitudinal layer may help to open the lumen to initiate micturition rather than to constrict it.

CLINICAL CORRELATES OF URETHRAL ANATOMY AND EFFECTS OF AGING

There are several important clinical correlates of urethral muscular anatomy. Perhaps the most important is that SUI is caused by problems with the urethral sphincter mechanism as well as with urethral support. Although this is a relatively new concept, the supporting scientific evidence is strong.

The usual argument for urethral support playing an important role in SUI is that urethral support operations cure SUI without changing urethral function. Unfortunately, this logic is just as flawed as suggesting that obesity is caused by an enlarged stomach because gastric stapling surgery, which makes the stomach smaller, is effective in alleviating obesity. The fact that urethral support operations cure SUI does not implicate urethral hypermobility as the cause of SUI.

Most studies have shown not only that there is substantial variation in resting urethral closure pressures in normal women compared with those with SUI, but also that the severity of SUI correlates quite well with resting urethral closure pressure.

Loss of urethral closure pressure probably results from age-related deterioration of the urethral musculature as well as from neurologic injury (Hilton & Stanton, 1983; Snooks et al., 1986; Smith et al., 1989a, 1989b). For example, the total number of striated muscle fibres within the ventral wall of the urethra has been found to decrease seven-fold as women progress from 15 to 80 years of age, with an average loss of 2% per year (Fig. 3.5) (Perucchini et al., 2002a).

Because the mean fibre diameter does not change significantly with age, the cross-sectional area of striated muscle in the ventral wall decreases significantly with age; however, nulliparous women seemed relatively protected (Perucchini et al., 2002b). This 65% age-related loss in the number of striated muscle fibres found *in vitro* is consistent with the 54% age-related loss in closure pressure found *in vivo* by Rud et al., 1980,

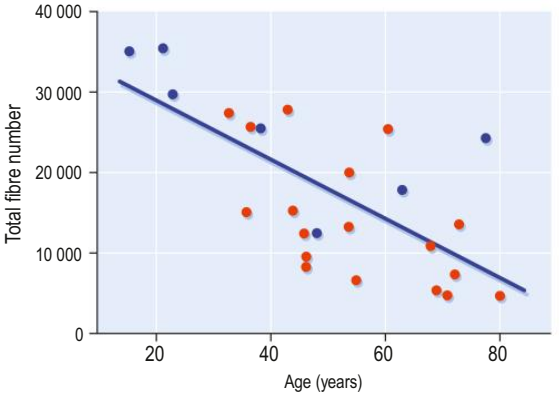

Fig. 3.5 Decrease in Total Number of Striated Muscle Fibres in the Ventral Wall with Age. The red circles denote data from nulliparous women, and the blue circles denote data from parous women. (From Perucchini et al., 2002a, with permission of Lippincott Williams & Wilkins, Baltimore, MD.)

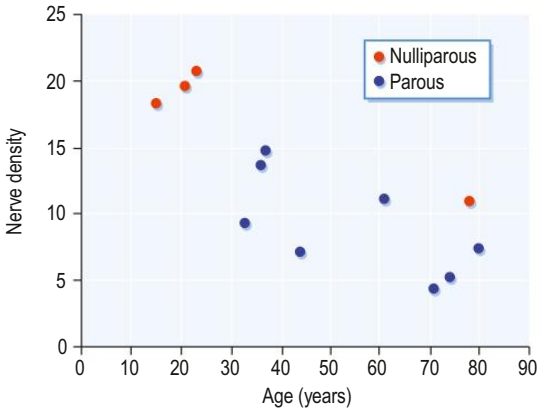

Fig. 3.6 Decreasing Nerve Density (number per mm^2) in the Ventral Wall of the Urethra with Age. This is a subgroup of the data in Fig. 3.5 (Perucchini et al., 2002a). The red circles denote data from nulliparous women and the blue circles denote data from parous women. (From Pandit et al., 2000, with permission of Lippincott Williams & Wilkins, Baltimore, MD.)

suggesting that it may be a contributing factor. However, prospective studies are needed to directly correlate the loss in the number of striated muscle fibres with a loss in closure pressure *in vivo*.

It is noteworthy that in our *in vitro* study thinning of the striated muscle layers was particularly evident in the proximal vesical neck and along the dorsal wall of the urethra in older women (Perucchini et al., 2002b). The concomitant seven-fold age-related loss of nerve fibres in these same striated urogenital sphincters (Fig. 3.6) directly correlated with the loss in striated muscle fibres