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Research Progress of Postpartum Stress Urinary Incontinence

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Abstract. Postpartum stress urinary incontinence (SUI) is one of the gynecological disorders that plague postpartum women, which severely affects women's daily work and life and may even cause anxiety and depression among women while decreasing their quality of life. In recent years, conservative treatment methods have been explored and applied extensively in postpartum SUI rehabilitation. Experimental data suggest that providing specific treatments for specific groups can greatly improve the efficiency of treatment and that classifying different types of treatments can also effectively promote the advancement of related diagnostic methods. This article systematically reviews the literature on the pathological mechanisms, and conservative treatment of postpartum urinary incontinence (UI), and discusses the optimal treatment methods for different etiologies, providing some insights for the rehabilitation and management of patients with postpartum SUI.

Keywords: stress urinary incontinence, postpartum, pathophysiology, risk factors, conservation treatment

1. Introduction

Pelvic floor dysfunction (PFD) is one of the five most common chronic conditions suffered by women, mainly including symptoms such as stress urinary incontinence (SUI), genital prolapse, fecal incontinence, constipation, sexual dysfunction, and pelvic pain. Among them, SUI is a condition in which urine is involuntarily leaked due to physical exertion, sneezing, coughing, or other actions that increase intra-abdominal pressure [1]. According to an epidemiological survey, the prevalence of SUI is as high as 6-10% in developed regions like the United States and Europe [2]. In contrast, an epidemiological survey conducted in Nanjing shows that the prevalence of UI is 5.85%, of which SUI accounts for 67.5% [3]. Against the backdrop of late marriage and late childbirth, the number of older mothers is increasing year by year, which may raise the incidence of postpartum diseases, including SUI. SUI is usually accompanied by skin ulcers and infections, which can even cause shame and lack of confidence in patients, severely affecting their quality of life and sexual intercourse [4-6]. SUI is not only a personal problem, which has a significant impact on the public health of the community. Therefore, it is necessary to build a comprehensive understanding of postpartum SUI and develop effective treatment accordingly.

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2. Normal urethral anatomy and the physiology of urination

2.1. PFMs and urethral anatomy

The PFMs refer to the muscles of the pelvic floor, which are divided into three layers. The superficial layer includes the superficial transverse perineal muscle, the bulbocavernosus muscle, and so on; the middle layer consists of the deep transverse perineal muscle, the urethral sphincter and so on; and the deep layer includes the anal levator muscle.

The female urethra is located behind the pubic symphysis and in front of the vagina, passing from the internal bladder opening to the urogenital diaphragm and opening in the vaginal vestibule, with a total length of 30-50 mm and a diameter of 8-10 mm. The upper segment of the internal urethral sphincter is a circular smooth muscle, which is connected to the circular muscle of the bladder neck, and the muscle fibers are particularly thick, forming a strong internal sphincter. In addition, the levator muscle and deep perineal muscle groups play a secondary role in controlling urination.

The upper segment of the female urethral mucosa is the migratory epithelium and the lower segment is the squamous epithelium. The submucosa of the urethra is richly distributed with urethral glands opening on the mucosal surface [7].

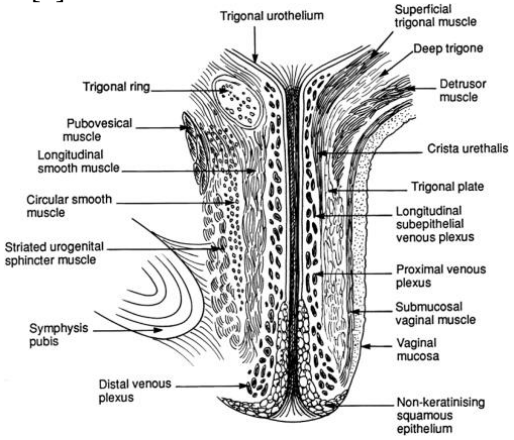


Figure 1. Midsagittal section showing the anatomy of the older female urethra (© DeLancey 1997).

2.2. Normal physiology of the urethra

2.2.1. A normal female lower urinary tract control is divided into the storage and voiding phases. During the storage phase (both during rest and during increased abdominal pressure), the urethral closure pressure must be greater than the bladder pressure to retain urine in the bladder, i.e., the positive urethral phase closure pressure maintains urinary control. With the rise of abdominal pressure, the pressure acts on the bladder surface and transmits to the proximal urethra, causing active contraction of the urethral sphincter to prevent urine discharge. [8] During the micturition period, the urethral closure pressure decreases and is lower than the bladder pressure, thus allowing urine to pass out of the body.

2.2.2. *Normal voiding mechanism.* Active Mechanism The active mechanism is the control of intraurethral pressure by the upper, middle, and lowers urethral sphincter, which includes the upper circular smooth muscle, the middle transverse muscle, and a small number of muscle fibers in the lower part of the urethra. Among them, the urethral smooth muscle provides approximately 50% of the pressure in the urethra, with the deep transverse perineal muscle on its outer circular smooth muscle

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being the most beneficial for urinary control. The urethral smooth muscle is flanked by three layers of the transverse urethral sphincter providing50% of urethral pressure [9].

Anatomic Structural Support The position of the urethra is influenced by the position of the vagina and the pelvic floor structures. Most of the urethra is fused to the vaginal wall, so the structures that determine the position of the urethra and the position of the distal anterior vaginal wall are the same. The anterior vaginal wall and urethral support system consist of all the structures external to the urethra that provide a support layer for the anterior vaginal wall and urethral support system. The main components of this support structure are the vaginal wall, the intrapelvic fascia, the pelvic fascial tendon arch, and the anal levator muscle [10]. The anal levator muscle is a part of the PFM, which is an important structure of the pelvic floor. Other connective tissue in the body will be deformed when it is stretched for a long time. However, this deformation will not happen in the pelvic floor structure because of the PFM. If the PFM is damaged, the ligaments and fascia of the pelvic floor will change their shape when they are stretched for a long time, which will affect the function of the pelvic floor organs. Therefore the PFM has the function of actively supporting and shaping the pelvic organs [11].

Mechanisms Of Bladder Protection and Urothelial Mucosal Closure The bladder lumen is lined with a layer of intravesical mucosa that protects the underlying detrusor muscle from toxins in the urine and communicates with nerve cells that coordinate the storage and voiding phases [11]. The lumen of the urethra is lined with the urethral mucosa, and the submucosa consists of connective tissue between the uroepithelium and the longitudinal smooth muscle layer of the urethral wall, which is richly vascularized and extremely sensitive to estrogen. When the submucosa is vascularized and within normal limits, the lumen of the urethra is closed and this response helps maintain urethral resting pressure [8, 12].

3. Pathological mechanisms

Although the anatomy of the bladder, urethra and urinary sphincter has been well studied, the pathologic mechanisms behind SUI are still controversial. We explored the pathological mechanisms of SUI from several aspects, including the damage to the endopelvic fascia and PFM supporting the urethra, decreased urethral sphincter function, urethral mucosal closure mechanisms, and changes in the extracellular matrix (ECM) of the connective tissue of the urethral pelvic floor.

3.1. PFM injury

For young, healthy women, the pelvic organs maintain the normal anatomical position of the genitourinary organs through a complex interaction between the levator muscle, the vagina, and the connective tissue of the perineum. Therefore, injury to any of these parts can cause abnormalities in the anatomy of the genitourinary organs.

There is an important hypothesis for the pathogenesis of SUI due to birth injury to the PFMs - the hammock hypothesis, which is a widely accepted pathophysiological explanation for SUI with high urethral mobility and was proposed mainly based on the theory of normal anatomy of the urethra and pelvic floor. The hypothesis proposes that the increase and decrease of intraurethral closure pressure depend mainly on the urethral compression of the suburethral support tissue [10]. The deep layer of the PFM is the toughest layer of the pelvic floor, including the levator muscle and other connective tissues, which plays an important role in supporting the pelvic organs; the inner layer of the PFM, located above the levator muscle, is a tough connective tissue membrane that covers the pelvic floor and pelvic wall, with some thicker connective tissues that merge upward with the muscle fibers of the pelvic organs to form the corresponding ligaments. These ligaments also have a strong supporting effect on the pelvic organs. Through these muscles and ligaments, PFM creates a “hammock”, i.e., under normal conditions the urethra is compressed both at rest and during activity, and based on this compression, together with the active mechanism of the urethra and the mucosal closure mechanism, the urethral cavity is closed, preventing involuntary urine flow. And when the PFM tone decreases and muscle atrophy causes a decrease in the supporting force of the pelvic floor structures, this loss of support can lead to excessive urethral mobility. Therefore, the urethra does not move downward when intra-abdominal pressure

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increases. Instead, it moves downward when the position of the bladder neck moves down, the tilt of the urethra increases and the posterior angle of the urethra becomes flat.

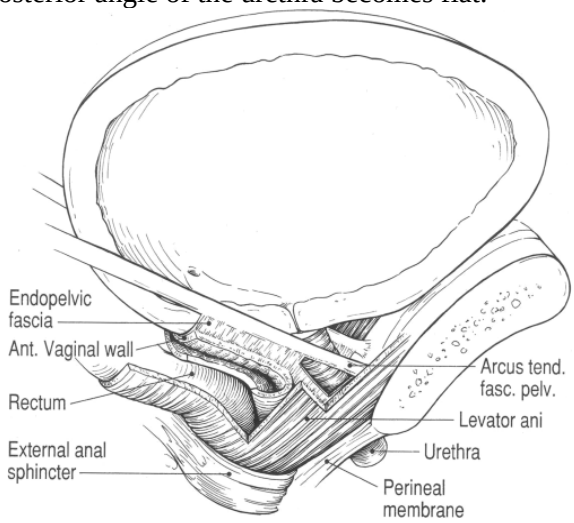


Figure 2. The anatomy of the female pelvic floor structure(From © DeLancey 2005, redrawn from DeLancey 1994, with permission of C.V. Mosby Company, St. Louis).

3.1.1. Injury to the anal raphe muscles. Morphological changes in the PFMs of patients who develop SUI are both neurogenic and myogenic [13]. Pudendal nerve injury and muscle contraction of the anal raphe muscles are sometimes observed at the end of the second stage of labor, and even strains or ruptures of the pubic fascia, vaginal septum, and perineal body may be observed due to compression of nearby nerves and muscles as the fetal previa crosses the birth canal.

The micturition process is a spinal reflex process, which is under the casual control of higher centers. When the urinary reflex arc is abnormal or the sacral spinal urinary center fails to receive control from the higher center, symptoms such as abnormal urination will occur. Therefore, the mechanism of SUI nerve injury is divided into the injury of the higher spinal cord and the nerve injury in the peripheral area. However, since there is only a very low probability of high spinal cord injury during pregnancy and delivery, the types of nerve injury in postpartum SUI are mostly peripheral nerve injury and indirect denervation injury.

Medio-lateral episiotomy can avoid mechanical-type compression of the PFMs due to delivery, but this procedure can damage the nerves in the pubic area. A clinical study showed that a mediolateral episiotomy performed during labor did not prevent postpartum SUI and anal incontinence and genital detrusor [14], and even complicated SUI after the procedure, suggesting that damage to the PFMs in women was related to the pudendal nerves. Another animal study applied electron microscopy to analyze the pelvic and perineal nerves of multiparous rabbits compared to young rabbits, which found an increase in the density of unmyelinated axons and the thickness of myelin sheaths of both nerves in middle-aged multiparous rabbits [15]. These changes indicate that birth can cause persistent damage to the pelvic and perineal nerves, as well as regenerative axonal i.e., denervation at the site of injury. In general, indirect denervation due to muscle pulling causes damage to the nerve endings of the innervated skeletal muscles resulting in a decrease in their strength. It is estimated that the associated pudendal nerve is stretched by 13-35% during childbirth [16], which can lead to axonal demyelination, partial muscle denervation, and impaired function. Muscles undergo denervation after adjacent axons send out lateral branches to allow for reinnervation [17], so enzyme staining experiments (ATPase staining) in patients with denervation injury will observe homozygous muscle fiber aggregation in muscle sections from this patient. In a study analyzing sections of anal raphe in patients with postpartum SUI, ATPase

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staining revealed almost all type I muscle fibers [13], indicating denervation of the anal raphe in patients with postpartum SUI. In addition, according to studies of electromyography (EMG) and pudendal nerve conduction [18, 19], denervation of the PFMs after delivery was observed, which again confirmed the direct nerve damage and indirect denervation of the anal raphe due to prolonged mechanical pulling and compression of the pelvic floor by factors of labor and pregnancy.

PFMs are likely to get injured during pregnancy and delivery because of mechanical compression by fetal weight. During pregnancy and delivery, soft tissue stretching of approximately 147 ± 39% leads to muscle damage [12], as evidenced by changes in muscle fiber type composition, nuclear concentration, and fibrosis [20]. In addition, mechanical compression may cause a decrease in vascular perfusion to the anal raphe and thus lead to muscle ischemia and atrophy of the anal raphe, which is manifested by a significant decrease in its muscle volume and an increase in gap width [21].

3.1.2. Vaginal prolapse. Vaginal prolapse is a part of pelvic organ prolapse (POP), as mentioned before the fusion of the vaginal wall and most of the urethra, so vaginal prolapse usually coexists with urinary tract dysfunction, where UI is mostly associated with anterior vaginal wall prolapse [22]. However, it is worth mentioning that the two common problems, SUI and vaginal prolapse, are not causal but have common pathological factors. Severe pelvic nerve degeneration and reinnervation (decreased muscle tone of the PFMs) or changes in the collagen fibers of the pelvic floor can lead to the creation of a low resistance zone and thus produce POP [23,24]. The creation of POP has a high probability of sequentially triggering SUI due to disruption of the normal anatomical position of the pelvic septum [25]. Studies have shown that up to 60% of women with POP are also diagnosed with UI, and nearly 40% of women with UI are found to have some degree of POP [26]. Another epidemiological study was conducted from 2011 to 2013 in 14 patients hospitalized for secondary POP in the urology clinic of the acute care hospital in Craiova County, Romania, in which SUI related to POP was found in 8 (57.14%) cases [27]. It can be seen that women with symptoms of vaginal prolapse face the risk of developing SUI.

3.2. Urethral sphincter injury

Functional damage to the internal and external urethral sphincters is also a major factor causing postpartum SUI. Symptoms of downward urethral movement due to injury of the PFM can decrease urethral sphincter tone [28, 29]. In this case, the increase of intra-abdominal pressure will increase the pressure in the bladder; and the weakness of the urethral sphincter will cause the pressure in the bladder to be greater than the urethral closure pressure, thus leading to SUI.

The function of the external urethral sphincter, as mentioned earlier, is to provide pressure to the urethra to maintain normal voiding mechanisms. Urodynamics examination shows that patients with SUI have abnormal conduction in the perineal branch of the pudendal nerve. The perineal branch innervates the perineal transverse muscle [19]. Therefore, SUI may be triggered in the presence of damage to the external urethral sphincter. Barber et al. showed that the anal raphe was predominantly innervated by direct nerves from sacral nerve direct innervation from roots S3 to S5 [30]. However, a later study by Nyangoh Timoh et al. showed that the anal raphe was innervated by three parts [31], of which the pudendal nerve was both a somatic and autonomic nerve providing innervation to the inferior aspect of the anal raphe. It is noteworthy that the contraction of the external urethral sphincter is also innervated by the pudendal nerve, which is a somatic motor nerve and originates from the 2nd to 4th sacral medullary anterior horn motor neurons with the same innervation as the inferior aspect of the anal raphe and vulva. Therefore, it can be speculated that a direct nerve injury or indirect denervation of the anal raphe during childbirth is likely to cause damage to the external urethral sphincter indirectly and produce SUI because they have the same innervation.

In 1981, the International College of Surgeons (ICS) defined urethral instability (URI) as “an involuntary drop in urethral pressure during filling that results in urinary leakage in the absence of forced urinary activity”. However, the term URI has not been redefined in subsequent standards documents. According to this theory of urogenic forceps instability, urethral pressure imbalance caused by the injured urethral sphincter affects the function of the bladder forceps when urine enters the pressure-

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imbalanced urethra causing reflex forceps contraction. Functional impairment of the bladder forcing muscle can lead to URI and subsequently to SUI [32-34]. A study comparing the total urethral diameter in patients with SUI and normal patients showed that the reduction in total urethral diameter and consequently the reduction in circumference was directly related to the reduction in the thickness of the urethral smooth muscle, the internal urethral sphincter, but not to the transverse urethral muscle, the external urethral sphincter [35]. Furthermore, the circular configuration of the internal urethral sphincter implies a constricting effect on the lumen.

3.3. Damage to the mucosal closure mechanism of the urethra

Sex hormones such as estrogen, estradiol (E2), and progesterone (P4) have a very strong influence on the female urinary system; and unstable changes in sex hormones can lead to changes in the tissue morphology as well as physiological functions of the urinary system. Therefore, urinary symptoms in women due to sex hormone changes may occur after the menstrual cycle, pregnancy, and menopause [36]. Compared with menstrual cycle, E2 and P4 levels increase 100-fold and 10-fold respectively during pregnancy [37]. The urethral mucosa is extremely sensitive to E2, as described by the physiological mechanisms of the urethra, and stabilization of E2 levels causes vascular congestion in the submucosa, thus maintaining urethral resting pressure. However, when the placenta is expelled during delivery, E2 and P4 levels drop rapidly and remain low for a long time after delivery; and there is a postpartum withdrawal response to E2. The decrease of E2 not only induces postpartum depression but also leads to pressure imbalance in the urethra. However, present research mainly focuses on the effects of reduced E2 on prenatal and postpartum depression, while studies of the effects of postpartum SUI are still limited. E2 is a major regulator of vaginal physiological function, and topical use of E2 has a role in the treatment of lower urinary tract symptoms [38]. Further research is needed on the mechanisms by which E2 affects postpartum SUI; and accurate estimates of the risk of endometriosis with continued vaginal E2 use are unclear, requiring additional long-term studies as well.

3.4. Extracellular matrix changes

The tissues and organs of the female reproductive tract and pelvic floor undergo significant remodeling and alterations during pregnancy and childbirth to allow fetal growth and delivery, including collagen [39]. Collagen is the most abundant ECM protein in the body, which plays a crucial role in providing tensile strength to the female pelvic floor tissues and reproductive tract. Since collagen of connective tissue contributes to the structural support of the bladder neck, women will face increased risk of SUI when collagen is abnormal. In connective tissue, when the elastic fiber content decreases, fiber dispersion distribution and elastic fiber maturation are manifested by decreased levels of desmosine concentration and reduced expression levels of fibrillin-5 and LOX [40-43]. A study shows that patients with SUI have significantly lower type I and type III collagen expression levels and less mature elastic fiber in the PFM of than normal population [43]. During pregnancy, reduced stretch properties of connective tissue, lower total collagen content, increased glycosaminoglycans, and changes in collagen may all lead to greater bladder neck mobility. Mobility of ureterovesical junction (UVJ-M) is the “gold standard” for screening UI because increased UVJ-M can cause low bladder neck position during bladder neck tension and lead to impaired intra-abdominal pressure transmission, then resulting in SUI.

3.5. Changes in central and peripheral neurotransmitters

A study demonstrated that UI was associated with an increased incidence of depression and anxiety [44]. Fritel’s study showed that the prevalence of depressive symptoms was higher in women with postpartum SUI than in women without SUI (22.1% versus 15.9%, p=0.045) [45]. Melville demonstrated that major depression could predict UI in older women [46]. And a Czech clinical trial also demonstrated that there was a correlation between postpartum SUI and postpartum depression, though trials of larger scale are needed to demonstrate this relationship [47]. Patients with postpartum depression produce abnormalities in central neurotransmitter metabolism and corresponding alterations in receptor function, abnormal neurotransmitter levels, and reduced functional activity in the synaptic gap of the brain, involving

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neurotransmitters such as 5-hydroxytryptamine (5-HT), dopamine (DA), and noradrenaline (NA), which are often clinically lower in depressed patients [48-50]. Meanwhile, postpartum depression may be accompanied by maternal postpartum SUI, thereby reducing brain synaptic gap neurotransmitter levels. Some nerves containing propagators that mediate contraction or relaxation do exist in the muscle components of the smooth and transverse muscles of the female urethra. NA and 5-HT are usually considered to be the main factors in maintaining smooth muscle tone in the urethra. Neuroanatomical studies have shown that some of the highest densities of 5-HT and NE-containing terminals in the spinal cord [51]. These terminals are found in the lateral pathway (LP), a region of the spinal cord where bladder primary afferent fibers make synaptic connections. The tubular view is supported by another study [52], which found NA receptors and 5-HT in the Lissauer tract and sacral parasympathetic nucleus, bladder primary afferent fibers in the Lissauer tract, and bladder preganglionic neurons in sacral parasympathetic nucleus. In addition, Von Heyden observed the nerve density of acetylcholine [53], NA, neuropeptide Y (NPY), galanin, vasoactive intestinal polypeptide (VIP), and calcitonin gene-related peptide (CGRP) in the urethral sphincter in patients without voiding disorders, confirming the presence of a large number of NAergic nerves in the urethral smooth muscle and that reduced NAergic nerve activity can relaxation of the urethra. It is evident that postpartum depression and postpartum SUI share some pathological factors.

Several clinical trials have shown a link between brain-derived neurotrophic factor (BDNF) and SUI [54]; and observations in animal models suggest that increased levels of BDNF in Serum contribute to the resurrection of the external nerves of the urethral sphincter, and that BDNF is essential for normal sphincter function [55, 56]. BDNF decreases significantly during pregnancy and even keep low levels in the 2-3 months after delivery [57]. Many factors contribute to low BDNF levels during pregnancy, including depression [58], but the mechanisms remain unclear. Therefore, modulation of BDNF could play a role in the treatment of SUI and other lower urinary tract dysfunctions.

4. Non-invasive treatment methods for SUI

Methods of treating SUI for postpartum women can be non-invasive and invasive. Invasive methods are subdivided into less invasive minimally invasive surgery and conventional surgery, where conventional surgery is strongly associated with high postoperative recurrence rates of SUI, and even more than 15% of postoperative complications (bleeding, erosion, urethral injury, infection, chronic pain, and urinary retention) arising from SUI are associated with minimally invasive surgery [59]. Therefore, we focus on non-invasive treatment methods for SUI, which are presented in the following.

4.1. Pelvic floor muscle training (PFMT) and biofeedback (BF) techniques

PFMT is now used as a first-line conservative treatment for SUI, with a recovery rate ranging from 56 to 79% in a randomized controlled study [60], as the function of the PFMs can be improved by pelvic floor exercises and pudendal nerve stimulation even after neuromuscular injury [61]. It has also been shown that performing PFMT exercises before delivery can effectively reduce the risk of SUI in the three months after delivery [62]. PFMT promotes blood circulation in the PFMs through contraction and relaxation of the PFMs so as to exercise the fascial ligaments of the PFMs and increase urethral closure pressure and vaginal resistance. This exercise reduces urinary incontinence by generating urethral closure pressure and decreasing the stimulus of the central nervous system on urinary muscle, which is effective for both UUI (urgent urinary incontinence) and SUI.

The therapist needs to instruct the patient to perform contractions of the PFMs, helping the patient to separate the PFMs (while avoiding contractions of the hip, abdominal, or thigh muscles). The patient will perform three to four times per week, each of which including three groups of eight to ten sessions. One PFM contraction per group is held for 6-8 seconds, and the contraction time can be extended to 10 seconds if possible. The exercise should be maintained for at least 15-20 weeks. Data suggest that up to 38% of patients with SUI who follow a PFM exercise program for at least three months will be cured [63-65].

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PFME is used to treat mild to moderate SUI, which has the advantages of low cost and safety. Patients can choose to have training in the hospital or at home on their own. However, since the training requires some autonomy and more than 30% of women are unable to contract the PFMs correctly during PFMT [66], it has been proposed to combine this training with BF therapy.

BF therapy refers to the use of relevant devices to provide visual or audio feedback about pelvic muscle contractions to the patient. BF is not a treatment method itself, but can be combined with different SUI treatment methods to indicate PFMT activities during rest, contraction, and relaxation. In addition, it can be used to teach patients the correct PFMT technique and give them motivation [67].

However, facts have shown that the combination of training with BF during PFMT exercises does not differ from common PFMT exercises for patients who can contract the PFMs adequately [68]. Besides, BF therapy doesn't help increase the frequency of PFMT exercise [69]. Nevertheless, this combination may be a good option for patients who are unable to contract and separate the PFMs sufficiently. It should be noted, however, that this method does not apply to patients with sphincter disorders. Therefore, the therapeutic effect of PFME and which combination can achieve a better training effect still needs to be further explored.

4.2. Electrical stimulation (ES)

ES uses electrical currents of different resistance to stimulate the vagina rectum and urethra, thus stimulating muscle excitation and enhancing its strength elasticity. Studies have shown that ES causes contraction of the PFMs and increases PFM pressure so as to increase the number of muscle fibers in the rapidly contracting pelvic floor muscles [70]. ES facilitates blood flow in the PFM as well as the urethra, improving neuromuscular connections, and muscle fiber function [71]. Another animal test tested whether ES of the perineal nerve in middle-aged multiparous rabbits induced muscle contraction and urethral closure [15]. The results showed that stimulation of the perineal nerve in middle-aged multiparous animals effectively reversed the baseline deficit, doubling it ($p = 0.02$). This result indicates that selective neuromodulation of PFMs is a potential treatment for SUI and that the use of ES can cause acute depolarization of partially damaged nerves at low (2-5 Hz) or medium (10-20 Hz) frequencies to induce urethral an increase in the proportion of pressure. Therefore, the use of ES excites and activates nerve cells, promotes axonal regeneration, improves cell function, induces nerve conduction, and increases muscle excitability. In addition, the pubic nerve also innervates the external urethral sphincter, so this treatment method can increase urethral closure pressure at the same time. Another role of ES is to promote proprioception and coordination during pelvic floor contractions [72].

ES means that electrodes are placed on different parts of the body to stimulate the nerves innervating the PFMs to treat SUI. There are currently three types of ES, including vaginal electrical stimulation (VES), surface electrical stimulation (SES), and electroacupuncture (EA). The main difference between the three types of ES is the placement of electrons. VES places electrodes in the vagina; SES uses electrodes outside the vagina or on the surface of the sacral region; and EA inserts milli-needles into the third sacral foramen and bilateral points near the end of the coccyx. Although the difference in efficacy and safety between the three types of ES have not been investigated, studies have shown that all ES can reduce the frequency of SUI over a long period and keep urine from leaking for a short period [73] The National Institute for Health and Care Excellence (NICE) guideline recommends the use of ES to treat patients with SUI who are unable to actively contract their PFMs and also recommends against the combination of ES with PFMT.

4.2.1. VES and SES. VSE. treatment can be performed at home and usually consists of two 15-minute sessions per day for 12 weeks. SES usually requires two to three sessions per week for six to eight weeks [63]. Similar to pelvic floor training and vaginal cones, both VES and SES require long-term adherence. Ceasing treatment directly due to improvement will result in the loss of previous treatment effect within

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four to six weeks, whereas continued daily treatment for three to six months after improvement contributes to maintaining PFM to a healthy state [74].

The disadvantage of VES is that the therapist needs to sterilize the electrodes before and after each treatment, and the patient may experience discomfort and even get vaginal and urinary tract infections during treatment [75]. However, pelvic floor ES can directly and maximally increase urethral closure pressure. Compared with VES, SES does not have a significant risk of causing vaginal infections because of the direct ES on the skin surface. However, previous studies show that VES treatment improve the intensity and pressure of PFM contraction and reduce leakage [76], while SESG only improve PFM pressure and reduce leakage. This is because ES involving the lumbosacral region may cause muscle contractions and mimic only PFM training. VES is a treatment method that the patient feels invasive. Although VES is effective, its invasive feeling reduces a patient's acceptance and adherence to this treatment method. In contrast, SES is more acceptable to the patient and more comfortable for the physiotherapist. To sum up, both VES and SES have their pros and cons. Patients can choose one according to their own conditions.

4.2.2. EA. EA is performed by attaching electrodes laterally to the needle handles of bilateral acupuncture points based on acupuncture points after acupuncture with milli needles. The acupuncture sites are the bilateral Zhong Xiao (BL33, located at the third sacral foramen) and Hui Yang (BL35, 0.5 inches [≈10 mm] from the sacrococcygeal side) in the foot solar bladder meridian. The lower cut needle direction is 30° to 45° for Zhongxiao and slightly superior lateral direction for Huiyang. After insertion of the needles, all needles are rotated and lifted until pain, numbness, distension, and heaviness can be felt. The electrodes are then attached laterally to the needle handles of BL33 and BL35 bilaterally, and the ES lasts 30 minutes at a frequency of 50 Hz and a current intensity of 1 to 5 mA. The treatment should be received three times a week (preferably every other day) for 6 weeks, for a total of 18 sessions [77].

Acupuncture is a traditional treatment method in Chinese medicine and the mechanism of acupuncture in SUI remains unclear. However, intrinsic urethral sphincter deficiency and excessive urethral flow due to PFM weakness are its main pathophysiology. The TCM theoretical basis of acupuncture therapy is meridians, but we can also infer the specific nerves stimulated by their acupuncture sites i.e. S3 nerve through BL33 and pubic nerve through BL35. Since acupuncture technique as an invasive technique produces some pain and discomfort and its mechanism is still unclear, further research should be conducted [78].

4.3. Laser therapy

Fistonic et al. evaluated the effect of laser treatment on SUI using the International Consultation on Incontinence Questionnaire–Urinary Incontinence Short Form (ICIQ-UI SF). Their results showed that in menopausal women with moderate or severe SUI symptoms, the use of laser treatment for SUI was associated with significantly higher ICIQ-UI SF scores ($p < 0.01$) [79].

Several studies have shown that among laser treatment techniques, Er: YAG is very effective in the treatment of mild to moderate postpartum SUI [80-82]. Er: YAG can restructure the patient's vaginal mucosa, improve its morphology and function, and relieve symptoms. This technology is also widely used in the medical aesthetic industry. Er: YAG laser SMOOTH® is a non-invasive, non-ablative laser procedure. The laser energy is strongly absorbed by water, so the laser pulses achieve controlled heating of the collagen in the deep mucosal layer and the temperature of the instrument during the treatment can reach 65°C. The increase in temperature causes the intermolecular cross-linking of the stable collagen triple helix structure in the treated area to be broken, resulting in an increase in collagen fibers and an improvement in the stiffness of the collagen fibers, i.e. an increase in collagen fibers and a contraction of loose collagen fibers after the treatment. The improvement of the collagen structure at the treated site and the simultaneous initiation of new collagenases can be observed within 6 months after treatment with this technique. Also morphology shows that the volume density of capillaries and the thickness of the epithelial layer at the treated site increase by 61.1 and 64.5%, respectively [83, 84]. It is worth

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mentioning that the vaginal mucosal surface does not ablate or overheat, which greatly reduces the risk of accidental lesions of the urethra, bladder or rectum due to overheating and perforation.

The recommended parameters for this treatmentare as follows: laser spot size of 7 mm, frequency of 1.6 Hz, and laser energy emitted per unit area of 6.0 J/cm2. The treatment plan includes three sessions separated by 3 to 4 weeks. If the patient's ability to reorganize collagen is not restored during the first session, then these collagen fibers will be restored during the second and third sessions. No general anesthesia is required during the treatment and the lower third of the vagina can be covered with a thin layer of an anesthetic cream. Minor side effects include a warm sensation, increased vaginal discharge, and transient UI [85].

Er: YAG is safe, with no adverse effects, which has a shorter recovery time compared to other techniques at lower cost. Therefore, this technique is also one of the effective methods for treating SUI. However, it should be noted that the cause of hypermobility includes not only abnormalities of the supporting structures of the pelvic floor but also abnormalities of the urethral sphincter; and this treatment technique is aimed at patients with abnormalities of the supporting structures of the pelvic floor due to collagen loss.

4.4. Exercise therapy

Mild to moderate physical activity may reduce the odds of developing UI. It is believed that the pelvic floor structures may be injured when women playing sports. However, the available literature suggests that most physical activities do not damage the pelvic floor but provide many health benefits for women. Moderate to vigorous physical exercise in the early postpartum period is protective [86], which does not affect other parameters of pelvic floor health. In addition light to moderate exercise during pregnancy may also prevent maternal SUI [87]. However, further studies need to be made to fill the research gap. Most of current exercise programs are designed to improve SUI by activating core muscles and strengthening PFMs to increase PFM function.

An experiment showed that yoga interventions could help the recovery of the levator ani hiatus.(LAH) [88]. Compared with the control group, the difference in LAH area during contraction and Valsalva was significantly increased in the experimental group.

In this experiment, yoga training was performed once a week for 60 minutes in 3-5 sets of low to moderate intensity. The movements are shown in Fig.3.

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	Yoga asana practice methods	Image
1	Yoga meditation breathing (perineal constriction): vajra sitting position, sitting on the yoga ball, so that the perineal area fully contact the sphere. When inhaling, the abdomen and pelvic floor are relaxed. As exhaling, pull abdomen back slightly and contract pelvic floor muscles	
2	Hip lift: vajra sitting position, with yoga balls between legs. When inhaling, the spine extend. As exhaling, squeeze the ball between legs and lift hips until thighs are perpendicular to the ground, and then reduce it.	
3	Clock type: lie on the back, bend the knees, tighten the feet together, and place the hands on lower abdomen. When inhaling, pelvis slightly forward, abdomen and inner thighs relax. As exhaling, the pelvis slightly backward, abdominal and inner thighs tightened, while lifting the pubis to tighten the pelvic floor muscles, and then reduce it.	
4	Clip ball bridge: lie on the back with the knees bent, yoga balls tucked between the legs. When inhaling, relax the abdomen and thighs. As exhaling, the legs clamp the ball to tighten the pelvic floor muscles while lifting the buttocks upward, and then reduce it.	
5	Cattle face style: mountain sitting position, bend the left knee on the outside of the right hip, bend the right knee on the opposite side, hands folded on the knee. When inhaling, the spine extends. As exhaling, thigh medial and pelvic floor muscles tighten, slightly lift buttocks. It can be alternated left and right, and then reduce it.	
6	Magic chair: mountain pose, arms up, hips flexed to the magic chair position. When inhaling, the spine extends; As exhaling, the legs are adducted and tightened, and the pelvic floor and gluteal muscles are accepted until the body is upright, and then reduce it.	
7	Bridge: lie on the back with the knees bent, the feet merged and the hands at body sides. When inhaling, relax the abdomen and thighs. As exhaling, tighten the inner side of both legs and pelvic floor muscles, at the same time, lift the buttocks up, to the thigh, hip and abdomen a plane, leg vertical ground, and then reduce it.	
8	Side recumbent leg retraction: lie on body side, bend the lower elbow to support the head, hold the ground with the other hand, bend the upper leg, step on the ground, and form a straight line. When inhaling, lower foot back hook. As exhaling, tighten the lower inner leg and pelvic floor muscles and lift them up off the floor, and then reduce it.	

Figure 3. yoga training.

As mentioned earlier, some women with SUI have symptoms that can even lead to depression, and women with depression may also indirectly develop SUI. It is widely acknowledged that aerobic exercise can help reduce the symptoms of depression [89]. Although the exact mechanism is not clear, BDNF levels were indeed detected significantly increased during aerobic exercise in pregnancy, implying that aerobic exercise could reduce the incidence of depression as well as the risk of having SUI [59]. Moreover, because the anxiety, stigma, and social withdrawal that some patients experience after the onset of SUI can make it difficult for the next step in treatment, psychological treatment can increase treatment adherence.

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4.5. Medication therapy

The antidepressant duloxetine has been proved effective in relieving symptoms of moderate to severe SUI [49], whose pharmacological effects are consistent with the pathology of SUI. Duloxetine is a serotonin-NA reuptake inhibitor, in which 5-HT and NA are important monoamine neurotransmitters, monoamines are involved in certain central nervous system physiological processes and inhibit the micturition reflex by dual reuptake of 5-HT and NA in the synaptic cleft thereby inducing urethral sphincter closure.

During the use of duloxetine, most women discontinued treatment within four weeks, due to adverse effects (45%) including mood disorders and insomnia, or lack of effectiveness (24%) [90, 91]. Another survey showed that about one-third of women dispensing duloxetine reported treatment-related adverse effects (most commonly nausea), and about one-eighth women discontinued treatment for this discomfort [92]. Therefore, despite its positive impact on improving the quality of life of women with SUI, duloxetine is not encouraged as a first-line treatment.

Adrenergic agonists such as pseudoephedrine and phenylephrine, inferred according to the pharmacology and pathology of SUI, constrict the urethra and should theoretically alleviate the symptoms of SUI, but their adverse effects range from insomnia and anxiety to hypertension, arrhythmias, and stroke [93], which are not recommended for patients who are already anxious and depressed.

Research on the use of estrogen is still limited, but it is known that estrogen supplementation works in patients suffering from SUI caused by the damage to the urethral mucosal mechanism [63, 94].

Medication will affect the development of the nursing infant, which is generally not recommended.

5. Discussion

This paper summarizes the treatment for as well as prevention methods of postpartum SUI by analyzing the pathological mechanisms, and the theoretical basis of conservative treatment methods commonly used in clinical practice. The methods include PFMT technique, ES therapy, laser therapy, exercise therapy, and pharmacotherapy. The classification of existing treatments for postpartum SUI can help patients find the most suitable treatment method and improve the efficiency of treatment. However, previous articles did not systematically classify the treatment methods for postpartum SUI based on pathological factors. Although this paper classifies the treatment methods based on the pathological mechanism of postpartum SUI, there is a lack of experimental data and long-term randomized controlled trial (RCT). Therefore, a large number of clinical trials are still needed.

In summary, the generally accepted pathology of postpartum SUI is divided into several mechanisms. First is the damage to the supporting structures of the pelvic floor; second is the weakness of the urethral sphincter; and the last is the damage to the mucosal mechanism of the urethra.

When the pelvic floor is damaged, the lack of pelvic floor support leads to the high mobility of the urethra; and treatment methods for this condition include PFM training, exercise therapy, and ES. However, it should be noted that damage to the supporting structures of the pelvic floor caused by childbirth is divided into myogenic and neurogenic, so there are different treatments available for the two different types. PFMT and exercise therapy are mostly applicable to myogenic because the core principle of both therapies is to strengthen the core muscles and PFMs; VES and SES in ES therapy both directly stimulate the nerves that innervate the PFMs, which are more applicable to pelvic floor injuries of neurogenic origin. It is believed that SUI caused by changes in the POP of the PFMs, i.e., changes in the collagen composition of the PFMs, can also be classified as a condition of inadequate pelvic floor support. And such a condition is most effectively treated with laser therapy according to the article.

The weakness of the urethral sphincter is mostly of neurogenic origin. In addition, its causes are also linked to a decrease in neurotransmitters such as 5-HT, NA, or BDNF. Urethral sphincter weakness is mostly treated clinically by minimally invasive surgery and conventional surgery because the cure rate of conservative treatment is low. Among them, electroacupuncture in traditional Chinese medicine is a combination of minimally invasive therapy and conservative therapy. Some studies have shown it is

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effective for this type of disease, whose principles need to be further studied. As for postpartum SUI caused by a decrease in neurotransmitters is mostly addressed clinically with drugs such as duloxetine. It is important to note that exercise therapy may also alleviate postpartum SUI caused by these two conditions to some extent because the reduction of neurotransmitters and BDNF is associated with postpartum depression.

The last mechanism of damage is caused by the reduction of estrogen in the female body, so the easiest and most direct way is to supplement the estrogen in the patient's body with estrogen medication.

However, it should be noted that all methods have their contraindications, including conservative treatment methods and treatment methods should be chosen carefully.

6. Conclusion

This paper offers a method to find the most suitable conservation treatment for different types of postpartum SUI patients. Since SUI is a common disease that affects postpartum women, using different treatments for postpartum women with different pathology of SUI will make treatment more efficient and effective. The paper starts by emphasizing the importance of curing and finding the most suitable therapy for postpartum SUI patients. Then the second part analyzes previous related studies. Finally, categorization of treatments based on the pathophysiology? of postpartum SUI and the basic principles of treatment methods are made. Based on existing research, this article analyzes the pathophysiological characteristics of postpartum SUI, risk factors, and the rationale for each treatment method to classify the different treatment methods, and to find the relatively appropriate treatment methods for different etiologies, which could provide strong evidence and theoretical support to this field.

The presented study has some limitations. First of all, the conclusions drawn in this paper are based on theoretical inference without experimental validation; and the existence of bias in the RCT data is not considered, which reduces the reliability of the results. Besides, there lacks long-term RCTs studies, which may lead to inaccurate explanations of research findings. Finally, this study includes only RCTs published in English in addition to 3 Chinese papers. There is still much to be done to improve the classification of postpartum SUI conservation treatments.

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