Intravaginal Surface Electromyography in the Diagnosis and Treatment of Vulvovaginal Pain Disorders

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History, Definitions and Treatment of Vulvodynia

Although my clinical practice encompasses a wide range of lower urogenital tract pain disorders, as well as urinary and bowel retention and incontinence disorders, my research has been focused on essential vulvovaginal pain syndromes known collectively as vulvodynia. Vulvodynia is a descriptive, not a diagnostic, term covering a wide range of disorders that have, as one component, pain in the vulvar area. In no other area of biofeedback practice is it more important to rule out all organic causes for the symptoms prior to commencing treatment and to treat patients only under referral from a specialty physician, not on self-referral. Sources of vulvovaginal discomfort include vaginal infections, hormonal changes, dermatoses, venereal disease, oncological disease, and trauma. Many women experience transient vulvar irritation from any of the above sources or from contact with irritants, including soaps, detergents, topical vulvar preparations used to treat some of the above conditions, prolonged or inadequately lubricated penile vaginal intercourse, and vulvar trauma associated with accidents or surgery. In most cases, the irritation does not need to be addressed once the underlying causes have been identified and treated. In vulvodynia the regional pain persists after resolution of the provocation and any organic sequelae. Vulvodynia is an essential pain disorder, diagnosed by exclusion of identifiable organic pathology. This extremely limited overview of the sources of vulvar irritative symptoms is given to emphasize the necessity for a complete diagnostic workup and appropriate medical treatment before any biofeedback intervention is considered.

One subset of vulvodynia, vulvar vestibulitis syndrome (VVS), is characterized by introital dyspareunia (painful intercourse) and may involve swelling, erythema, and exquisite tenderness to touch localized to the vestibule of the vagina (Friedrich, 1987). Patients with this condition typically suffer no discomfort unless there is direct pressure on the vestibule. These patients are often intercourse abstinent and, eventually, totally sexually abstinent for prolonged periods of time. Because this condition has no known etiology and is symptomatically manifest and functionally limiting primarily in the area of sexual activity, it is not surprising that some have suggested that this is a psychogenic disorder. Research in this area has demonstrated clearly that this population shows no significant medical, psychological, or sexual history differences from normal matched controls, thus ruling out this hypothesis (Meana, Binik, Khalif, & Cohen, 1997). Conservative medical treatment for this condition includes low-dose tricyclics or anticonvulsants to block the nerve-mediated pain, antihistamines, cox-2 inhibitors and leukotriene inhibitors to reduce the localized inflammation, alpha-interferon injections, topical palliatives such as OiIated colloidal oatmeal, and topical anesthetics. If these interventions produce unsatisfactory results, the gold standard treatment has been the surgical excision of the affected area, a skinning vestibulectomy with vaginal advancement, and perineoplasty (Marinoff & Turner, 1992).

Essential or dysesthetic, meaning unpleasant altered sensation, vulvodynia is a condition of diffuse, unpro- voked vulvar burning, which can vary from mild to extreme and from intermittent to chronic. It tends to be progressive with respect to chronicity and intensity of symptoms. It is of unknown etiology and may have no visible vulvar changes. Like vestibulitis, it tends to reduce sexual activity,
leading frequently to sexual abstinence and the associated psychological and interpersonal consequences. Medical treatments for this condition include hormone replacement therapy (HRT), tricyclics, antihistamines, anticonvulsants, muscle relaxants, topical palliatives, and anesthetics. Surgery has not been shown to have any beneficial role in the treatment of this condition.

**Origins of Biofeedback for Treatment of Vulvodynia**
Cutaneous Vulvar Clinic physicians at Columbia University College of Physicians and Surgeons first approached me in 1991. They had noticed that during intravaginal digital palpation, the levator muscles of women suffering from vulvodynia manifest considerable chronic “tension and spasticity.” These specialists requested the use of biofeedback to correct this muscle abnormality. The pain did not appear to be of myofascial origin, but rather the dysesthesia and hyperalgesia seemed more consistent with sympathetically mediated chronic regional pain syndrome, previously known as reflex sympathetic dystrophy. I began to work with the vulvodynia patient population using the standardized protocols and treatment regimes developed for the urological disorders of retention and incontinence (Perry, 1984). It was immediately noticeable that the SEMG patterns of this population’s pelvic muscles showed abnormal resting hypertonicity and instability, as well as hypotonicity and instability during phasic, tonic, and endurance voluntary contractions. After a period of “trial and error” in working with these patients, I began to turn my focus away from the resting hypertonus and contractile hypotonus. This came about because several patients had significantly increased their contractile amplitude using biofeedback contractile up-regulation, but still showed little, if any, symptomatic benefit and, in fact, were sometimes worse. It appeared that the resting hypertonicity was most associated with lower urogenital tract pain. Focusing training on down-regulating resting amplitude proved to be only marginally more successful, still leaving a sizable portion of the population with little to no benefit. Statistical analyses suggested that the variability of the SEMG signal, and not the amplitude, was a critical determinant of pain reduction (Glazer, Rodke, Swencionis, Hertz, & Young, 1995). Of course, signal variability measures were clearly noted to vary directly in proportion to amplitude (i.e., higher signal amplitudes are more variable) both at rest and during contractions. Correcting for amplitude-related variability is accomplished by using the “coefficient of variation” as an amplitude-independent measure of SEMG signal variability. This measure of SEMG signal variability can then be used to compare group differences and to predict pain changes. The evaluation protocols have further evolved to include measures of contractile recruitment and recovery latencies as well as Fast Fourier Transformation measures for power density spectral frequency analysis.

**Protocol**
The “Glazer” protocol for pelvic floor muscle evaluation uses a five-segment evaluation sequence assessing preand postbaseline rest, as well as phasic, tonic, and endurance contractions. Patients are first taught how to lift or elevate the pelvic floor muscles and their relationship to body position and activity of the surrounding musculature. Rapid; flicking (phasic); intermediate 10- second sustained (tonic); and long-duration, 60-second sustained (endurance) contractions assess the sexual, sphincteric, and support functions of the pelvic floor. Automated protocol software instructs patients with both on-screen text and voice prompts to “flick,” “work,” or “rest” to let the patient know when to contract and when to relax the pelvic floor muscles. This protocol is a similar sequence to that used in assessing pelvic floor muscles for incontinence. The difference is not in the sequence of muscle actions but the measurements taken. During the pelvic pain protocol, in each contraction and relaxation period one measures integrated SEMG amplitude, standard deviation, coefficient of variability, and recruit/recovery latencies and takes power density spectral frequencies for tonic and endurance contractions. Another difference between the Glazer protocol and previous incontinence protocols is that accessory muscles are not necessarily minimized. Each patient is assessed with the use of different combinations of accessory muscles. This is done in order to determine the best balance between keeping the patient’s focus on the internal “lifting” sensation and, at the same time, maximizing the use of the muscle contraction to result in a reduction in amplitude and variability in the subsequent rest period. We look for an exercise position, contraction type, contraction duration, and number of repetitions that maximize the therapeutic value of the exercise. All patients are started on two 20-minute exercise sessions per day, each one consisting of 60 repetitions of 10-second contractions alternated with 10-second relaxation phases. All patients are required to use home training devices and intravaginal sensors in the conduct of their home exercises.

Patients return for office evaluations every 2 weeks for their second and third visits and then monthly for subsequent visits. The frequency of office visits is determined by the observation of the clinician of both SEMG and symptomatic changes and compliance of the patient in the conduct of
home exercises. Over time and with continued training, we look for increased contractile amplitudes and spectral frequencies with decreased contractile coefficients of variability and rise and recovery times. In relaxation measures, we look for reduced amplitude and reduced coefficients of variability. Amplitude changes are not enough and we have seen, as mentioned earlier, many patients showing improved contractile amplitude with reduced resting amplitude and little therapeutic benefit. We believe that the spectral frequencies, rise and recovery times, and coefficients of variability are related to the predominant fiber type being recruited and the coordination of use of that fiber type. The critical combination of higher amplitude contractions with higher spectral frequency, faster rise/fall times, and reduced coefficients of variability suggest a predominance of coordinated fast twitch (type II) fibers. In the presence of this phenomenon, the consequence is reduced amplitude and variability during rest and a reduction of the hypertonicity and instability associated with chronic uncoordinated discharge of fast twitch fibers as seen in the resting SEMG of untreated vulvovaginal pain patients. A comparison of normative pelvic floor SEMG readings and those of various groups of lower urogenital tract pain patients can be found in several studies including Glazer, Jantos, Hartmann, and Swencionis (1998); Glazer et al. (1995); White, Jantos, and Glazer (1997); and Hetrick et al. (2005).

**Research on Pelvic Floor Muscle SEMG Biofeedback for Vulvodynia**

The first publication using SEMG-assisted rehabilitation of pelvic floor musculature in the treatment of vulvovaginal pain (Glazer et al., 1995) demonstrated a slightly more than 50% cure rate with an average self-reported improvement of 83%, and 80% of sexually abstinent patients resuming regular intercourse. Two main findings emerged statistically. First, there were neither demographic nor SEMG characteristics on initial evaluation that predicted response to this treatment modality. Second, the research showed that only changes in the standard deviation of the resting SEMG signal predicted pain change. This finding confirmed my anecdotal experience that the treatment is essentially an SEMG stabilizing program. This paper also concluded that “The response to this therapy suggests that whatever the initial insult or etiologic factor, vulvar vestibulitis syndrome may be a result of autonomically mediated pain. This mechanism, as a final common pathway for multiple etiologies, may explain the lack of consensus on a single antecedent, despite consistency in symptomatology of the syndrome.”

A 1996 paper presented evidence that by guiding patients to use the naturally occurring cocontractions of internal obturator, lower abdominals, and adductor longus muscles, one could support and enhance the amplitude of the pelvic floor contraction and reduce resting hypertonicity. Thus the Glazer protocols require the individualized “testing” of the patient with different positions and the use of different combinations of accessory muscles that enhance, rather than interfere, with the correct use of the pelvic floor muscles (Glazer & MacConkey, 1996).

A 1997 paper (White, Jantos, & Glazer, 1997) compared a cohort of 32 vulvar vestibulitis syndrome patients with a matched control group of normal patients and found several SEMG characteristics that reliably differentiated the two groups. Cutoffs for these SEMG characteristics were developed and summarized in this paper, resulting in over 80% diagnostic accuracy for vulvodynia using pelvic floor SEMG measures. A 1998 paper (Glazer et al., 1998) compared dysesthetic vulvodynia patients to a matched control group of normal patients and reported similar findings, demonstrating over 80% differential diagnostic accuracy for vulvar dysesthesia using pelvic floor SEMG measures. In 2000 a study was published that concluded that 3–5 years after successful treatment, 100% of those studied remained completely asymptomatic with no reports of either vulvar dysesthesia or introital dyspareunia (Glazer, 2000). Unexpectedly, measures of sexual interest, frequency, and satisfaction did not fully return to presymptomatic levels. It was concluded that full functional rehabilitation must include not only pain relief but psychosexual rehabilitation as well to achieve both a symptomatic and functionally favorable outcome.

Two studies on this subject were published in 2001. The first (McKay et al., 2001) studied the effectiveness of pelvic floor SEMG biofeedback in the management of patients with moderate to severe vulvar vestibulitis syndrome and reported that 84.7% of treated patients reported either negligible or mild pain at the end of the study and 70% resumed sexual activity; this compares favorably to the results of perineoplasty surgery for the treatment of vulvar vestibulitis. The second study (Bergeron et al., 2001) reported a randomized controlled comparison of vestibuloplasty, electromyographic biofeedback, and group sex therapy/pain management in the treatment of dyspareunia resulting from vulvar vestibulitis. This study concluded that both medical and psychological treatments are effective in relieving dyspareunia and recommended a multimodal approach to treatment.

In 2002 we presented a technological advancement in the field with a study demonstrating that
complete patient evaluation and treatment protocols can be conducted remotely and in real time using a web-enabled SEMG protocol (Glazer, Marinoff, & Sleight, 2002). At the present time we are setting up remote office sites in western Europe that will allow the conduct of live, real time, audio/video enabled patient intake and pelvic floor SEMG evaluation and treatment sessions over the Internet.

The most recent research published (Hetrick et al., in press) compares a group of male patients meeting criteria for National Institutes of Health (NIH) type IIIa prostatitis, also known as prostatodynia, with an asymptomatic matched control group. This study is now in press and demonstrates intra-anal pelvic floor SEMG readings that parallel differences between vulvodynia sufferers and their asymptomatic controls. We are designing future studies to evaluate the clinical efficacy of pelvic floor SEMG biofeedback in the treatment of chronic prostatodynia.

**Conclusion**

Free-form observations of SEMG—with or without direct pelvic muscle palpation—do not comprise an adequate evaluation. Replicable protocols, applied to the patient over time, are necessary to assess progress. Similarly, amplitude and standard deviation measures alone are not adequate to assess changes. Spectral frequencies, rise and recovery times, and coefficients of variability must all be utilized to ensure that correct rehabilitation of the pelvic floor muscle is taking place. For those trained in the traditions of incontinence, it is also important to remember that one must explore various positions, the use of accessories, contraction duration, and number of repetitions to best achieve the desired SEMG changes and symptomatic benefit.

**References:**


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