



Review

Role of pelvic floor in lower urinary tract function

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ABSTRACT

The pelvic floor plays an integral part in lower urinary tract storage and evacuation. Normal urine storage necessitates that continence be maintained with normal urethral closure and urethral support. The endopelvic fascia of the anterior vaginal wall, its connections to the arcus tendineus fascia pelvis (ATFP), and the medial portion of the levator ani muscles must remain intact to provide normal urethral support. Thus, normal pelvic floor function is required for urine storage. Normal urine evacuation involves a series of coordinated events, the first of which involves complete relaxation of the external urethral sphincter and levator ani muscles. Acquired dysfunction of these muscles will initially result in sensory urgency and detrusor overactivity; however, with time the acquired voiding dysfunction can result in intermittent urine flow and incomplete bladder emptying, progressing to urinary retention in severe cases. This review will start with a discussion of normal pelvic floor anatomy and function. Next various injuries to the pelvic floor will be reviewed. The dysfunctional pelvic floor will be covered subsequently, with a focus on levator ani spasticity and stress urinary incontinence (SUI). Finally, future research directions of the interaction between the pelvic floor and lower urinary tract function will be discussed.

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Contents

1. Introduction	43
2. Pelvic floor anatomy and physiology	44
3. Normal pelvic floor function in bladder storage and emptying	44
3.1. Pelvic floor function and bladder storage	44
3.2. Pelvic floor function and bladder emptying	45
4. Injury to pelvic floor	45
4.1. Childbirth	45
4.2. Aging	45
4.3. Menopause	46
5. Pelvic floor dysfunction	46
5.1. Levator ani spasticity	46
5.2. Stress urinary incontinence	46
6. Future directions and conclusion	47
References	48

1. Introduction

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endopelvic fascia of the anterior vaginal wall, its connections to the arcus tendineus fascia pelvis (ATFP), and the medial portion of the levator ani muscles must remain intact to provide normal urethral support. Thus, normal pelvic floor function is required for urine storage. Normal urine evacuation involves a series of coordinated events, the first of which involves complete relaxation of the external urethral sphincter and levator ani muscles. Acquired dysfunction of these muscles will initially result in sensory urgency and detrusor overactivity; however, with time the acquired voiding dysfunction can result in

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intermittent urine flow and incomplete bladder emptying, progressing to urinary retention in severe cases. This review will start with a discussion of normal pelvic floor anatomy and function. Next various injuries to the pelvic floor will be reviewed. The dysfunctional pelvic floor will be covered subsequently, with a focus on levator ani spasticity and stress urinary incontinence (SUI). Finally, future research directions of the interaction between the pelvic floor and lower urinary tract function will be discussed.

2. Pelvic floor anatomy and physiology

The pelvic floor is an interdependent complex system comprised of striated muscle, smooth muscle, and connective tissue (DeLancey, 1994a). The levator ani muscle complex, containing striated muscle, is inferior to most portion of the pelvic floor and it consists of the pubococcygeus, ileococcygeus, and puborectalis muscles. The pubococcygeus is the anterior component of the levator ani muscles, and it originates from the back of the pubis and it attaches to the perineal body (see Fig. 1). The tonic contractions from the type 1 (slow-twitch) muscle fibers of the pubococcygeus keep the urogenital hiatus closed by compressing the vagina, urethra, and rectum toward the pubic bone, thereby elevating the pelvic organs. This serves as a very important role in the competence of the urethral and rectal sphincters, particularly during periods of increased abdominal pressure. The iliococcygeus and puborectalis muscles form the posterior component of the levator ani muscles. They form a flat, horizontal shelf that spans between the two pelvic sidewalls. The two sides of these muscles fuse in midline posterior to the rectum and attach to the coccyx. The posterior levator ani group not only stabilizes the upper vagina and cervix in a horizontal plane but also prevent downward forces onto the perineal body (Wall and Menefee, 2002).

The endopelvic fascia is a dense condensation of fibrous connective tissue that surrounds the vagina and attaches the vagina to each ATPF laterally (Ashton-Miller and DeLancey, 2007). The urethra is normally suspended on the anterior vaginal wall by attachment points along the length of each ATPF, which runs from the pubic bone to the ischial spine and attaches to each pelvic sidewall. A highly interdependent

load sharing relationship exists between the connective tissue and striated muscle within the pelvic floor to maintain normal vaginal support (Abramowitch et al., 2009). It is thought that the pelvic floor connective tissues are kept under minimal tension as long as the levator ani muscles function normally to close the urogenital hiatus and carry the weight of the abdominal and pelvic organs. When the pelvic floor muscles are damaged, the connective tissues may stretch, thereby resulting in urethral hypermobility and SUI in some women. DeLancey et al. undertook a case-control study comparing 151 women with advanced stage prolapse with 135 controls that had normal support (DeLancey et al., 2007). Although more than half of the women with prolapse had major (more than half missing) levator ani defects, 45% of these women either had minor defects (16%) or no defect (29%). This supports the fact that additional components must be involved in the development of POP, including direct mechanical injury to the connective tissue attachments, denervation, ischemia and defective soft tissue remodeling.

Traditionally, it has been taught that the levator ani muscles are innervated by the pudendal nerve on the perineal surface and direct branches from the sacral nerves on the pelvic surface (Wall, 1993). However, Barber et al. performed detailed gross dissections of the pelvic muscles in female cadavers and histologic evaluation of nerve biopsy specimens and showed that the levator ani muscles are innervated solely by the levator ani nerve, which originates from S3, S4, and/or S5 foramina (Barber et al., 2002). This nerve travels on the superior (intrapelvic) surface of the muscles without the contribution of the pudendal nerve (see Fig. 2). During sacrospinous ligament fixation for the correction of vaginal vault prolapse, the levator ani nerve may be injured (Sze and Karram, 1997). This can cause pelvic floor denervation and atrophy, thereby resulting in anterior compartment vaginal prolapse after sacrospinous vault suspension.

3. Normal pelvic floor function in bladder storage and emptying

3.1. Pelvic floor function and bladder storage

Normal bladder storage is dependent upon both structural and functional components. As mentioned previously, the structural components

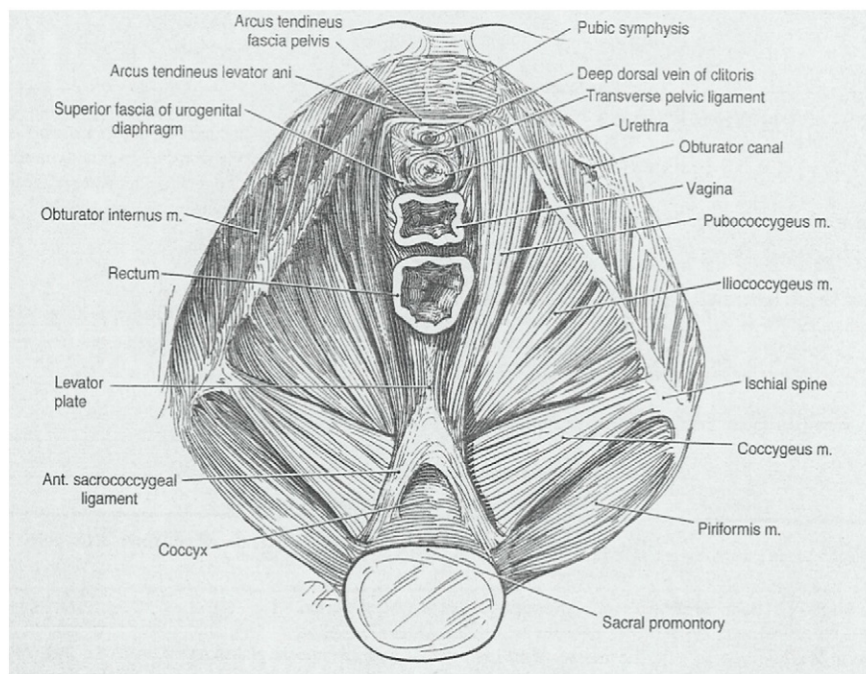


Fig. 1. Muscles of the female pelvic diaphragm.

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