Stress Urinary Incontinence in Female Athletes

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Objective: The purpose of this study is to educate allied health professionals and female athletes of the anatomy of the pelvic floor, and the pathology, etiology, and prevalence of stress urinary incontinence in female athletes. **Background:** Urinary incontinence is not a life-threatening or dangerous condition, but it is socially embarrassing, may cause the individual to remove herself from social situations, and decrease quality of life. While typically associated with parous women who had vaginal delivery, research has shown prevalence of the condition in physically active women of all ages. Stress urinary incontinence has shown to lead to withdrawal from participation in high-impact activities such as gymnastics, aerobics, and running. It may be considered a barrier for life-long athletics participation in women. **Description:** An in-depth introduction to the cause and origin of stress urinary incontinence including review of the female pelvic floor anatomy and prevalence of stress urinary incontinence in the female athletic population. **Clinical Advantages:** Athletic trainers and other allied health professionals will develop an understanding of the multiple mechanisms that cause stress urinary incontinence. Clinician competency of the dynamics and mechanism of urinary incontinence prepares the individual to learn diagnostics, prevention, pharmacological intervention, and treatment of this pathology. **Key Words:** pelvic floor dysfunction, pelvic floor muscles, exercise

Urinary incontinence is defined by the International Continence Society (ICS) as "the complaint of any involuntary leakage of urine".[1] The condition of urinary incontinence is a social or hygienic problem and is objectively demonstrable.[1] There are three types of urinary incontinence: urge, stress, and mixed. Urge urinary incontinence is the complaint of "involuntary leakage accompanied by or immediately proceeded by urgency."[1] Stress urinary incontinence (SUI) is the complaint of "involuntary leakage on effort or exertion"[1] such as coughing, sneezing, laughing, and exercise. Genuine stress incontinence is urodynamically proved involuntary loss of urine when intravesical pressure exceeds that of the urethra without simultaneous detrusor contraction.[1] Mixed urinary incontinence is the complaint of "involuntary leakage associated with urgency and also with exercise, effort, sneezing, and coughing."[1]

Prevalence

Urinary incontinence is more common in women of all ages than in men.[2] "Modern living conditions coupled with the increased female work force and the desire to keep in shape through fitness programs result in urologic symptoms representing a high risk to the pelvic floor."[3] SUI is the most prevalent type of urinary incontinence[2] with prevalence rates between 10% and 55% in women between ages 15 and 64 years.[3,4] Often associated with the older multiparous women, SUI has been reported to occur in two-thirds of female gymnasts[5] and 28% of female collegiate athletes.[6] Data refutes the customary thinking that physically fit women do not suffer from urinary incontinence, and the participation in sports and fitness activities should offer protection from this condition.[7]

Vaginal delivery is one of the highest risk factors[6] for developing urinary incontinence, but prevalence has been observed and measured in nulliparous physically active females. "It can occur during various physical activities, but is especially noted in exercise that involves chronic,

repetitive motion and involves high impact landings, jumping, and running."[8] Female athletes involved with track and field (long jump, triple jump, high jump, hurdles), gymnastics (floor exercises, asymmetric bars, trampolining), basketball, volleyball, handball, combat sports (karate, judo), bodybuilding, and horseback riding are at highest risk, although frequency of SUI is noted in tennis players, skiers, skaters, and joggers.[9] Bø et al[10] demonstrated that 26% of female physical education students had involuntary urinary leakage during various physical activities, a significantly higher prevalence as compared to sedentary students.

The relationship between urinary incontinence and physical activity was assessed by Nygaard et al[9], who surveyed 326 women, 290 of whom exercise regularly, with a mean age of 38.5 years. Forty-seven percent of participants noted some degree of incontinence, which correlated positively with the number of vaginal deliveries. Thirty percent noted incontinence during at least one type of exercise. Urinary incontinence exclusively during exercise was seen in only one woman. Exercises involving repetitive bouncing were associated with the highest incidence of incontinence. Twenty percent of incontinent exercisers discontinued an exercise because of incontinence, whereas 18% changed the way a specific exercise was performed and 55% wore an absorbent pad during exercise.[9]

Boucier[11] researched female SUI in sports and fitness activities. Fifty-nine women with a mean age of 25 years were split into two groups: female athletes and women who practice sports on a regular basis. Degree of incontinence was classified as severe, moderate, or mild. Severe refers to dripping incontinence with exercising. Moderate is defined as urinary leakage with heavy lifting or running. Mild is leakage with jumping. The prevalence of SUI symptoms was 7% severe, 24% moderate, and 33% mild in the athlete group.

Approximately forty-two percent of the 291 elite female ballet dancers, gymnasts, badminton, volleyball, handball, and basketball players surveyed had experienced urine loss in daily life, while 43% experienced urine loss during their sport. Of those who leaked during sport participation, 95.2% experienced urine loss while training versus only 51.2% during competition. Sixty percent of individuals reporting SUI wore absorbent pads during activity. [6]

Risk factors for SUI in female athletes extend beyond the anatomy of the pelvic floor. It is hypothesized that hypothalamic amenorrhea attributed to rigorous exercise, eating disorders, or a combination of both. These factors may result in lower estrogen levels, contributing to urinary incontinence.[4] Bø and Borgen[2] found that the prevalence of SUI and urge incontinence in eating disordered athletes in comparison to healthy athletes was 38.8% and 15%, respectively. In contrast, a study conducted in 2001 demonstrated no difference in prevalence of either urinary incontinence between eating disordered non-athletes and healthy non-athletes.[2]

Urinary incontinence is not a life-threatening or dangerous condition, but it is socially embarrassing, may cause the individual to remove herself from social situations, decrease quality of life, and increase sedentary behavior. A sedentary lifestyle is one of the top ten leading causes of death and doubles the risk of cardiovascular disease, type II diabetes, and obesity.[12] Sedentary lifestyle also increases the risks of colon and breast cancer, high blood pressure, lipid disorders, osteoporosis, depression and anxiety.[13]

Anatomy: Pelvic Floor

The anatomy of the pelvic floor includes structures responsible for active and passive support of the urethrovesical junction, vagina, and anorectum. "Intrinsic and extrinsic properties of the urethrovesical neck and anorectum allow maintenance of urinary and anal continence at rest and with activity."[13] The continence mechanism refers to the many structures that may contribute to a woman's continence: urethra, bladder, sphincter and pelvic floor muscles (PFM),

neurologic innervation, hormonal status, skeletal structure, resting abdominal pressure, and collagen makeup.[3,7]

Three bones form the human pelvis: the left and right innominate, the sacrum, and the coccyx. Each innominate bone is formed by fusion of the ilium, ischium, and pubis. The bones are attached anteriorly by the symphysis pubis and posteriorly to the sacrum by the sacroiliac joints. The pelvis accommodates the bladder, urethra and ureters, vagina, and rectum.

The bladder wall is composed of bundles of smooth (detrusor) muscle. Its unique compliance allows it to function as a low-pressure, high compliance organ that adapts to increasing volume with minimal increase in internal pressure. As the bladder fills during the storage phase, the walls stretch and it assumes a spherical shape. The detrusor muscle has a oblique arrangement of muscle fibers that is ideally suited to empty the spherically shaped bladder. Two U-shaped bands of fibers, known as the detrusor loops, lie at the bladder neck (urethrovesical junction) and can function as a spincter favoring closure.[14]

The trigonal muscle is a specialized smooth muscle surrounding the urethra at the bladder neck. It is composed of three parts: the trigone, the trigonal ring, and the trigonal plate. It is believed that the trigone contributes to the continence mechanism through alpha-andrenergic innervation, keeping this region of the bladder neck closed.[14]

The urethra is a hollow muscular tube, embedded in the anterior vagina wall. The wall is composed of a hormonally sensitive mucosa, a submucosal vascular plexus, and three muscular layers. The musculature is composed of longitudinal and circular layers of smooth muscle, and the striated urogenital sphincter muscle. The striated urogenital sphincter and the vascular submucosa are each responsible for contributing to approximately one third of the total urethral closing pressure at rest.[15]

The urogenital sphincter is divided into two parts that function as a unit: the sphincter urethra (which lies adjacent to the proximal one centimeter of the urethral lumen) and the urethrovaginal sphincter (which extends below this level). Both are primarily composed of slow twitch fibers, which maintain constant tone but have the ability to generate additional force when recruited, as would occur during coughing.[7] The striated muscle of this sphincter is contracted voluntarily both to interrupt urine stream and when a sense of urge is felt but the social situation is inappropriate for voiding. It is contracted reflexively during increases in intra-abdominal pressure when urethral resistance needs to be augmented.[14]

An estrogen-dependant submucosal vascular plexus encircles the urethral mucosa. Together, the mucosa and submucosa form a cushion that contributes to urethral closing pressure. The submucosa is a highly organized arteriovenous complex capable of filling and emptying. Therefore, it is thought to contribute to the continence mechanism mainly at rest, by filling the urethral wall and mucosa with blood and forming a hermetic seal.[15]

Interconnections of the three structures support the vesicle neck and proximal urethyra: the arcus tendineus fasciae pelvis, the endopelvic fascia (Figure 1), and the levator ani muscles. The arcus tendineus fasciae pelvis is a fibrous band of fascia that is attached ventrally to the pubic bone and dorsally to the ischial spine and provides a lateral attachment for the pelvic floor muscles and ligaments. The endopelvic fascia is the first layer of the pelvic floor. It consists of the pubourethra ligaments, the urethropelvic ligaments, and the uterosacral ligaments and attaches the uterus and vagina to the pelvic wall. The pelvis is bordered caudally and dorsally by the PFM that provide support for the pelvic organs to relieve constant strain on the pelvic ligaments.[7]

The pelvic floor consists of three separate components of the levator ani muscles: pubococcygeus arises from the back of the pubis and is directed posteriorly along the side of the anal canal toward the coccyx and sacrum, the ileococcygeus comes from the ischial spine and is attached to the coccyx, the coccygeus arises from the spine of the ischium and inserts into the margin of the coccyx and into the side of the inferior sacrum (Figure 2).[3, 15] These three muscles comprise two functional groups: the pubococcygeus acts like a sling, effectively closing the urogenital hiatus tightly against the pubic bone; the other two muscles act synergistically in providing a "levator plate"[7] on which the pelvic organs rest when tension is placed on their ligamentous structures. The opening within the levator ani muscle, through which the urethra and vagina pass, is called the urogenital hiatus.[3]

The levator ani muscles are primarily slow twitch fibers[7], providing tone to the pelvic floor to support the viscera during normal activity. The remaining fast twitch fibers are reflexively contracted during Valsalva maneuvers, during sudden increases in pressure, or during voluntary contraction.[15] Contraction of the fast twitch fibers elevates the pelvic viscera and closes the vaginal introitus.[7,15]

The neuronal control of urination involves a series of complex pathways that connect the cerebral cortex to the midbrain, spinal cord, bladder, urethra, and pelvic floor. These pathways allow for reflex subconscious control and voluntary cortical control of the lower urinary tract. Autonomic and somatic peripheral activity within the lower urinary tract are under the control of the central nervous system.[15]

The combination of suspension of the genital tract by the ligaments and fascia and contraction of the pelvic floor by the levator ani is responsible for normal urinary continence. The levator ani receives sensory and motor innervation from the pudendal nerve branches and directly from the sacral nerve plexus. At rest, the urinary sphincters are closed due to active firing of the sympathetic fibers to smooth muscle and of the somatic pudendal neurons to the striated muscle of the urethra and the urinary sphincters. Further, the detrusor motor neurons are inhibited via supranuclear control. As urine accumulates, the bladder wall distends. With small urinary volumes there is an adjustment of tone via stretch receptors in the bladder wall so that intravesical (urinary bladder) tension does not, at first, increase. This is accomplished primarily through physical changes in the detrusor. Storage phase activity is modulated by both the sympathetic and parasympathetic systems. The voiding phase, however, is primarily mediated by parasympathetic activity. The micturition reflex begins with a sudden and complete relaxation of the striated muscles of the pelvic floor and a decrease in urethral pressure. Seconds later, intravascular pressure increases due to a contraction of the detrusor muscle.[15] Decent and funneling of the bladder neck occur and flow begins. During micturition, the PFM relax allowing the bladder neck to rotate downward. Decent is limited by fascial attachments to the arcus tendineus fascia. Once voiding has been completed, the levator muscles resume normal resting tone, bringing the bladder neck back up to its original position.[7]

"During a voluntary contraction, the PFM lift inwardly. The urethra closes and the PFM resist downward movement, thereby stabilizing the urethra."[3] During sudden stress, such as coughing, a fast-acting contraction of the distal urethra contributes reflexively to the compressive forces of the proximal urethra, preventing urine leakage. When the pressure of a cough is generated by contraction of the thoracoabdominal muscles, the PFM also contract in coordination with the respiratory diaphragm, as well as intercostal and abdominal wall muscles, to maintain continence.[7] Additionally, the levator ani muscles contract and the pubococcygeus portion is pulled anteriorly towards the pubic symphysis to compress the urethra.[15]

Urinary Incontinence

Etiology

SUI is diagnosed based on urine leakage concurrent with an increase in abdominal pressure, with or without pelvic floor prolapse or urethral hypermobility and in the absence of bladder detrusor activity. It is generally believed that SUI results from damage to one or more of the support mechanisms that stabilize the vesicle neck and proximal urethra.

Known risk factors for SUI include weak connective tissue and PFM, pregnancy, vaginal delivery with injury to the peripheral nerves, fascia, ligaments, and PFM, obesity, strenuous work including physical activity, and old age.[2] A National Institute of Health consensus panel developed a list of 6 risk factors associated directly with exercise-induced stress urinary incontinence including "increasing age, female gender, increased parity, heavy physical activity, high-impact sports, hypoestrogenic amenorrhea, and obesity."[10] Other factors are less clear, including strenuous work, exercise, constipation with straining on stool, coughing or other conditions that can increase abdominal pressure chronically.[3]

Activities most commonly associated with a sudden increase in intra-abdominal pressure are jumping, landings, and dismounts. "The PFM must be able to contract very forcefully and rapidly to withstand the constant, repetitive deceleration of the abdominal viscera on the pelvic floor caused by repetitive running or jumping."[8] It is speculated that athletes are more incontinent during sports that require running into a jump, which adds momentum to the dynamic impact of the abdominal viscera on the pelvic floor. "Force-plate studies have illustrated a wide range of maximum vertical ground-reaction forces for different activities. For example, long jumpers, who land on their heels, can generate a maximum ground-reaction force of up to 16 times their body weight."[8] Generalized muscle fatigue, including PMF fatigue, may also play a role in stress urinary incontinence.[8]

Pathophysiology

A major cause of SUI is the loss of anatomic support of the urethra and urethrovesical junction. This anatomical support is provided anteriorly by the pubo-urethra ligament. The posterior urethra is pushed anteriorly during activation of the pelvic floor muscles. When the bladder and proximal urethra are well supported, increases in intra-abdominal pressure are transmitted equally to both structures, and continence is maintained. This occurs at least in part, because coughing or straining compresses the anterior urethral wall against the well-supported posterior wall, thereby occluding the lumen. Loss of anatomic support allows a displacement of the bladder neck causing the opening of the posterior aspect of the urethrovesical junction during physically stressful activities. Increases in intra-abdominal pressure are then fully transmitted to the bladder, and to a lesser extent to the urethra, and urine loss occurs.[15]

Chronic increased intra-abdominal pressure may place a woman at risk due to resultant pelvic relaxation. When the pressure is too high or the levator ani are damaged, this disturbed anatomic relationship leads to the loss of support to the reproductive organs. In the biomechanical concept of pathophysiology of pelvic floor dysfunction, it is claimed that the muscular abdominal wall is strong (voluntary or reflex abdominal contraction) and rigid prohibiting the levator ani muscles to play the role of "shock absorber."[12]

Vaginal delivery causes partial denervation of the pelvic floor in most women having their first baby. Damage to the pudendal nerve releases tension placed on the pelvic floor tissue and is associated with urinary and fecal incontinence in some women.[12] For some it is likely to be the first step along a path leading to prolapse of the abdominal viscera and/or stress urinary incontinence.[16] When abnormal intra-abdominal pressure exists, the suspensory mechanism is

called on to support an increased load. While the ligaments can support the abdominal viscera for a short amount of time, "the connective tissue will eventually become damaged."[12] The anatomic pathophysiology of organ prolapse in the new mother and that of the athlete become similar in nature. If the pelvic floor is no longer functionally efficient, the connective tissue will become damaged and stability is not maintained.

Conclusion

Although urinary incontinence is not a life-threatening or dangerous condition, it is socially embarrassing, may cause the individual to become withdrawn from social situations, and decrease quality of life. Research on SUI has revealed that this condition may lead to withdrawal from participation in high-impact activities such as gymnastics, aerobics, and running.[6,9-12] It may be considered a barrier to life-long athletic participation in women. Educating allied health professionals on the anatomy of the pelvic floor, pathology, etiology, and prevalence of stress urinary incontinence may remove barriers for individuals seeking care for this condition.

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Figure 1. The arcus tendineus is a thickened linear component of the obturator fascia. It lies between the pelvic surface of the body of pubis and the ischial spine and gives origin to the muscles of the levator ani. The uterosacral ligaments are paired crescent-shaped folds of peritoneum. The uterovesical ligament is a thickened fold of peritoneum that is reflected from the anterior aspect of the junction between the cervix and body of the uterus, to the posterior aspect of the bladder. The pubovesical ligaments act to stabilize the bladder, attaching at the neck.
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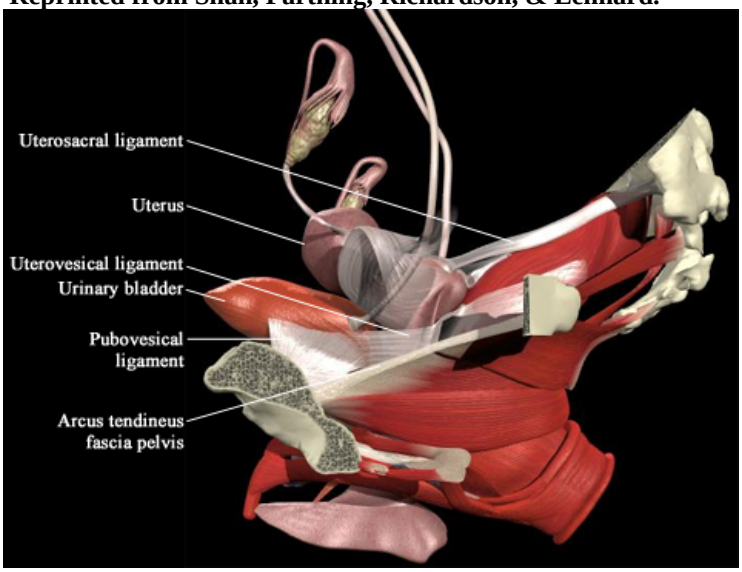

Figure 2. Inferior view of the female pelvic floor. The pubococcygeus forms the anterior part of the levator ani muscle. The iliococcygeus forms the posterior part of the levator ani muscle. The coccygeus is often described as the muscular belly of the sacrospinous ligament with which it is fused on its pelvic aspect.
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