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Pelvic Floor Muscle Physical Therapy: What to Expect

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Patients with chronic vulvar pain typically benefit from a multimodal treatment approach that includes pelvic floor physical therapy. In addition to a comprehensive pelvic floor muscle (PFM) evaluation, the physical therapist (PT) performs a lower quadrant orthopedic examination. After the evaluation, the PT should explain the findings to the patient, describing the plan of care and what to expect during and after treatment sessions. To foster a team approach, the PT should also send the findings and plan of care to the referring health care provider.

Many PTs provide treatment at the end of the evaluation to give the patient some relief and familiarize her with the type of technique likely to be used during treatment. Letting patients know what to expect can ease anxiety, correct misinformation and mentally prepare the patient for treatment. Patients with chronic vulvar pain typically present with overactive PFMs. These muscles are tense, painful and shortened, and may have myofascial trigger points (muscle knots), which are difficult to relax. Therefore, soft tissue mobilization (STM) and manual therapies, such as myofascial release and PFM massage, are the main physical therapy methods used in vulvodynia patients. Since symptoms vary among vulvar pain patients, treatments can produce vastly different results.

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Recent Research Findings of NIH-Funded Vulvodynia Studies

In response to the NVA's urgent plea, the National Institutes of Health (NIH) took a first step toward funding vulvodynia research in 2000. Among the initial NIH-funded studies was Harlow and Stewart's landmark prevalence study,[1] which found that vulvodynia was more common than anyone had thought. In the past 18 years, NIH has supported 35 vulvodynia studies. Although it is a small number compared to the number of studies they funded on other health conditions during that period, NIH involvement has helped to stimulate international interest in vulvodynia research. What follows are summaries of select NIH-funded vulvodynia studies.

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DC Foster, MB Kotok, et al.
Desipramine and/or Topical Lidocaine for LPV[2]

For several decades, tricyclic antidepressants (TCAs) were the first-line therapy for vulvodynia. This practice was solely based on the reported efficacy of TCAs in the treatment of other chronic pain conditions. In 2002, Foster and colleagues were awarded an NIH grant to determine the effectiveness of desipramine and/or topical lidocaine in the treatment of localized provoked vestibulodynia (LPV), the most common subtype of vulvodynia. They conducted a 12-week randomized placebo-controlled, double-blinded clinical trial with 133 LPV patients. Women ages 18 to 50, who reported greater than three continuous months of painful sexual intercourse and/or pain with tampon insertion, were eligible to participate. All study candidates completed a medical history questionnaire and underwent a physical examination. The diagnostic criterion was tenderness localized within the vestibule confirmed by a cotton swab test. Additionally, atrophic vaginitis, vulvar dermatoses, specific neuropathology, fungal/bacterial infections and herpes simplex had to be ruled out.

Women who fulfilled the criteria were assigned to one of four groups: (i) topical lidocaine only; (ii) oral desipramine only; (iii) topical lidocaine and oral desipramine; and (iv) placebo. The initial dosage of desipramine was 25 mg at bedtime, increased once a week by 25 mg until the target dose of 150 mg was reached. The second group applied 5% lidocaine cream to the vulvar vestibule four times daily, and the third group used both treatment regimens concurrently. The placebo group was either given tablets identical to the 25 mg desipramine tablets or placebo cream that was identical in color and texture to the 5% lidocaine cream.

At the end of 12 weeks, participants' vestibular pain was re-evaluated with a cotton swab test and vulvar algesiometer, plus a battery of health-related quality of life questionnaires. Clinical response was assessed by multiple outcome measures: pain with tampon insertion, daily pain intensity, intercourse pain intensity and frequency of intercourse.

There was no significant difference in pain reduction between the desipramine or lidocaine groups and their placebo groups. The investigators concluded that oral desipramine and topical lidocaine, as single therapies or in combination, failed to relieve the pain associated with LPV. Interestingly, the only significant difference was that sexual satisfaction improved in the desipramine group compared to placebo.

CS Brown, GA Bachmann, DC Foster, et al.
RCT of Gabapentin for PVD[3]

Brown, Bachmann and Foster conducted a multicenter randomized clinical trial (RCT) of the anticonvulsant gabapentin in the treatment of provoked

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The NVA is not a medical authority and strongly recommends that you consult your own health care provider regarding any course of treatment or medication.

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vestibulodynia (PVD). Gabapentin was selected because of its reported efficacy in treating other neuropathic pain conditions and the promising preliminary data on its use in women with localized or generalized vulvodynia. The three centers used for data collection were the University of Tennessee Health Science Center, Robert Wood Johnson Medical School and the University of Rochester Medical Center. The primary goal of the study was to determine whether extended release gabapentin, 1,200 – 3,000 mg daily, reduces pain with tampon insertion in patients with PVD. This 18-week placebo-controlled, double-blind crossover study enrolled women between the ages of 18 and 50.

The recruitment criteria were tenderness localized to the vulvar vestibule, pain with tampon insertion and introital dyspareunia, if sexually active. Of the 230 women screened (average age of 37), 89 were enrolled and 66 completed the study. The majority had been experiencing vulvar pain for over five years and there was close to an equal split of women classified with 'primary' versus 'secondary' onset of vulvar pain. Participants were randomly assigned to either the gabapentin or placebo group. After reaching and remaining on a therapeutic dose of gabapentin for two weeks, women in the gabapentin group had their dosage gradually tapered, followed by a four day washout period. In the study's next phase, the women who had previously received gabapentin were given the placebo and vice versa.

The primary outcome measure was pain with tampon insertion, because many women with PVD abstain from sexual intercourse. Secondary outcomes were daily vulvar pain and pain with intercourse. This multicenter study's results did not corroborate the findings of prior uncontrolled studies of gabapentin use in vulvodynia. No significant difference was found in pain with tampon insertion between the gabapentin and control groups. There was also no difference between the two groups on secondary outcome measures. These data do not support the use of gabapentin alone in the treatment of PVD.

After many decades as a first-line treatment for vulvodynia, two studies have shown that oral medication alone is ineffective in the treatment of PVD, the most common subtype of vulvodynia. It has not yet been determined whether a combination of pharmacologic and non-pharmacologic measures is effective in the treatment of PVD. The investigators recommend that health care providers consider all therapeutic options in treating vulvodynia, including non-pharmacologic interventions such as physical therapy, cognitive behavior therapy and sexual counseling.

BL Harlow, CG Kunitz, et al.
Prevalence of Vulvodynia in Two Populations[4]

Harlow and colleagues used validated specific questions associated with a clinically confirmed diagnosis of vulvodynia to compare (i) the cumulative incidence of unexplained vulvar pain and (ii) the prevalence of care-seeking behavior in women from the Boston metropolitan area (BMA) and Minneapolis/St. Paul (MSP). In the BMA, women were screened using census-based data, whereas the MSP data was gathered from women seeking care at outpatient community clinics.

Harlow received self-administered questionnaires from 5,440 women in the BMA and 13,681 in MSP. The questions asked pertained to a woman's history of vulvar burning and pain on contact lasting more than three months, which limited or prevented her from having sexual intercourse. The study's analysis was based on women between 18 and 40 years of age.

Data analysis found that 7-8 percent of women experienced symptoms consistent with vulvodynia by age 40. There was no appreciable difference in the cumulative incidence of vulvodynia between the BMA and MSP samples. In both samples, first onset of vulvar pain was greatest before age 25. Data was also analyzed by race and confirmed Harlow's earlier finding that women of Hispanic/Latina origin have a greater risk of developing vulvar pain symptoms than Caucasians.

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Harlow's second aim was to compare the prevalence of care-seeking behavior in the two samples. Although a higher percentage of women in the BMA sought treatment for vulvar pain compared to those in MSP, a substantial percentage in each sample (48% in MSP and 30% in the BMA) did not seek treatment for it. Women in the MSP sample sought treatment for other medical conditions at a clinic, indicating that the stigma of having vulvar pain prevented them from seeking care.

In summary, Harlow reported a high prevalence of vulvodynia in two geographic regions, with peak onset in the early 20s. Hispanic/Latina women have an increased risk of developing vulvar pain. He concluded that access to health care does not increase the likelihood that women will seek treatment for vulvodynia.

BD Reed, SD Harlow, et al.
Remission, Relapse and Persistence of Vulvodynia[5]

Reed and colleagues conducted a survey-based assessment in a longitudinal population-based study of women who screened positive for vulvodynia. After completing a short interview, participants were sent a 26-page survey, which included a previously validated screen for vulvodynia, as well as questions about demographics, health history, exposures, current symptoms and other potential risk factors. The survey also included a limited set of screens for other pain conditions, such as fibromyalgia and interstitial cystitis. Those completing the baseline survey were sent additional follow-up surveys every six months. Only women who completed at least four follow-up surveys were included in the analysis, which was based on information obtained from baseline through the 36-month follow-up survey. Outcome measures were remission without relapse, relapse after remission and persistence of a positive vulvodynia screen.

Of the 441 women screening positive for vulvodynia, 239 completed four additional surveys. Of these, almost 10 percent had consistently positive vulvodynia screens, over 50 percent had a remission without relapse, and almost 40 percent relapsed following remission. Compared to remission alone, overall factors associated with both relapse and persistence of pain were increased severity of pain, pain after intercourse, longer duration of symptoms and screening positive for fibromyalgia. Factors associated with persistence only were more severe symptoms with intercourse and pain with oral sex or partner touch. Relapse only was associated with having provoked pain or screening positive for interstitial cystitis at first positive vulvodynia screen. Demographic characteristics, age at pain onset, and whether vulvodynia was classified as primary or secondary did not predict outcome.

Reed concluded that persistence of vulvodynia symptoms without remission is the exception rather than the rule. However, about 50 percent of women in remission experience a relapse within 6 to 30 months. Pain history and comorbid conditions were associated with the more severe outcomes of relapse and/or persistence compared to those who remitted only. These findings further support the observation that vulvodynia is heterogeneous and often occurs in an episodic pattern.

ML Falsetta, **DC Foster**, **et al.**
Inflammatory Mechanisms in LPV[6]

Over 20 years ago, researchers at the University of Rochester Medical Center, led by Dr. David Foster, began studying inflammatory mechanisms in patients with localized provoked vestibulodynia (LPV). Initially, Foster demonstrated that fibroblasts generate heightened pro-inflammatory mediator responses in the vulvar vestibule of women with LPV. Specifically, he found that pro-inflammatory mediators associated with pain, interleukin 6 (IL-6) and prostaglandin E2 (PGE2), were elevated in the vestibule of LPV patients. Further research found that the presence of yeast activates fibroblasts in the vulvar vestibule to produce elevated levels of pro-inflammatory mediators.

In the current study, Falsetta and Foster sought to determine the precise mechanism by which yeast triggers

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inflammatory pathways in LPV patients. In a control matched study, the researchers obtained primary fibroblast strains from biopsies of vestibular tissue in women with and without LPV. Biopsies were also obtained from non-painful external vulvar sites in LPV patients. They grew vestibular fibroblasts in culture dishes and then introduced *Candida albicans*, the most commonly diagnosed strain in women with active yeast infections. Their main focus was examining the intracellular mechanisms by which these fibroblasts recognize *C. albicans*.

To understand their findings, it is important to know that the receptor Dectin-1 and the protein complex NFκB play important roles in regulating the body's immune response to yeast infection. Dectin-1 senses the presence of live yeast infection and then activates NFκB, which produces pro-inflammatory mediators associated with pain, e.g., IL-6.

The researchers found that vestibular fibroblasts from LPV patients expressed slightly elevated levels of Dectin-1 compared to controls. Additionally, Dectin-1 was modestly elevated in vestibular fibroblasts, but not in external vulvar fibroblasts of LPV patients. The results also showed that when fibroblasts were stimulated with live yeast, the NFκB pathway was activated. Blocking the function of Dectin-1 results in a significant decrease in IL-6 and PGE2 production and inhibiting NFκB stops secretion of these pro-inflammatory mediators in vulvar fibroblasts. Thus, Dectin-1 and NFκB are two potential targets for the development of new treatments for LPV.

Additional results from Falsetta's study found that, even in response to a low dose of *C. albicans* cells, vestibular cells of LPV patients produced pro-inflammatory mediators IL-6 and PGE2. Such a low dose is unlikely to be detected by standard diagnostic methods and is not associated with active yeast infection. Doses required to elicit a response in control fibroblasts and in the external vulvar fibroblasts of LPV patients were roughly 1000-fold greater and consistent with active infection. This finding suggests that the vulvar vestibule of women with LPV is inherently sensitive to yeast. Apparently, *C. albicans* infection can be mild and undetected by clinical screening methods, but still be sensed by fibroblasts, which generate an extreme immune response. This finding adds support to the proposition that a fibroblast-mediated pro-inflammatory response to *C. albicans* contributes to the induction of pain in women with LPV. Thus, treatments developed to inhibit this response might be useful in the early stages of LPV and/or potentially prevent its development.

MA Farmer, AM Taylor, et al.
Recurrent Vulvovaginal Candidiasis: A Mouse Model of Vestibulodynia[7]

Farmer and colleagues developed a mouse model to empirically evaluate the hypothesis that prolonged vulvovaginal inflammation initiates a chronic state of vulvar allodynia. She induced a yeast infection by repeatedly exposing the vulva of a subset of mice to *Candida albicans*, a fungal pathogen. Her intent was to mimic the human condition of recurrent yeast infections. For testing, Farmer used a mechanical sensitivity method, in which von Frey filaments were applied to the mouse vulva to assess pain with contact. Each post-infection vulvar sensitivity measurement took place three weeks after negative yeast cultures were obtained. The tissue also was examined for nerve fiber density and expression of pain fiber activation.

After repeated exposure to *C. albicans*, the mice exhibited vulvar allodynia, pain with light touch to the vulva, and hyperinnervation. The outcome measure for allodynia was observing a mouse jump with all four paws in the air upon vulvar contact. Long-lasting behavioral allodynia was also observed after a single, extended Candida infection or repeated vulvar inflammation induced by live yeast or zymosan, a mixture of fungal antigens. Both the hypersensitivity and hyperinnervation were present at least three weeks after resolution of the infection or inflammation.

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For example, some patients experience immediate relief with STM and others feel pain. Educating patients about the physiology of the body's response to STM will better prepare them for their subsequent physical therapy visits.

Physical Therapy Evaluation

During the examination, the PT assesses the superficial and deep PFMs for pain, tension and myofascial trigger points, as well as muscle strength and length. This is done by external and internal (transvaginal or transrectal) palpation and observation. Surface electromyography biofeedback helps the PT assess muscle instability and PFM activity at rest, during and in response to a contraction. In the lower quadrant orthopedic examination, the PT assesses spinal, pelvic and hip alignment; leg and core strength; movement and postural disorders; and neurologic function; she also performs specific orthopedic tests. This examination helps to determine whether further medical tests are warranted or whether orthopedic issues could be causing or perpetuating the vulvar pain. The PT may also palpate abdominopelvic, spinal and lower quarter muscles to assess for tension, and evaluate myofascial trigger points and flexibility.

At the end of the evaluation, the PT explains relevant findings and develops an individualized treatment plan. She may also provide treatment, e.g., soft tissue mobilization down-training, and teach the patient relaxation techniques and stretching exercises to perform at home. The prescribed number of sessions ranges from one to three times per week, depending on the severity of the problem. Treatment usually takes somewhere between three months and one year.

Pelvic Floor Muscle Physical Therapy

After the evaluation, all sessions are spent on treatment. For a portion of each session, the PT should perform STM on the pelvic floor muscles. STM of the superficial layer of the PFMs includes effleurage, circular massage strokes with the palm of the hand, plus cross-friction massage (repeated strokes against the direction of muscle fibers). Skin rolling is used to identify and treat areas of muscle restriction or abnormal cross-linking of fascia or connective tissue. Other gentle myofascial release techniques to dissipate connective tissue restrictions may also be performed. Since the skin rolling technique can provoke pain if performed too aggressively, the patient should be forewarned about this possibility and can choose whether to include it in the treatment.

Soft tissue mobilization performed on the deep PFM layer can be external, transvaginal, and/or transrectal. With the patient's permission, the PT inserts a lubricated gloved finger into the vaginal or anal opening and performs long repeated petrissage strokes (massage applying deep pressure) along the PFMs, compressing the underlying muscles. STM can be performed for several minutes on each side of the body. The original term for this treatment was, "Thiele's massage," a classic manual technique for relieving pain and trigger points in the levator ani and coccygeus muscles. In this technique, the PT's inserted finger performs a "stripping" or sweeping motion of the muscles from origin to insertion, using minimum to moderate pressure, as tolerated. The movement is done in a "U-shaped" manner, back and forth in the vaginal canal. It must be done carefully, because manipulation of soft tissue using too much biomechanical pressure can cause tissue damage, such as microtearing, bruising, or inflammation. If a therapist is too aggressive or the treatment is performed for too long, it can cause more pain. Likewise, if the patient has localized vestibulodynia, direct manipulation of the vestibule can increase her pain. In this case, the therapist should perform STM techniques with an adapted hand position to avoid direct contact with the vestibule or suggest a transrectal approach to avoid touching it altogether. The benefit of sustained pressure is that it can release or resolve a myofascial trigger point or stretch/lengthen deep PFMs. Six muscles on each side of the body are treated: the pubococcygeus, iliococcygeus,

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coccygeus, puborectalis, obturator internus, and piriformis. Tension in the obturator internus and piriformis (deep hip rotator muscles) can impact the PFMs.

Understanding Muscle Physiology

PFMs are similar to other skeletal muscles. When skeletal muscles are overworked or maintain prolonged tension, lactic acid accumulates and an imbalance of electrolytes can occur. Overworked muscles require more energy, electrolytes and oxygen. The presence of oxygen normally generates energy for muscles to use. When there is a good supply of oxygen, pyruvate (output of glucose metabolism) is shuttled via an aerobic pathway to be broken down for more energy. Overuse of muscles requires more energy than the body can generate aerobically so it relies on an anaerobic process. In this process, pyruvate is converted to lactate, which creates more energy. However, lactate can accumulate in the muscles, causing muscle soreness and fatigue. The perpetual overactivity of the PFMs in women with chronic vulvar pain causes an overabundance of lactic acid and an imbalance of electrolytes, which results in a burning sensation in the PFMs. This is not to be confused with the concurrent burning that can be felt in the vulvar vestibule or generalized burning in the vulva. In a normal muscle recovery phase, lactate and electrolytes are cleared or balanced by the body, but in overactive PFMs, the accumulation of lactic acid and electrolyte imbalance can result in muscle tension or cramping, muscle fatigue, swelling, hardness, myofascial trigger points, tender points, and/or inability to relax a muscle. This inability to relax a muscle causes it to maintain a chronic shortened state. Shortened muscles with sustained contraction are susceptible to cramping.

Another possible trigger of PFM dysfunction is an injury, such as a fall or trauma. The injury causes an inflammatory and immune response that leads to an overabundance of calcium and phosphate, which can stimulate activity in the actin and myosin filaments (the contractile proteins in muscle). This excess can lead to increased metabolic activity, and when the muscle fails to adjust to the increase, muscle hardening occurs. Increased calcium and phosphate play a central role in muscle fatigue. As the injury evolves, the result is fibrosis, an overgrowth and hardening of connective tissue.

Benefits of Soft Tissue Mobilization

As with all skeletal muscles, the benefits of STM or massage of the PFMs are improved circulation of blood and lymphatics, improved muscle tone, resolution of myofascial trigger points, modulation or decrease of afferent neuronal pain pathways, and improved connective tissue mobility. Since one characteristic of overactive PFMs is shortening of muscles, a goal of performing STM is to passively stretch or lengthen muscles. The PT applies progressive pressure to a muscle in a downward and outward manner, away from the center on either side (as tolerated). Sustained pressure for 30 seconds or more can help to stretch a PFM. A stretched muscle can soften, relax and begin to assume its normal resting position and contract fully. The stretching process should not cause pain, which means that tissue damage and inflammation is beginning. Instead, the stretching should feel like a strong pulling or pressure sensation. The patient must communicate how the STM feels so the therapist can adjust the pressure to the patient's comfort level. Avoiding a pain response is of paramount importance, because causing more pain to a painful muscle can result in muscle tightening, increase patient anxiety and perpetuate a pain cycle. Another beneficial outcome of STM is desensitization, i.e., normal sensation and tolerance to touch in the perineum, PFMs and associated musculature is restored. The overall goal is to decrease the perception of pain and increase the threshold of pain at the vaginal opening and in the PFMs.

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Patient Response to PFM Therapy

Pain is an aversive sensory and emotional experience. Patients respond differently to STM because they have different pain thresholds and pain perception, as well as differences in prior experience with physical or massage therapy. Some patients take pain medication before the visit, which can influence a patient's response. Cultural background, nociceptive stimuli and the environment impact pain perception. Prior trauma, anxiety and history of depression can lead to a lowered pain tolerance. All of these factors, plus one's belief system, emotional responses and expectations, can influence how a patient feels after treatment.

While some patients perceive immediate pain relief and decreased tension during treatment, others report an increase in pain or have low tolerance for any pressure applied to the PFMs. If the patient has a history of chronic regional pain syndrome, fibromyalgia, or endometriosis, she usually has a lower tolerance for pressure applied to soft tissue. The PT would have to adjust the amount of pressure or change the manual technique to an indirect or gentler approach. Some patients feel pain during the session, but experience relief of pain after heat or ice application. Ice is predominantly used to treat pain and inflammation, but if it isn't well-tolerated, heat may be used as an alternative. Ice or heat may be applied to the vulvar region for 15 - 20 minutes before and/or after the treatment session to help relax the muscles further or alleviate any inflammatory process or pain response. Another common occurrence after soft tissue mobilization of PFMs is an increase in pain for a day or two after therapy, because sometimes it can cause short-term inflammation. Afterwards, however, the patient should experience a significant reduction in PFM pain. If patients experience pain for many days after STM, it means that the PT is working too aggressively for the patient's tolerance level and may be causing tissue damage or irritation of the pudendal or levator ani nerve. Furthermore, if pain is provoked in the vestibule from direct contact by the therapist, the patient should let her know. Providing feedback to the PT is crucial, especially for new patients. The PT can adjust the STM pressure or length of time spent working on the PFMs to minimize negative reactions to treatment.

(*Editor's note*: To obtain references, contact Lisa Goldstein at lisa@nva.org or at 301-299-0775.)■

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Footnotes

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